Original Article

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Prevalence of Hypothyroidism among Pregnant Women in the Sub Mountain State of Manipur

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Abstract

Background: Hypothyroidism is among the most common endocrine disorder encountered during the pregnancy and reproductive age group. Endemic iodine deficiency and autoimmune disease remain a major cause of hypothyroidism. This study was conducted at Jawaharlal Nehru Institute of Medical Sciences, situated in Manipur, a submountain area of India. The aim of this study was to find out the prevalence of hypothyroidism among pregnant women in Manipur.

Method: All the consecutive 400 first- and second-trimester pregnant women were registered for the study after institutional ethics approval and consent from the study subjects. The pregnant women with diagnosed thyroid disorder and on thyroid medication were excluded from the study. Apart from routine obstetrical investigations, thyroid stimulating hormone (TSH) was done. Test for free T4 was done in patients with TSH level > 3 mIu/L.

Result: The mean (SD) age of study subjects was 26.8 (±8.2) years. About 92 (23%) subjects had TSH values > 3.0 mIu/L, the cut-off value used for the upper limit of normal in this study. Out of these 72 (18%) had normal FT4 value and, therefore, labeled as subclinical hypothyroidism (SCH) and 18 (4.5%) had low FT4, hence termed overt hypothyroidism. Two women had low FT4 values and normal TSH, hence labeled as isolated hypothyroidism.

Conclusion: Prevalence of hypothyroidism in pregnancy was found to be higher, more so the SCH in the present study.

Key words: Autoimmune disease, Endemiclodine deficiency, Overt and subclinical hypothyroidism, Pregnancy, Sub mountain area of Manipur

INTRODUCTION

Thyroid disorder is a common occurrence in pregnancy and reproductive age group next to diabetes mellitus.[1] Endemic iodine deficiency and Hashimoto's disease remain a major cause of hypothyroidism.[2] Pregnancy has a profound impact on the thyroid gland and its functions. During pregnancy, the thyroid gland may enlarge by 10% in iodine-replete countries and by 20-40% in areas of iodine deficiency.[3] Production of thyroid hormones and iodine requirement each increases by approximately 50% during pregnancy.[3] Iodine requirement in pregnancy is increased due to increased renal loss cause by increased renal blood flow, increased glomerular filtration rate and increased renal clearance due to reduced tubular reabsorption of iodine. Throughout pregnancy, maternal thyroxin is transferred to the fetus.[4-7] Maternal thyroxin is important for normal fetal brain development, neural implication, migration and structural organization, thus, future intellectual development especially before the development of fetal thyroid gland.[3] This insult is likely to occur in the first trimester and, therefore, preconceptional optimization of thyroxin therapy is important.[8] Maternal thyroxin contribution remains important sources before 12 weeks of gestation after which fetal thyroid synthesized hormone.[9] The increased demand for thyroid hormone starts very early, reaching a plateau at 16-20 weeks.

Pregnancy is a stress test for the thyroid and the physiological changes may result in hypothyroidism in

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the later stages in women with limited thyroidal reserve with underlying autoimmune disease or iodine deficiency who were euthyroid prior to conception.[3] Serum thyroid stimulating hormone (TSH) level in early pregnancy decline because of weak TSH reception stimulation from massive quantities of human chorionic gonadotropin (hcG) secreted by placental trophoblast during the first trimester. The TSH level is the lowest and FT4 level is the highest when hcG levels peak.[2]

The pregnancy-related changes in thyroid physiology make diagnosis of thyroid disorder difficult, because it can simulate signs and symptoms of physiological changes of pregnancy.[7] Symptoms of heat intolerance, sluggishness, fatigue, and examination findings of tachycardia, edema, hair changes, and weight gain are common to pregnancy and thyroid disease much in same way.[7] The management of therefore based principally on biochemical measures.

Although it is well accepted that overt hypothyroidism (OH) have a deleterious impact on pregnancy, studies are now focusing on potential impact of subclinical hypothyroidism (SCH) on maternal and fetal heath, the association between miscarriage and preterm delivery in euthyroid women positive thyroid peroxidase (TPO) and/or thyroglobulin antibody.[2,3] Undiagnosed SCH is likely associated with some adverse pregnancy outcomes. It may progresses to overt thyroid failure, and the rate of progression is affected by TSH level, age of women, disorder such as diabetes, and presence and concentration of TPO antibody.[2]

The prevalence reports of hypothyroidism during pregnancy in India, a country considered to be a relative moderate iodine deficiency, ranges from 4.8% to 11%.[8] The prevalence of OH and SCH complicating pregnancy has been reported 3% and 9%, respectively.[9] There are few published Indian studies on this topic. Therefore, this study was conducted with sincere effort to throw some light on this topic.

MATERIALS AND METHODS

This study was conducted in the Department of Obstetrics and Gynecology. All consecutive pregnant women who gave consent were included in this study.

