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Original Research Article

Study of hypothyroidism in pregnancy and it's fetomaternal outcome: a prospective study at tertiary care hospital

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ABSTRACT

Background: Pregnancy is a stress test of maternal thyroid function. The prevalence of thyroid dysfunction in pregnant women is high. This study is aimed to evaluate maternal and fetal outcomes in pregnant women with deranged thyroid profile. The relevance of this study is to document the association of hypothyroidism and its adverse effects on mother and fetus.
Methods: All pregnant mother included in the study are subjected to written consent and detailed history taking and fasting blood sample collection. Blood sample collected in fasting state, because lipidemic content in blood interferes with serum thyroid stimulating hormone (TSH) level if blood sample collected in the fed state.
Results: A total of 1000 cases were studied. Out of 1000 cases, 71(7.1%) cases found to be hypothyroid. Among them, 14 (19.4%) had overt hypothyroidism and 57 (80.5%) demonstrated subclinical hypothyroidism.
Conclusions: Prevalence of subclinical hypothyroidism is 5.6% in 3rd trimester of pregnancy. Anemia, pre-eclampsia, high caesarean rates and neonatal morbidities is significantly associated with hypothyroidism.

Keywords: Hypothyroidism and anemia, Pregnancy and hypothyroidism, Subclinical hypothyroidism, Thyroid dysfunction in pregnancy

INTRODUCTION

The second most common endocrinological disorder in pregnancy is thyroid dysfunction. The most common cause of hypothyroidism is primary abnormality in thyroid. In antenatal women most common causes are Hashimotos thyroiditis, iodine deficiency, radioactive iodine therapy and surgical removal of thyroid. The commonest cause of hypothyroidism is iodine deficiency. Hashimotos thyroiditis is the commonest cause in the developed countries. The incidence of overt hypothyroidism is 0.3 to 0.5% and subclinical hypothyroidism 2-3%.

Hypothyroidism during pregnancy has an adverse effect on both mother and child. Children born to untreated or undertreated mothers have profound effect on future intellectual development.[1] Production of thyroid hormones and iodine requirement both increases by approximately 50% during pregnancy as part of physiology.

Normal thyroid function is critical for normal functioning of the gonadal axis, thus important in maintaining normal reproduction. Gonadal steroid synthesis by oocytes depends on an adequate level of thyroid hormones. T3 modulates the regulating action of luteinizing hormone (LH) and follicle stimulating hormone (FSH) on steroid biosynthesis, thyroid hormones increase and enhance estrogenic responses**.** Dysthyroidism is associated with anovulatory cycles, subfertility or infertility Abortion rate as high as 60% in inadequately treated overt hypothyroids and 70% in subclinical hypothyroids. Matsua et al showed

that free T3 and free T4 values were significantly lower in women whose pregnancies terminated in abortions.

Thyroid hormone is essential for the normal development of the placenta. There is evidence that preeclampsia, placental abruption and preterm labour are all causatively linked to faulty early placentation. Thyroid hormone is important for normal neuronal migration, synaptic transmission and myelination during the early stages of neurodevelopment.

Pregnancy can be viewed as a state in which a combination of events concurs to modify the thyroidal status. There is change in the level of thyroxine-binding globulin, total thyroid-hormone level and change in the level of TSH during normal pregnancy.[2]

Thyroid dysfunction has varied impact on pregnancy outcome. The risk of miscarriage is increased in autoimmune thyroid disease. Thyroid dysfunction (TD) may be overlooked in pregnancy because of the nonspecific symptoms and hypermetabolic state of normal pregnancy. Severe maternal hypothyroidism can result in irreversible neurological deficit in the offspring. Graves' disease (GD) can lead to pregnancy loss as well as fetal thyroid dysfunction.

