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Original Research Article

The study of maternal and fetal outcome in pregnant women with thyroid disorders

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ABSTRACT

Background: Thyroid disorders are among the common endocrine disorders in pregnant women after diabetes mellitus. Several changes are observed in maternal thyroid function during pregnancy and failure to adapt to these physiological changes results in thyroid dysfunction. It is well established that not only overt, but subclinical thyroid dysfunction also has adverse effect on mother and the fetus, like miscarriages, preterm delivery, preeclampsia, eclampsia, polihydromnios, placental abruption, post-partum haemorrhage, low birth weight, neonatal hypothyroidism. Decreased availability of thyroid hormones may also impair neurological and intellectual development of foetus. With this background, we are conducting a study to know the effect of thyroid disorders on pregnancy and its maternal and the fetal outcome.
Methods: The present study was conducted in ESI Hospital Rajaji Nagar, Bangalore. It is a prospective study which involved 100 patients diagnosed to have thyroid disorder during their antenatal checkup in the first trimister. It also includes known cases of thyroid disorder. TSH level was estimated. If it is deranged, then FT3 and FT4 levels estimated. Patients were managed accordingly and followed till delivery. Their obstetric and perinatal outcomes were noted.
Results: In our study out of 100 cases, 96 cases are subclinical hypohyroid and 4 cases are subclinical hyperthyroid. Subclinical hypothyroidism in pregnancy are associated with abortions (2.1%), Anaemia (4.20%), PIH (14.7%), GDM (4.2%), Preterm labour (3.1%), oligohydromnios (16.67%), Lscs (22.9%), PPH (6.3%), LBW (21.9%), Hyperbilirubinemia (9.4%), NICU admission (14.6%), Which are co-relatine with other studies and hyperthyroid cases in our study were not sufficient for outcome analysis.
Conclusions: Thyroid disorders in pregnancy have adverse effects on maternal and fetal outcome emphasizing the importance of routine antenatal thyroid screening.

Keywords: Hyperthyroidism, Hypothyroidism, Oligohydromnios, PIH, Pregnancy, Thyroid dysfunction

INTRODUCTION

Thyroid disorders are the second most common cause of endocrine dysfunction in women of child bearing age after diabetes mellitus.[1-3] Development of maternal thyroid disorders during early pregnancy can influence the pregnancy outcome and fetal development. It is now well established that not only overt, but also subclinical thyroid dysfunction has significant adverse effects on pregnancy and fetal development. The adverse pregnancy outcomes include, miscarriage, pregnancy induced hypertension, and its more severe form pre-eclampsia, as well as placental abruption, anaemia, post-partum hemorrhage, and increased fetal morbidity and mortality. These obstetric complications contribute to overall increase in the frequency of adverse neonatal outcomes, which include preterm birth, low birth weight, increase

admission to neonatal intensive care and increase perinatal morbidity and mortality.[4-9]

Iodine deficiency significantly raises the risk of still birth and abortion amongst pregnant women and also leads to decreased availability of iodine to the fetus. It retards the neurological development in fetus and also impairs the cognitive development thereby leading to learning disability and lowered achievement motivation in later stages of childhood.[10,11]

Hyperthyroidism in pregnancy is less common than hypothyroidism. Untreated hyperthyroidism during pregnancy is associated with maternal and fetal morbidity. Neonatal Graves' disease can be seen because of the passage of TRAb to the fetus from the mother and may be seen in about 1-5% of the babies.[12]

In view of potential adverse outcomes associated with maternal thyroid disorders and obvious benefits of treatment, some expert panels have suggested routine thyroid function screening in all pregnant women.

The present study is being undertaken to know the maternal and fetal outcome in pregnant women with thyroid disorders. The objective of the study was to determine the maternal and fetal outcome in pregnant women with thyroid disorders.

METHODS

Study population

Pregnant women attending the antenatal OPD, registered since their first trimester, department of Obstetrics and Gynaecology, ESI Medical College, Rajaji Nagar, Bangalore. Source of the study: OPD and IPD records and the structured proforma. Sample size:100 patients

Inclusion criteria

- Singleton pregnancy between 6-12 weeks
- Primigravida / multigravida
- Known case of hypothyroidism / hyperthyroidism on treatment.

