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Review Article

Thyroid dysfunction in India: what is different

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ABSTRACT

Thyroid dysfunction (TD) is a common endocrine condition worldwide. Thyroid hormone is essential for growth, development, and energy metabolism. TD if left untreated leads to various serious health consequences. Various risk factors for the development of TD are iodine deficiency, ageing, genetic susceptibility, smoking status, ethnicity, and endocrine disruptors. Indians are at high risk for the development of TD as compared to Caucasians. Indian thyroid patients differ than Caucasians in many aspects such as higher prevalence of congenital hypothyroidism and other TD, higher rate of progression of subclinical hypothyroidism (SCH) to overt hypothyroidism (OH), and lower prevalence of thyroid associated ophthalmopathy (TAO). In this article, we tried to summarize the current evidences regarding environmental and biological factors that place Indians at high risk of TD as compared to other ethnic groups. High prevalence and different characteristics of Indian TD call for investigation into cause of increased susceptibility and way to prevent TD at individual and population levels.

Keywords: Thyroid dysfunction, Hypothyroidism, Thyrotoxicosis, Congenital hypothyroidism

INTRODUCTION

Thyroid dysfunction (TD) is a common endocrine problem worldwide and currently, 200 million people (40% of world population) worldwide are suffering from TD.[1] TD equally affects individuals from developed countries, developing countries and under developed countries. Indians seem to be at high risk of developing TD.[2-4] While the prevalence of TD in India is high and increasing, however, there is considerable heterogeneity across the different states.[2] Reasons for high heterogeneity among states are many such as different geographical region (coastal vs. plain), variations in lifestyle factors (rural vs. urban), and differing states of socioeconomic development (rich state vs. poor state).[2] Indian TD patients differ from western patients in many aspects. First, the prevalence of TD is higher in India as compared to the western world.[3] Second prevalence of congenital hypothyroidism is higher than in developed countries.[5] Third, the rate of progression of subclinical hypothyroidism to overt hypothyroidism is higher in Indian patients as compared to western populations.[6] Fourth, the ratio of total T3/total T4 (TT3/TT4) in Graves' disease is lower than Caucasian's.[7] Fifth, the prevalence of TAO (thyroid associated ophthalmopathy) is lower in Indians as compared to white people.[7,8]

Higher prevalence and different characteristics of TD in Indian are likely indicative of underlying different biological factors in Indians along with rapid changes in activity, dietary, and other lifestyle behavior. Herein, we discuss the current evidence comparing Indian thyroid patients with other populations with regard to biological and environmental factors.

CONGENITAL HYPOTHYROIDISM (CH)

CH is the commonest cause of preventable mental retardation and with timely screening and treatment, there is improvement in the outcome of affected child.[5] Prevalence (per 1000 neonates screened) of CH in India is higher than in several other countries.[5] In non-endemic region of India prevalence is 1 in 1031 (0.97) neonates. However in an endemic area, in neonates born to mothers with thyroid disorder and in preterm neonates the prevalence is 79, 50 and 14, respectively.[5] In developed countries such as Japan, USA, UK and Germany the prevalence is 1 in 2500-3500, 1 in 2000-4000, 1 in 1887 and 1 in 3330 children, respectively.[9-12] Prevalence is also lower in developing countries such as Egypt and Serbia.[13,14] This shows that prevalence is higher in India. Even Asians (including Indians) living in USA have higher incidence of CH.[10] However in USA prevalence is also increasing and this is due to more preterm delivery and changing testing strategy from primary T4-follow up TSH approach to primary TSH testing.[15] Detection of milder cases of CH is also a reason for the increase in the incidence of CH in Western countries.[15] In India, transient hypothyroidism is present in 14% of cases while permanent CH is present in 86% cases.[5] Transient hypothyroidism is more in Europe (1:100) and less in USA (1:50,000) as compared to India.[16] At a cut-off value of 20 mIU/l, screen positivity rate were 0.91% for post natal TSH and and 5.6% for cord TSH.[5] In earlier studies from India and also from Canada, 75-85% of CH was due to thyroid dysgenesis and dyshormonogenesis was responsible for 15-25% of cases.[5,17] However recent metaanalysis suggests that thyroid dysgenesis and dyshormonogenesis account for 56.6% (95% CI: 50.9%-62.2%) and 38.7% (95% CI: 33.2%-44.3%), respectively.[5] Various gene variants are associated with CH but what proportion of CH is attributable to known genetic cause is unknown.[18-21] DUOX2 gene mutation is most commonly associated with Asian populations (16-32%) while in Europeans, variants of thyroglobulin gene (TG) gene is more common (55%) than DUOX2 vari-ants (18%).[18-21] This shows that different genes may be responsible for variations in prevalence of CH in different ethnicities. Other reasons for high CH in India as compared to other developed countries is due to more consanguineous marriage, high prevalence of maternal hypothyroidism, presence of iodine deficiency, more pre-term delivery, and industrial contaminants.[22-25]

