ORIGINAL RESEARCH PAPER

Obstetrics & Gynaecology

TO STUDY THE PREVALENCE OF MATERNAL THYROID DISORDER AND ITS IMPACT ON MATERNAL AND FETAL OUTCOME- AN OBSERVATIONAL STUDY.

KEY WORDS:
Hypothyroidism, Pregnancy, Anaemia, β-HCG

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ABSTRACT

Introduction: Thyroid disorders constitute one of the most common endocrine disorders seen in pregnancy. Maternal thyroid function changes during pregnancy and inadequate adaptation to these changes result in thyroid dysfunction. There's a need to treat thyroid disorders in reproductive-age women before conception. In pregnancy, overt hypothyroidism is seen in 0.2 % and subclinical hypothyroidism in 2.3 % of cases. The main obstetric complications are abortion, pre-eclampsia, abruptio-placenta, and preterm labor and the fetal complications are prematurity, low birth weight, stillbirth, and perinatal death. **Method:** It's a prospective observational study conducted on 198 pregnant patients, who were admitted to SHKBM, Jhalawar. After getting informed consent from all patients, TSH, fT3, fT4 estimation was done from all patients by blood sample. Feto maternal outcome was recorded.

Aims And Objectives Of The Study:

- To study the prevalence of thyroid dysfunction in pregnancy
- To study the feto-maternal outcome in patients with thyroid dysfuction

Statistical Analysis: All maternal and fetal outcomes were analysed using Chi-square test. **Results:** Of the 198 women screened, 22 (11%) had abnormal thyroid function. In women with subclinical and overt hypothyroidism, anemia was present in 23.6% being significantly associated with hypothyroidism. With respect to fetal outcome, LBW 31.6%, NICU admission 41.1%, and low APGAR Score 21.1% were statistically associated with hypothyroidism. **Conclusion:** Prevalence of subclinical hypothyroidism is 5.9% in 3rd trimester of pregnancy. Anemia, pre-eclampsia, and neonatal morbidities are significantly associated with hypothyroidism

INTRODUCTION

One of the most prevalent endocrine disorders that pregnant women experience is thyroid disease. During pregnancy, the mother's thyroid function changes, and a lack of adjustment to these changes leads to thyroid dysfunction. These modifications are the result of a number of factors, including an increase in thyroglobulin due to elevated estrogen and human chorionic gonadotrophin, increased renal iodine losses due to increased glomerular filtration rate, modifications in peripheral thyroid hormone metabolism, and modifications in iodine transfer to the placenta. During pregnancy, the need for iodine and the production of thyroid hormone both rise by 50%. Maternal thyroid disorders influence multitudes of mother and fetal outcomes. There's a need to treat thyroid disorders in reproductive-age women before conception. Overt hyperthyroidism commonly leads to complications like fetal loss, fetal growth restriction, preeclampsia, and preterm delivery[1].

Poor obstetric and fetal outcomes have been observed in thyroid disorders in the early pregnancy. Preterm labor, abortion, pre-eclampsia, abruptio placenta, and stillbirth are the main obstetric complications. Fetal complications include prematurity, low birth weight, stillbirth, and perinatal death. The future intellectual development of children born to untreated mothers is significantly affected. Children born to mothers who were hypothyroid have been reported to experience adverse prenatal and postnatal outcomes, including attention deficit and hyperactivity syndrome. The prevalence of thyroid disorders during pregnancy has a wide geographic variation. Western literature shows a prevalence of hypothyroidism in pregnancy of 2.5% and hyperthyroidism in pregnancy has a prevalence of 0.1 to 0.4%. There is a paucity of data on the prevalence of thyroid disorders in Indian pregnant women. Few reports show a prevalence of 4.8% to 11% amongst the Indian pregnant population[1].

Physiology of maternal and fetal thyroid in pregnancy: During pregnancy, the thyroid gland undergoes physiological changes, such as moderate enlargement of the gland and an increase in vascularization. Due to its structural similarity to thyroid-stimulating hormone (TSH), beta-Human Chorionic Gonadotropin (-HCG) causes thyroid stimulation since the first trimester. The thyrotropic activity of -hCG also causes a decrease in serum TSH in the first trimester, so pregnant women have lower serum TSH concentrations than non-pregnant women.

As a result of estrogen stimulation, thyroid-binding globulin (TBG) levels in the blood also rise. On the other hand, fetal intake and placenta metabolism both result in an increased renal clearance, which causes a relative decline in iodide availability.