Exclusion criteria

- Multiple pregnancies
- Chronic hypertension and diabetes
- Bad obstetric history.

Design of the study: Longitudinal / Cohort study.

This is a prospective study involving 100 pregnant women attending the antenatal clinic, booked since their first trimester. Apart from the routine obstetrical investigations, TSH will be done as a screening test by chemiluminescence method. Estimation of free T3 (FT3) and Free T4 are advised if TSH is abnormal. Cut off values for TSH will be taken as per American Thyroid Association - 1st trimester: 0.1 -2.5 micro iu/L, 2nd trimester: 0.2-3.0 micro iu/L, 3rd trimester: 0.3 -3 micro iu/L. Those with the abnormal tests are categorized as Subclinical hypothyroid (Normal FT4 with high TSH), Overt hypothyroidism (Low FT4 with high TSH) and hyperthyroidism (High T4 with low TSH). Patients will be put on treatment, and thyroid function tests will be repeated every 6-8 weeks during pregnancy and drug dosages titrated accordingly. Patients will be followed up throughout the pregnancy.

Maternal outcomes are assessed by the following

- Abortion
- Anaemia
- Oligohydromnios
- Preeclampsia.
- Preterm delivery.

Fetal outcomes are assessed by the following

- Birth weight
- Apgar score
- Hyperbilirubinemia
- NICU admission
- Neonatal hypo/hyperthyroidism.

RESULTS

The present study was done in ESI Hospital, Rajaji Nagar, Bangalore. A total of 100 patients with thyroid disorder were started on treatment and TSH was repeated every 6-8 weeks with TSH and followed up till delivery and outcomes were recorded.

Table 1: Demographic profile.

Age distribution	Cases (n=100)
<20	01
20-25	34
25-30	49
30-35	14
>35	02

Table 2: Parity.

Parity	%
Primigravida	49
Multigravida	51

Our study had maximum number of pregnant women in the age group 25-30 years who had Thyroid disorder i.e. 49%. Least in the age group less than 20 years i.e. 1% and more than 35 years i.e. 2%. 34% women were in the age group 20-25 years who had thyroid disorder.

Our study had 49% of primigravida and 51% multigravidas with thyroid disorder.

Table 3: Type of thyroid disorder.

Types	Cases
Hypothyroidism	96
Hyperthyroidism	04

Out of 100 cases, 96 cases are hypothyroid. In our study, all hypothyroidism cases were subclinical and other 4 cases were subclinical hyperthyroidism.

Table 4: Known case of thyroid disorders versus newly detected cases.

Cases	%
Newly detected cases	58
Known cases	42

In our study 58% were newly detected to be having Thyroid disorder during pregnancy and 42% were known cases of thyroid disorder.

Table 5: Cases affected by thyroid disorder in pregnancy.

Cases	%
Complicated cases	62
Uncomplicated cases	38

In our study, thyroid disorder complicating pregnancy is 62% and 38% of the patients had uneventful pregnancy outcomes.

Table 6: Number of cases with 'N' number of complications.

Complication	Number
1 Complication	26
2 Complications	16
3 Complications	10
4 Complications	03
5 Complications	02
6 Complications	01

Number of complications

Out of 100 pregnant patients with thyroid disorder, 96 cases are associated with subclinical hypothyroidism and 4 are associated with subclinical hyperthyroidism.

- 1 pregnant women with thyroid disorder was associated with 6 complications
- 2 pregnant women with thyroid disorder was associated with 5 complications
- 3 pregnant women with thyroid disorder was associated with 4 complications
- 26 pregnant women with thyroid disorder was associated with 1 complication.

Table 7: Complications associated with 96 cases of subclinical hypothyroidism.