HYPOTHYROIDISM

Prevalence of hypothyroidism is high in India.[2,3,26] Prevalence of hypothyroidism in India is around 10.95% as compared with 4.6% in USA and 2% in UK.[3,26] In a meta-analysis from Europe the prevalence of undiagnosed subclinical hypothyroidism was 4.11% and overt hypothyroidism was 0.65%.[27] In Black individual hypothyroidism appears to be lower than white individuals.[28] However in comparing different prevalence studies one should consider many things which affect the prevalence rate such as the use of different cut off value (3.5-5.5 mIU/l), study design (population based study vs. cohort study), sample size (few hundred to ten thousands), lab test performed (second generation TSH vs. third generation TSH). Studies since 1995 uses 3rd generation TSH and this represent 10 fold increase in the sensitivity over 2nd generation TSH assay. In India prevalence varies from region to region.[2] Higher prevalence was seen in those areas, which are located inland (e.g., Delhi, Bangalore, Hyderabad and Ahmedabad) as compared to coastal cities (Chennai, Goa and Mumbai).[2] Average prevalence in coastal cities is around 9.5% as compared to 11.7% in inland cities.[2] Prevalence also varies with age.[29,30] As compared to people aged 18-35 years, people aged 46-54 years have higher prevalence (13.11% vs. 7.53%) of hypothyroidism.[2] Reason behind high prevalence in India as compared to western countries is possibly due to long standing iodine deficiency in the country, which is partly corrected.[26,31,32] Iodine supplementation can be also blamed for more autoimmune thyroid disorder as iodine supplementation can induce or ag-gravate autoimmunity.[31,32] Besides this endocrine disruptors, cyanogenic compounds and goitrogens have an adverse effect on thyroid metabolism.[26,31,32] Preva-lence of SCH is also high in Indians (8.02-19.3%) as compared to Caucasians (3-8%).[2,26,29] Patients of SCH may be asymptomatic but one third may have symptoms suggestive of thyroid hormone deficiency. SCH can be an early stage of thyroid hor-mone deficiency and may progress to overt hypothyroidism. Rate of progression of SCH to overt hypothyroidism in India is higher than Caucasians (2-4% annually).[6] In a study by Vanderpump et al rate of progression was 2.6% and 4.3% each year in TPO Abs negative and TPO Abs positive patients, respectively.[33] However an Indian study concluded that the rate of progression was 18.97% (35% in TPO Abs positive vs. 10.53% in TPO Abs negative patients) in one year.[6] The high rate of progression could be due to smaller thyroid gland size and weight as compared to Caucasians.[34] Another reason could be younger age at presentation.[6]

THYROTOXICOSIS

Prevalence of overt hyperthyroidism and subclinical hyperthyroidism in India is 0.67% and 1.27%, respectively.[2] Similar prevalence rate is seen in Europe and USA (0.7% vs. 0.5%).[26,35,36] A lower prevalence rate (0.3%) of both overt and subclinical hyperthyroidism was reported from Australia.[37] Prevalence of hyperthyroidism varies from region to region and it is primarily due to different iodine intake of that region.[38] Higher rate of hyperthyroidism was seen in iodine-deficient countries and this is mostly due to increase prevalence of toxic nodular disease in elderly patients.[26] In iodine deficient village (Pescopagano) of Italy, the prevalence of hyperthyroidism was reported as 2.9% in 1999, which was higher than what we see in iodine sufficient countries.[39] In a study from China a lower prevalence of hyperthyroidism was seen in iodine sufficient area as compared to iodine

deficient area (1.0% vs. 1.2%; p<0.001).[40] There is lack of comprehensive population based studies from Africa regarding the prevalence of thyroid dysfunction. However a small old aged home based study from Cape Town a prevalence of 0.6% of hyperthyroidism was seen.[41]