A few weeks after conception, the circulating level of TBG rises along with increased hepatic synthesis and estrogen-mediated prolongation of TBG half-life from 15 minutes to 3 days, before reaching a plateau in the middle of pregnancy. Thyroxine (T4) and triiodothyronine (T3) total concentrations rise in the first trimester of pregnancy and plateau early in the second trimester, peaking at a concentration value that is 30–100% higher than in prepregnancy, primarily following the rise in TBG. Due to the physiological changes in thyroid function during pregnancy, reference ranges of TSH or free thyroxine (fT4) obtained from non-pregnant populations vary in pregnant women. Pregnancy causes physiological and hormonal changes that increase thyroxin (T4) and triiodothyronine (T3) production by up to 50%, increasing a woman's daily iodide requirement. TSH levels also decrease, especially in the first trimester.

Thyroglobulin frequently increases during pregnancy reflecting an enhanced activity of the thyroid gland. After 12 weeks of pregnancy, the fetal thyroid starts to concentrate iodine and synthesize thyroid hormones. Until then, any thyroid hormones needed to support the fetal brain's physiological development are provided by the mother's reserves.

In women with low thyroid reserve, the stress of pregnancy can cause clinical or subclinical hypothyroidism. In women with low thyroid reserves stress of pregnancy manifests as

overt disease. In an iodide-sufficient area, thyroid adaptations are well tolerated, as stored inner thyroid iodide is adequate; however, in iodide-deficient areas, these physiological adaptations lead to significant changes in pregnancy. Hypothyroidism is widely prevalent in pregnant women and the rate of detection, especially in a developing country like India, has not kept pace with the magnitude of the problem. Since hypothyroidism is easily treated, timely detection and treatment of the dysfunction could reduce the burden of adverse fetal and maternal outcomes in pregnancy which are commonly encountered. Prevalence of overt thyroid dysfunction is 2–3% in pregnant women, subclinical dysfunction is 10%, while rate of autoimmunity is 5–10%[2].

Method

This study was performed in the Department of Obstetrics and Gynaecology at Jhalawar Medical College and SHKBM Hospital, Jhalawar. Study design: Prospective observational study

Inclusion Criteria

All pregnant women were admitted to SHKBM with a deranged thyroid function test.

- Pregnant women in the third trimester
- Singleton pregnancy

Exclusion Criteria

- Multiple pregnancy
- Known cases of other medical disorders like diabetes mellitus, hypertension, tuberculosis, cardiac and pulmonary pathology

Routine hematological parameters and estimation of T3, T4, and TSH were conducted. Patients with abnormal thyroid profiles were then evaluated for complications that could affect the mother or the fetus. The primary study variables included infertility, family history of thyroid disorders, menstrual history, recurrent abortions, mean T3, T4, TSH, hemoglobin levels, and maternal and fetal outcomes.

TSH was estimated using the Enhanced Chemiluminescence technique. Then, when TSH levels were abnormal, estimates of free T3 and free T4 were made. Cut-off values used for TSH were those indicated by the American Pregnancy and Thyroid Association: 1st trimester: 0.1–4.0mIU/L, 2nd trimester: 0.2–4.5mIU/L, 3rd trimester: 0.3 -5mIU/L. The normal free T4 level is 0.7 to 1.8 ng/dl and the free T3 level is 1.7 to 4.2 pg/ml. Patients with normal fT4 and high TSH were considered to have subclinical hypothyroidism (SCH); those with low fT4 and high TSH were considered to have overt hypothyroidism; those with normal fT4 and low TSH were considered to have subclinical hyperthyroidism; and those with highT4 and low TSH were considered to have overt hyperthyroidism[3].

Data management and analysis were done using Statistical Package for the Social Sciences (SPSS Version 25). The categorical variables were assessed using the Pearson chi-square test. The association of risk factors was calculated by binary logistic regression. The test was considered significant only when the p-value was less than 0.05. The study protocol was approved by the Scientific and Ethical Committee of the Institution. All the participants were also informed about the study procedure and the information that was required from them for the study. Written consent was obtained.

The outcome of hypothyroidism in pregnancy[4]

- Maternal:- Anaemia and CHF, pre-eclampsia, placental abnormalities, low birth weight infant, postpartum hemorrhage, myopathy.
- Foetal:- Cognitive impairment, neurological abnormalities, developmental abnormalities, and congenital hypothyroidism.
- Neonatal:- Hyperbilirubinemia and respiratory distress.

RESULTS

Out of the 198 women who underwent screening, 22 (11%) had abnormal thyroid function. Subclinical hypothyroidism is more prevalent during pregnancy, as shown by the prevalence of subclinical hypothyroidism, overt hypothyroidism, and subclinical hyperthyroidism, which were 5.9% (n = 12), 3.6% (n = 7), and 1.6% (n = 3) respectively. Women with subclinical hypothyroidism, overt hypothyroidism, and subclinical hyperthyroidism had mean serum TSH levels of 9.02 ± 1.25 mIU/ml, 11.60 ± 5.34 mIU/ml, and 0.09 ± 0.03 mIU/ml, respectively.