The prevalence of hypothyroidism in pregnancy is around 2.5% according to the Western literature.[3] The prevalence of GD is around 0.1–0.4% and that of thyroid autoimmunity (TAI) is around 5–10%.[4] There are a few reports of prevalence of hypothyroidism during pregnancy from India with prevalence rates ranging from 4.8% to 11%.[5]

Many changes occur in thyroid physiology during pregnancy. The cut-off values are changed during pregnancy for the diagnosis of hypothyroidism. The symptoms and signs also common for both the conditions. Severe hypothyroidism in pregnancy is not common because most of these women are infertile and they also have increased rates of abortions.

Complications of hypothyroidism in pregnancy

Fetal

Fetal complications include: spontaneous abortion, intrauterine growth restriction (IUGR), oligohydramnios, preterm delivery, fetal distress, and low birth weight

Maternal

Maternal complications include: pregnancy induced hypertension (preeclampsia, eclampsia), and placental abruption.

Starting thyroxine treatment in the 1st trimester (preferably prenatally) may decrease the incidence of complications. Starting treatment after completion of 1st trimester will not eliminate already established fetal neuro developmental delay, as during the first trimester the fetus depends completely on maternal thyroid hormone for the normal brain development.

METHODS

After taking institutional ethical committee clearance and taking informed written consent from the patients for participation, present study conducted in the department of obstetrics and gynaecology in Pannadhay Zanana Hospital, RNT Medical College, Udaipur, Rajasthan for period of one year from March 2021 to February 2022.

Design of study

It was a prospective study.

Sample size

1000 pregnant women were taken for the study.

Inclusion criteria

Patients of 1st and 2nd trimester pregnancy, singleton pregnancy, primigravida or multigravida, and known hypothyroid patients were included in the study.

Exclusion criteria

Patients with multi foetal gestation, known chronic disorders like diabetes and hypertension, liver disorders, renal disorders, and previous bad obstetric history with known cause, were excluded.

Methodology

All pregnant mother included in the study are subjected to written consent and detailed history taking and fasting blood sample collection. Blood sample collected in fasting state, because lipaemic content in blood interferes with serum TSH level if blood sample collected in the fed state.

Blood sample collected from the patients by venupuncture (2 ml), allowed to clot, and serum is separated by centrifugation at room temperature. The serum is stored at 2 to 8°C till its usage. The TSH is estimated by using enzyme-linked immunoassay (ELISA) method.

If serum TSH is abnormal, free T4 and free T3 is estimated. According to the biochemical values, those patients were divided into overt hypothyroidism, subclinical hypothyroidism, and euthyroid. Overt hypothyroidism and subclinical hypothyroidism patients were treated with L-thyroxine to maintain serum TSH near normal.

Serum TSH estimation will be repeat at 4- 6weeks interval. All the patients followed till delivery to know the

fetomaternal outcome. The normal patients with normal thyroid functions serve as controls.

RESULTS

A total of 1000 cases were studied. Out of 1000 cases, 71 (7.1%) cases found to be hypothyroid. Among them, 14 (19.4%) had overt hypothyroidism and 57 (80.5%) demonstrated subclinical hypothyroidism.

In Table 1, subclinical hypothyroid prevalence among the age group between 19-25 years (89.7%), and overt hypothyroid around 10.3%. The mean age of sub clinical hypothyroid population was 19-25 years in the current study. The mean age for overt hypothyroid was 35-43 years. Study by Ajmani et al found that maternal age was high in the overt hypothyroid with a mean age of 35±5 years. In our study, among 103 hypothyroid women, 26 (26.78%) were below 25 years as compared to 57 (58.71%) women above the age of 25 years.[6]

This shows increasing prevalence of hypothyroidism as maternal age advances. In the study of Akhter et al, it was observed that 62.1% of subclinical hypothyroid patients were in the 15-24 years age group and 66.7% of the overt hypothyroidism patients were in the 25–44-year age group.[7]

Table 1: Thyroid prevalence among different age groups.