The etiology of hyperthyroidism varies from country to country and this is primarily due to iodine intake of that country.[26] In iodine sufficient countries, Graves' disease (GD) accounts for 70-80% of hyperthyroid patients whereas in iodine deficient countries GD account for the 50% of cases and other half is due to toxic nodular goiter (TNG).[26,42] In a cross-sectional study of patients with thyrotoxicosis from India, Singh et al show that 84.21%, 11.84%, and 3.95% patients were suffering from GD, sub-acute thyroiditis (SAT) and TND, respectively after excluding drug-induced and pregnancy induced thyrotoxicosis.[7] Clinical phenotype of hyperthyroidism also varies according to aetiology.[42] Compared to patients with GD, patients of TNG are older, lower thyroid hormone level, are more likely subclinical than overt hyperthyroidism and associated with more cardiovascular disorder (CVD).[42,43] Ethnicity influences the risk of development of complication associated with thyotoxicosis. TAO are 6 times more common in Caucasians (42%) as compared to Asians (7.7%).[8] In a small study from India, Singh et al also found a lower prevalence (15.62%) of TAO in GD patients, which was lower than Caucasians.[7] This may be due to different genetic backgrounds and lower rates of smoking.[7] Thyrotoxic periodic paralysis (TPP) is also less commoner in Asians.[44,45] Ratio of TT3/TT4 for diagnosis of GD vs. thyroiditis is lower (>14.1 vs. >20) in Indians as compared to Japanese and others.[7,46] In a study from Japan, Amino et al proposed that ratio of TT3/TT4 of >20 is useful in differentiation of GD from SAT.[46] However a study from India, Singh et al had proposed the ratio of >14.1 was most appropriate for the diagnosis of GD.[7] This may be probably due to older age at presentation and iron deficiency in Indian patients.[7]

PREGNANCY AND THYROID DISORDER

Prevalence of hypothyroidism is high in Indian pregnant women as compared to western countries. In a meta-analysis, it was found that prevalence of hypothyroidism was 11.07%. SCH was present in 9.51% of cases and OH was present in 2.74% of cases.[47] In an another multicentric epidemiological study, using trimester-specific cutoff value (TSH >2.5, >3.0, >3.0 mIU/l for 1st, 2nd, and 3rd trimester, respectively), 44.3%, 32%, and 34% of pregnant women were found to have hypothyroidism.[48] However when TSH level of >4.5 mIU/l was used as cutoff value for diagnosis, 13.13% were found to have hypothyroid.[48] TPOAbs positivity was seen in 20.74% of all pregnant women while in hypothyroid pregnant women TPOAbs positive rate was 40%.[48] In a study by Rajput et al prevalence of SCH and TPOAbs positivity was found in 27.8% and 21.5% of pregnant women, respectively.[49] Globally prevalence of overt and subclinical hypothyroidism is 0.2-1% and 1.5-4%, respectively while incidence of subclinical and overt thyrotoxicosis is 2.5% and 0.2%, respectively.[50-53] In a study by Klein el from USA, the prevalence of hypothyroidism (TSH >6) was found to be 2.5% (OH: 0.3%; SCH: 2.2%) of cases.[54] In studies using newer criteria (trimester specific cutoff value) prevalence of SCH was found 6.8% (Belgium) and 15% (USA).[55,56] Thyroid autoantibody is present in 5-15% of women of reproductive age world widely.[53] However in a study from India, 18.9% of euthyroid pregnant women were found to be TPOAbs positive.[57] Initial studies report that the prevalence of IDD during pregnancy was high (53.68%) but a recent study by Pramanik et al did not report any IDD in pregnant women.[58,59] Reason for high prevalence of thyroid disorder in India during pregnancy is due to high prevalence of thyroid disorder in general population, increased autoimmunity and iodine deficiency.

CONCLUSION

In conclusion, current data suggests that a significant proportion of Indian popula-tion are suffering from TD. Indian thyroid patients differ in many aspect from Cau-casians. Autoimmunity appears to play important etiological role in pathogenesis of thyroid disorder. Identification of risk factors and causative factors in pathogenesis of TD in Indians is urgently warranted.