Women with subclinical hypothyroidism, overt hypothyroidism, and subclinical hyperthyroidism had mean serum fT3 levels that were, respectively, 3.07 ± 0.56 pg/ml, 0.88 ± 0.66 pg/ml, and 4.5 ± 0.40 pg/ml, respectively. Women with subclinical hypothyroidism, overt hypothyroidism, and subclinical hyperthyroidism had mean serum fT4 levels of 1.20 ± 0.30 ng/dl, 0.56 ± 0.24 ng/dl and 1.4 ± 0.10 ng/dl, respectively. (Table 1).

TABLE – 1

Thyroid status	Prevalence	Mean TSH mIU/L	Mean fT4 ng/dl	Mean fT3 pg/dl
Subclinical hypothyroidism	5.9%	9.02±1.25	1.20±0.23	3.07±0.56
Overt hypothyroidism	3.6%	11.60±5.34	0.56±0.42	3.07±0.56
Subclinical hyperthyroidism	1.6%	0.09±0.03	1.4± 0.10	4.5± 0.40

Anaemia was found in 23.6% of hypothyroid women, and the association between the occurrence of hypothyroidism and anemia was statistically significant. Also, 15.8% of women were found to have preeclampsia, and there was a statistically significant association between the presence of hypothyroidism and preeclampsia. There were 5.3% of hypothyroidism patients gave birth prematurely, but this was not significantly correlated with hypothyroidism. A significant correlation existed between LBW and hypothyroidism, with 31.6% of pregnancies being LBW. As a sign of fetal asphyxia, a cutoff value of 5 for a 1-min Apgar score was taken into consideration. Four (21.1%) of the hypothyroid women had babies with low Apgar scores, which were significant. Hypothyroidism was significantly associated with 41.1% of NICU admissions.

Table 2

OUTCOME	PERCENTAGE (%)	P VALUE
ANAEMIA	23.6%	<0.05
PRE-ECLAMPSIA	15.8%	<0.05
PRETERM	5.3%	>0.05
LOW APGAR SCORE	21.1%	<0.05
NICU ADMISSION	41.1%	<0.05
LOW BIRTH WEIGHT	31.6%	<0.05

DISCUSSION

Thyroid physiology changes significantly during pregnancy, and this has been recognized for the past 20 years. Untreated thyroid dysfunction during pregnancy is harmful to both the mother and the developing fetus.

While other authors have noted that anemia occurs in 26.3% of women with hypothyroidism, anemia was found in 23.6% of the women with hypothyroidism in the current study[5].

Pre-eclampsia was seen in 15.8% of the women with hypothyroidism in the current study. These findings are consistent with those of other studies, where preeclampsia was noted in 15.8% of hypothyroidism patients, 13.6% of SCH patients, and 14.7 of overt hypothyroidism patients[5,6,7].

Pre-term labor in subclinical hypothyroid patients is 3.1% in the study by Sreelatha S et al. and 5.3% in the current study[7].

In this study, 31.6% of women with hypothyroidism had LBW,

compared to 20% in a different study[8].

Thyroid dysfunction led to 41.1% of NICU admissions, which is comparable to the rates of 46.6 and 42%[9,8]. And 21.1% of infants born to hypothyroid mothers had low Apgar scores, compared to 20% in another study[6].

Pregnancy-related thyroid dysfunction that is left untreated increases the risk of stillbirth, low birth weight, fetal death, and miscarriages. In the above table 2 shows, there was a prevalence of pre-eclampsia and anemia. The harmful effects of thyroid dysfunction can also affect a child's early intellectual development, going beyond pregnancy and delivery. Therefore, any delay in hypothyroidism diagnosis and treatment may ultimately have an effect on the progeny's educational, socioeconomic, and public health outcomes.

CONCLUSION

Hypothyroidism has a strong correlation with anemia, pre-eclampsia, and neonatal morbidities. According to this study's findings, subclinical hypothyroidism affects the majority of pregnant women and is highly prevalent (11%). Major findings of this study include an association between maternal anemia, preeclampsia, the presence of LBW babies, low Apgar scores, and an increase in NICU admissions. There is a significant regional variation in the prevalence of thyroid disorders during pregnancy[10]. There is a paucity of data on the prevalence of thyroid disorders in Indian pregnant women due to thyroid profile not being done routinely till now as ANC profile and due to improper antenatal visit[11].

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