Complications	No.	(n=96%)
Abortion	02	2.1
Anaemia	04	4.2
oligohydromnios	16	16.7
Hypertensive disorders of pregnancy	14	14.7
GDM	04	4.2
PTL	03	3.1
LSCS	22	22.9
PPH	06	6.3

Maternal complications among 96 cases of S. Hypothyroidism. In our study abortion is associated with 2.1% cases, anaemia in 4.2% of cases, Oligohydromnios in 16.7% of cases, Hypertensive disorders in pregnancy in 14.7% of cases, GDM in 4.2% of cases, Pre-term labour in 3.1% of cases, PPH in 6.3% of cases and LSCS in 22.9% of cases.

Table 8: Fetal complications among 96 cases of S. Hypothyroidism.

Complications	No. of cases	%
Low birth weight	21	21.9
Hyperbilirubinemia	09	9.4
Nicu admission	14	14.6

Table 8 shows neonatal complications in hypothyroid patients. 21.9% of cases had babies with low birth weight. 9.4% of cases had babies with hyperbilirubinemia. and 14.6% had babies who had NICU admission. Neonatal TSH were normal in all babies we had only 4 hyperthyroid cases out of which only one patient had anaemia and oligohydromnios, second patient had low birth weight baby and 3rd patient had LSCS because of fetal distress and last patient had an eventful pregnancy outcome.

In Table 9, Age group of newly detected cases of hypothyroid and known cases of hypothyroid are compared. There is no statistical significance between both.

Table 9: Showing age group among newly detected and known cases of hypothyroidism.

Age group	Newly detected n (%)	Known cases %	Total %
00-20-year old	01 (1.9)	0 (0.0)	01 (1.0)
20-25-year old	22 (40.7)	10 (23.8)	32 (33.3)
25-30-year old	25 (46.3)	22 (52.4)	47 (49.0)
30-35-year old	06 (11.1)	8 (19.0)	14 (14.6)
>35	00 (0.0)	2 (4.8)	02 (2.1)
Total	54 (100.0)	42 (100.0)	96

Percent: (col). Chi2= 6.580 df (4) p= 0.1598

Table 10: Parity among newly detected cases and known cases of hypothyroidism.

Parity	Newly detected (%)	Known case (%)	Total (%)
Primigravida	29 (53.7)	18 (42.9)	47 (49.0)
Multigravida	25 (46.3)	24 (57.1)	49 (51.0)
Total	54 (100.0)	42 (100.0)	96

Percent: (col). Chi2= 1.112 df (1) p=0.2916

Table 11: Various outcomes in newly detected and known cases of hypothyroidism.

Outcome	Newly detected cases (%)	Known case (%)	Total	P value
Abortion	1 (1.9)	1(2.4)	2 (2.1)	0.8571
Anaemia	03 (5.6)	01 (2.4)	04 (4.2)	0.4400
Oligo	11 (20.4)	05 (11.9)	16 (16.7)	0.2695
PIH	40 (18.5)	04 (9.8)	14 (14.7)	0.2327
GDM	03 (5.6)	1 (2.4)	4 (4.2)	0.4400
Preterm labour	02 (3.7)	1 (2.4)	03 (3.1)	0.7117
PPH	04 (7.4)	2 (4.8)	6 (6.3)	0.5953
LBW	10 (18.5)	11 (26.2)	21 (21.9)	0.3670
Hyperbilirubinemia	6 (11.1)	3 (7.1)	9 (9.4)	0.5081
NICU ADM	9 (16.7)	5 (11.9)	14 (14.6)	0.5120
Neonatal hypo	0.0	0.0	0.0	

In Table 10, Parity among newly detected cases of hypothyroid with known cases of hypothyroid are compared. There is no statistical significance between both the groups.

In Table 11, Various complications between newly detected cases of hypothyroid and known cases of hypothyroid are compared and there is no statistical significance between both the groups.

Statistical analysis

The data was analyzed for mean, proportion and chi square was calculated for association. The P value =< 0.05 is considered as significant. Analysis was done using EPIDATA analysis software V2.