Age group (years)	Thyroid status, N (%)		
	Sub-clinical hypothyroid-ism (N=57)	Overt hypothyroid-ism (N=14)	Total (N=71) N%
19-25	20 (86.95)	3 (13.05)	23
26-30	17 (80.95)	4 (19.04)	21
31-34	11 (78.75)	3 (21.42)	14
35-43	9 (69.23)	4 (30.76)	13

Table 2 shows that hypothyroidism in pregnancy has been associated with adverse maternal outcomes in observational studies including preeclampsia, placental abnormalities, preterm labour, miscarriages, and low birth weight.

Table 2: Distribution of hypothyroid women according to complications associated with pregnancy (N=71).

Complications	N%
Nil	50
Pre-eclampsia	8
Placenta previa	3
IUGR	4
GDM	1
Severe pre-eclampsia	2
Oligohydroamnios	2
Severe anemia	1

A study done by Rao et al demonstrates that hypothyroidism has a statistically significant relationship with recurrent pregnancy loss in the first trimester and suggests that diagnosis of hypothyroidism could help couples with recurrent pregnancy loss to have a successful outcome in subsequent pregnancies.[8]

DISCUSSION

This study was conducted in RNT Medical College Udaipur and attached hospital.

The purpose of the study was to follow the pregnancy outcomes in pregnant women with hypothyroidism to see whether they developed complications if left untreated and if adequate treatment altered the occurrence of complications.

The total number of pregnant women included in this study were 1000. 71 women who have been diagnosed as hypothyroid treatment started. All antenatal women were screened using TSH at their first booking visit during first trimester. Those who had an elevated TSH levels, were further tested with FT4 and started on treatment with levothyroxine irrespective of whether FT4 was elevated or not. The cut-off level for TSH was taken as 2.5 mIU/ml. Serum thyrotropin (TSH) level in early pregnancy is decreased because of thyroid stimulation from the weak TSH effects of HCG.

In a study by Green in 2005, truly normal range of TSH is defined as 0.5 2.5 mIU/ml.[6] So adequate replacement therapy should be given when TSH is above 2.5mIU/ml and/or with low T4, FT4 in pregnancy.[9] Overt hypothyroidism, subclinical hypothyroidism patients were treated with L Thyroxine in the dose of 1.20 µg/kg/day for subclinical hypothyroidism with TSH less than 4.2 mIU/l, 1.42 µg/kg/day with TSH greater than 4.2 to 10, and 2.33 µg/kg/day for overt hypothyroidism this dosing was based on a study by Abalovich and colleagues which was published in the journal of overt and subclinical hypothyroidism and which has been confirmed by numerous other studies according to the body weight to maintain serum TSH near normal TSH levels were repeated for these patients after initiating the treatment at 16 weeks, 20 weeks, then at 32 weeks and thyroxine dosage titrated accordingly.[10] Based on whether they were started on treatment before 10 weeks and given prompt dosage titration, or after 10 weeks they were grouped as those receiving adequate treatment and inadequate treatment. In my study out of 1000 cases, 71 (7.1%) cases found to be hypothyroid. Among them, 14 (1.4%) had overt hypothyroidism and 57 (5.7%) demonstrated subclinical hypothyroidism.

CONCLUSION

Thyroid hormone is essential for early placental development in pregnancy. Especially during the first twelve weeks of pregnancy the fetus entirely depends upon

the maternal thyroid hormone for the normal neural and skeletal development. Hence early diagnosis and adequate treatment of maternal hypothyroidism in pregnancy is essential in decreasing the incidence of complications like abortion, pre-eclampsia, IUGR, placental abruption, oligohydramnios and low birth weight which are associated with hypothyroidism. Inadequately treated hypothyroid women in my study group had 3-fold higher risk of developing preeclampsia. There was a significant increase in the incidence of abortion or fetal growth restriction in the inadequately treated group. There was no case of placental abruption in my study group. Oligohydramnios was found to occur more commonly in the inadequately treated group.

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Conflict of interest: None declared
Ethical approval: The study was approved by the Institutional Ethics Committee

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