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REFERENCES

1. Kuyl JM. The evolution of thyroid function tests. J Endocrinol Metab Diabetes S Afr. 2015;20:11-6.
2. Unnikrishnan AG, Menon UV. Thyroid disorders in India: an epidemiological perspective. Indian J Endocrinol Metab. 2011;15(2):78-81.
3. Bagcchi S. Hypothyroidism in India: more to be done. Lancet Diabetes Endocrinol. 2014;2(10):778.
4. Singh SK, Singh R, Singh A. Prevalence of thyroid disorders (TD) in newly diagnosed type 2 diabetes mellitus patients - a cross sectional study. J Assoc Physicians India. 2021;69(10):11-2.
5. Anne RP, Rahiman EA. Congenital hypothyroidism in India: A systematic review and meta-analysis of prevalence, screen positivity rates, and etiology. Lancet Reg Health Southeast Asia. 2022;5:100040.
6. Singh SK, Singh R, Singh SK, Iquebal MA, Jaiswal S, Rai PK. Risk of progression to overt hypothyroidism in Indian patients with subclinical hypothyroidism: a prospective observational study. Int J Adv Med. 2022;9:1183-7.
7. Singh SK, Singh R, Singh SK, Iquebal MA, Jaiswal S, Rai PK. Aetiological sub-classification of thyrotoxicosis and relevance of TT3/TT4 ratio in sub-classification of patients with thyrotoxicosis; an Indian cross-sectional study. Int J Adv Med. 2023;10:830-5.

8. Tellez M, Cooper J, Edmonds C. Graves' ophthalmopathy in relation to cigarette smoking and ethnic origin. Clin Endocrinol (Oxf). 1992;36(3):291-4.
9. Nagasaki K, Sato H, Sasaki S, Nyuzuki H, Shibata N, Sawano K, et al. Re-evaluation of the prevalence of permanent congenital hypothyroidism in Niigata, Japan: a retrospective study. Int J Neonatal Screen. 2021;7(2):27.
10. Harris KB, Pass KA. Increase in congenital hypothyroidism in New York State and in the United States. Mol Genet Metab. 2007;91(3):268-77.
11. Knowles RL, Oerton J, Cheetham T, Butler G, Cavanagh C, Tetlow L, et al. Newborn screening for primary congenital hypothyroidism: estimating test performance at different TSH thresholds. J Clin Endocrinol Metab. 2018;103(10):3720-8.
12. Lüders A, Blankenstein O, Brockow I, Ensenauer R, Lindner M, Schulze A, et al. Neonatal screening for congenital metabolic and endocrine disorders–results from Germany for the years 2006–2018. Dtsch Arztebl Int. 2021;118(7):101-8.
13. Bekhit OE, Yousef RM. Permanent and transient congenital hypothyroidism in Fayoum, Egypt: a descriptive retrospective study. PLoS One. 2013;8(6):68048.
14. Mitrovic K, Vukovic R, Milenkovic T, Todorovic S, Radivojcevic J, Zdravkovic D. Changes in the incidence and etiology of congenital hypothyroidism detected during 30 years of a screening program in central Serbia. Eur J Pediatr. 2016;175(2):253-9.
15. Rastogi MV, LaFranchi SH. Congenital hypothyroidism. Orphanet J Rare Dis. 2010;5:17.
16. Gaudino R, Garel C, Czernichow P, Léger J. Proportion of various types of thyroid disorders among newborns with congenital hypothyroidism and normally located gland: a regional cohort study. Clin Endocrinol (Oxf). 2005;62(4):444-8.
17. Eugene D, Djemli A, VanVliet G. Sexual dimorphism of thyroid function in newborns with congenital hypothyroidism. J Clin Endocrinol Metab. 2005;90(5):2696-700.