Inclusion Criteria

- All pregnancy women registered in the hospital
- Duration of pregnancy: First trimester to the second trimester.

Exclusion Criteria

- Pregnant women with diagnosed thyroid disorder and on thyroid medication
- Multiple gestation
- Diabetes mellitus
- Hypertension
- History of recurrent pregnancy loss.

All subjects were subjected routine ante-natal checkup with an obstetric profile of investigation. TSH was tested in all subjects registered for the study. In patients with deranged TSH, free T4 test were done. The test was carried out by chemi-luminescence immunoassay (vitros 5600).

The reference ranges used in this study was based on the guidelines of American Thyroid Association (ATA) for the diagnosis and management of thyroid disease during pregnancy and postpartum. As per regulation 14.2 of ATA guidelines, if trimester-specific ranges for TSH are not available in the laboratory, the following reference ranges are recommended: first trimester, 0.1-2.5 mlu/L, second trimester, 0.3-3 mlu/L.[3]

RESULT

The patients were divided into the groups as shown in Table 1 according to TSH and FT4 value.

Euthyroid defined as serum level of TSH 0.2-3 mlu/L with FT4 normal level. SCH defined as serum TSH > 3 mlu/L with FT4 normal level. OH defined as TSH > 3 mlu/L with FT4 < 7.5 mcg/dl.

Two hundred sixteen subjects had both TSH and FT4 value within normal limit. About 92 (23%) subjects had TSH value > 3.0 mlu/L, the cut-off value for the upper limit of normal in this study. Out of this 72 (18%) had normal FT4 values and, therefore, labeled as SCH and 18 (4.5%) subjects had low FT4, hence termed OH. Whereas TSH value was normal, 2 (0.5%) had low FT4 and labeled as isolated hypothyroidism (Table 2).

DISCUSSION

Maternal hypothyroidism is defined as the presence of an elevated TSH concentration during gestation. More recently, normative data from healthy pregnant women suggest the

Table 1: The patients were divided into the following groups according to TSH and FT4 value[3]

Groups	TSH (mlu/L)	FT4 (mcg/dl)
Euthyroid	0.2-3.0	N
SCH	>3.0	N
OH	>3.0	<7.5

SCH: Subclinical hypothyroidism, OH: Overt hypothyroidism, TSH: Thyroid stimulating hormone

Table 2: Thyroid dysfunction in pregnant women (*n*=400)

Thyroid dysfunction	*n*	%
Euthyroid	216	54
Hypothyroidism	92	23
SCH	72	18
OH	18	4.5
Isolated hypothyroidism	2	0.5

SCH: Subclinical hypothyroidism, OH: Overt hypothyroidism

upper reference range may approximate 2.5-3 mlu/L.[3] When maternal TSH is elevated, measurement of serum FT4 concentration is necessary to classify the patient's status as either SCH or OH. The distinction of OH from SCH is important because published data relating to maternal and fetal effects attributable to OH are more consistent.[3]

Thyroid dysfunction during pregnancy has an immense impact on maternal and fetal outcomes.[10,11] More importantly, children born to hypothyroid mothers have a poor intellectual function during the latter part of their life.[3] Low IQS in infants of even very mild hypothyroid women have been reported.[5] There is an increased risk of preeclampsia placental abruption, intra-uterine growth restriction, prematurity and intra-uterine fetal demise.[2] Therefore, the majority of the developed countries have national neonatal screening program.[12] The prevalence of hypothyroidism is more in Asian countries compare with the west, it varies from 2.5% from the West to 11% from India.[12-14]

Rao *et al.* found hypothyroidism in 4.2% of recurrent pregnancy less which is statistically significant.[14] Sahu *et al.* have done thyroid function in the second trimester and reported prevalence of thyroid disorder especially OH and SCH 6.47%.[15]

Dhanwal *et al.* from Delhi in 2013 reported hypothyroidism prevalence of 14.3% with cut-off value of 4.5 mlu/L as the upper limit of normal in a cohort of 100 pregnant women.[12,13] In another study from Delhi Nagia AS *et al.* in 2013 reported a prevalence rate of 12% amongst 400 pregnant women.

In the present study, in contrast, has shown the prevalence of hypothyroidism as high as 23% with 4.5% OH and 18% SCH, thus necessitating the need for universal screening for thyroid function during pregnancy.

Various reasons have been proposed for increased prevalence of hypothyroidism in pregnant women especially in sub mountain areas (Kashmir to North East India). Geo-chemical nature in deficiency of iodine and micronutrients, due to glaciations, high rain fall, floods leading to decrease iodine content in soil and water is considered to be the cause of increase prevalence of hypothyroidism in this region.[16,17]

CONCLUSION

The study concludes that there is a high prevalence of hypothyroidism in pregnant women of Manipur majority being SCH. In view of deleterious effects of hypothyroidism in pregnancy routine screening of thyroid dysfunction may be recommended especially in endemic iodine deficiency area.

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