18. Park KJ, Park HK, Kim YJ, Lee KR, Park JH, Park JH, et al. DUOX2 Mutations are frequently associated with congenital hypothyroidism in the Korean population. Ann Lab Med. 2016;36(2):145-53.
19. Matsuo K, Tanahashi Y, Mukai T, Suzuki S, Tajima T, Azuma H, et al. High prevalence of DUOX2 mutations in Japanese patients with permanent congenital hypothyroidism or transient hypothyroidism. J Pediatr Endocrinol Metab. 2016;29(7):807-12.
20. Fan X, Fu C, Shen Y, Li C, Luo S, Li Q, et al. Next-generation sequencing analysis of twelve known causative genes in congenital hypothyroidism. Clin Chim Acta. 2017;468:76-80.
21. Nicholas AK, Serra EG, Cangul H. Comprehensive screening of eight known causative genes in congenital hypothyroidism with gland- *in situ*. J Clin Endocrinol Metab. 2016;101(12):4521-31.
22. Kalam MA, Sharma SK, Ghosh S, Roy S. Change in the prevalence and determinants of consanguineous marriages in India between national family and health surveys of 1992-1993 and 2015-2016. Human Biology. 2021;92(2):93-113.
23. Sharma SK, Kalam MA, Ghosh S, Roy S. Prevalence and determinants of consanguineous marriage and its types in India: evidence from the National Family Health Survey, 2015-2016. J Biosoc Sci. 2021;53(4):566-76.
24. Pandav CS, Yadav K, Srivastava R, Pandav R, Karmarkar MG. Iodine deficiency disorders (IDD) control in India. Indian J Med Res. 2013;138:418-33.
25. Kalra S, Unnikrishnan AG, Sahay R. Thyroidology and public health: the challenges ahead. Indian J Endocrinol Metab. 2011;15(2):73-5.
26. Taylor P, Albrecht D, Scholz A. Global epidemiology of hyperthyroidism and hypothyroidism. Nat Rev Endocrinol. 2018;14:301-16.
27. Mendes D, Alves C, Silverio N, Marques F. Prevalence of undiagnosed hypothyroidism in europe: a systematic review and meta-analysis. Eur Thyroid J. 2019;8(3):130-43.
28. Sichieri R, Baima J, Marante T, de Vasconcellos MT, Moura AS, Vaisman M. Low prevalence of hypothyroidism among black and Mulatto people in a population-based study of Brazilian women. Clin Endocrinol (Oxf). 2007;66(6):803-7.
29. Chaker L, Razvi S, Bensenor IM. Hypothyroidism. Nat Rev Dis Primers .2022;8:30.
30. Empson M, Flood V, Ma G, Eastman CJ, Mitchell P. Prevalence of thyroid disease in an older Australian population. Intern Med J. 2007;37(7):448-55.
31. Brent GA. Environmental exposures and autoimmune thyroid disease. Thyroid. 2010;20(7):755-61.
32. Marwaha RK, Tandon N, Ganie MA, Kanwar R, Sastry A, Garg MK, et al. Status of thyroid function in Indian adults: two decades after universal salt iodization. J Assoc Physicians India. 2012;60:32-6.
33. Vanderpump MP, Tunbridge WM, French JM, Appleton D, Bates D, Clark F, et al. The incidence of thyroid disorders in the community: a twenty-year follow-up of the Whickham Survey. Clin Endocrinol (Oxf). 1995;43(1):55-68.
34. Harjeet A, Sahni D, Jit I, Aggarwal AK. Shape, measurements and weight of the thyroid gland in northwest Indians. Surg Radiol Anat. 2004;26(2):91-5.
35. Hollowell JG, Staehling NW, Flanders WD, Hannon WH, Gunter EW, Spencer CA, et al. Serum TSH, T(4), 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.
36. Garmendia Madariaga A, Santos Palacios S, Guillén-Grima F, Galofré JC. The incidence and prevalence

of thyroid dysfunction in Europe: a meta-analysis. J Clin Endocrinol Metab. 2014;99(3):923-31.
37. Walsh JP. Managing thyroid disease in general practice. Med J Aust. 2016;205(4):179-84.
38. Laurberg P, Pedersen KM, Vestergaard H, Sigurdsson G. High incidence of multinodular toxic goitre in the elderly population in a low iodine intake area vs. high incidence of Graves' disease in the young in a high iodine intake area: comparative surveys of thyrotoxicosis epidemiology in East-Jutland Denmark and Iceland. J Intern Med. 1991;229(5):415-20.
39. Aghini-Lombardi F, Antonangeli L, Martino E, Vitti P, Maccherini D, Leoli F, Rago T, et al. The spectrum of thyroid disorders in an iodine-deficient community: the Pescopagano survey. J Clin Endocrinol Metab. 1999;84(2):561-6.
40. Du Y, Gao Y, Meng F, Liu S, Fan Z, Wu J, et al. Iodine deficiency and excess coexist in china and induce thyroid dysfunction and disease: a cross-sectional study. PLoS One. 2014;9(11):111937.
41. Muller GM, Levitt NS, Louw SJ. Thyroid dysfunction in the elderly. S Afr Med J. 1997;87(9):1119-23.
42. Nyström HF, Jansson S, Berg G. Incidence rate and clinical features of hyperthyroidism in a long-term iodine sufficient area of Sweden (Gothenburg) 2003-2005. Clin Endocrinol (Oxf). 2013;78(5):768-76.
43. Biondi B, Kahaly GJ. Cardiovascular involvement in patients with different causes of hyperthyroidism. Nat Rev Endocrinol. 2010;6(8):431-43.
44. Kelley DE, Gharib H, Kennedy FP, Duda RJ Jr, McManis PG. Thyrotoxic periodic paralysis. Report of 10 cases and review of electromyographic findings. Arch Intern Med. 1989;149(11):2597-600.
45. Okinaka S, Shizume K, Iino S. The association of periodic paralysis and hyperthyroidism in Japan. J Clin Endocrinol Metab. 1957;17(12):1454-9.
46. Amino N, Yabu Y, Miki T, Morimoto S, Kumahara Y, Mori H, et al. Serum ratio of triiodothyronine to thyroxine, and thyroxine-binding globulin and calcitonin concentrations in Graves' disease and destruction-induced thyrotoxicosis. J Clin Endocrinol Metab. 1981;53(1):113-6.
47. Yadav V, Dabar D, Goel AD, Bairwa M, Sood A, Prasad P, et al. Prevalence of hypothyroidism in pregnant women in India: a meta-analysis of observational studies. J Thyroid Res. 2021;2021:5515831.
48. Dhanwal DK, Bajaj S, Rajput R, Subramaniam KA, Chowdhury S, Bhandari R, et al. Prevalence of hypothyroidism in pregnancy: An epidemiological study from 11 cities in 9 states of India. Indian J Endocrinol Metab. 2016;20(3):387-90.
49. Rajput R, Goel V, Nanda S, Rajput M, Seth S. Prevalence of thyroid dysfunction among women during the first trimester of pregnancy at a tertiary care hospital in Haryana. Indian J Endocrinol Metab. 2015;19(3):416-9.
50. Allan WC, Haddow JE, Palomaki GE, Williams JR, Mitchell ML, Hermos RJ, et al. Maternal thyroid deficiency and pregnancy complications: implications for population screening. J Med Screen. 2000;7(3):127-30.
51. Casey BM, Dashe JS, Wells CE, McIntire DD, Byrd W, Leveno KJ, Cunningham FG. Subclinical hypothyroidism and pregnancy outcomes. Obstet Gynecol. 2005;105(2):239-45.
52. Vaidya B, Anthony S, Bilous M, Shields B, Drury J, Hutchison S, et al. Detection of thyroid dysfunction in early pregnancy: Universal screening or targeted high-risk case finding? J Clin Endocrinol Metab. 2007;92(1):203-7.
53. Negro R, Stagnaro-Green A. Clinical aspects of hyperthyroidism, hypothyroidism, and thyroid screening in pregnancy. Endocr Pract. 2014;20(6):597-607.
54. Klein RZ, Haddow JE, Faix JD, Brown RS, Hermos RJ, Pulkkinen A, et al. Prevalence of thyroid deficiency in pregnant women. Clin Endocrinol (Oxf). 1991;35(1):41-6.
55. Moreno-Reyes R, Glinoer D, Van Oyen H, Vandevijvere S. High prevalence of thyroid disorders in pregnant women in a mildly iodine-deficient country: a population-based study. J Clin Endocrinol Metab. 2013;98(9):3694-701.
56. Blatt AJ, Nakamoto JM, Kaufman HW. National status of testing for hypothyroidism during pregnancy and postpartum. J Clin Endocrinol Metab. 2012;97(3):777-84.
57. Rajput R, Yadav T, Seth S, Nanda S. Prevalence of thyroid peroxidase antibody and pregnancy outcome in euthyroid autoimmune positive pregnant women from a tertiary care center in Haryana. Indian J Endocrinol Metab. 2017;21(4):577-80.
58. Rajput R, Yadav L, Nanda S, Yadav R. Trimester specific nutritional status of iodine among euthyroid pregnant women. Thyroid Disord Ther. 2017;6:208.
59. Pramanik S, Mukhopadhyay P, Bhattacharjee K, Bhattacharjee R, Mukherjee B, Mondal SA, et al. Trimester-specific reference intervals for thyroid function parameters in indian pregnant women during final phase of transition to iodine sufficiency. Indian J Endocrinol Metab. 2020;24(2):160-4.

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