DISCUSSION

Thyroid disease is common in women of reproductive age, and it is most common endocrine disorder after diabetes. Subclinical hypothyroidism is the most common. Maternal thyroid deficiency even subclinical has been associated with adverse pregnancy outcome and may be improved by T4 replacement.[13] Fluctuations occur in T4 metabolism during pregnancy make it difficult to maintain meticulous normal thyroid harmone values during gestation in hypothyroid mothers.[14] Pregnancy cause increased thyroid gland vascularity, increased renal iodide clearance and iodide losses to the fetus.[15] Fluctuation in thyroxine metabolism that occurs during pregnancy may further impair maternal-fetal transfer of thyroxine despite apparently optimal thyroid status.[16]

About 10% of pregnant women who have TPO antibodies during early gestation are at increased risk of developing subclinical hypothyroidism during pregnancy and thyroid dysfunction post-partum. The latter condition occurs in 5-9% of women and 25-30% progress to permanent hypothyroidism.

In present study abortion rate is 2.1% which is co-relating with Tanuja PM, et al (1.7%).[17] Presence of antibodies to thyroid peroxiadase (TPO-AB) or thyroglobulin during pregnancy is associated with significant increment in miscarriages, premature deliveries, geastational diabetes, postpartum thyroiditis and permanent hypothyroidism.[18-21]

In present study GDM in subclinical hypothyroid patients is 4.2% and in a study done by Pavanaganga et al it is 6.4%.[22] The increase in preeclampsia incidence, of the pre-mature delivery, of the post-partum depression and haemorrhages could be explained through a maturation process of the placenta and they appear especially if there is a severe Hypothyroidism, but they have been also signaled in the cases of subclinical hypothyroidism.[18]

In our study PIH in subclinical hypothyroid patients is 14.7% which is co-relating with a study done by Ozdemir H et al (14.5%).[23] Thyroid hormones potentiate Beta adrenergic response by increasing the number of beta adrenoceptorswith an opposite action on alpha adrenergic receptors. In hypothyroid state, the density of alpha-1 adrenoceptors is increases while beta adrenoceptors are reduced on vascular beds. Action of alpha adrenoceptors mainly involves smooth muscle cell contraction, causing vasoconstriction in the blood vessel.

In present study anaemia in subclinical hypothyroid patients is 4.2% which is co-relating with a study done by Dr. Pavanaganga et al (5.08%).[22] In present study oligohydromnios in subclinical hypothyroid patients is 16.7% and in a study done by done by Pavanaganga et al, it is 8.35%.[22] In present study, pre-term labour in subclinical hypothyroid patients is 3.1% and in a study done by Sahu MT it is 4.75%.[6] In this study PPH in subclinical hypothyroid patients is 6.3% and in a study done by Mohammed et al it is 6%.[24] In current study, Primary LSCS in subclinical hypothyroid patients is 22.9% which is co-relating with a study done by Georgel M et al (20%).[25] It was more commonly because of fetal distress.

The utero placental interface is susceptible to both thrombosis and haemorrhge, particularly in association with structurally defective placentation. Several factors may mediate such pathogenesis: for ex tissue factor production in response to aberrant vascular endothelial growth factor and inflammatory cytokine release, which promotes thrombosis. In addition, shallow extravillous thromboblast invasion (EVT) may lead to placental ischemia and haemorrhage generating thrombin locally, which mediates the degradation of extracellular matrix, thus triggering pre-mature placental separation from the uterus.[21] In a study by Tanuja PM et al, abruption placenta rate is 0.3%, where as in this study we did not come across any patient with abruption.[17]

Reduced foetal thyroxine may cause disruption to the development of the pituitary-thyroid axis of the newborn, fetal pituitary GH secretion, vascular responsiveness and maturation, cardiovascular homeostasis in utero.[27-29]

These factors may be responsible for observation of reduced neonatal birth weight of offsprings born to mothers with inadequately controlled thyroid at initial presentation or at 3rd trimester. In present study LBW in subclinical hypothyroid patients is 21.9% which is co-relating with a study conducted by Sangeeta A et al (25%).[30]

Most of the low birth weight babies were due to PIH in mother, leading to IUGR and low birth weight. In present study hyperbilirubinemia in subclinical hypothyroidism is 9.4% which is co-relating with a study done by Georgel M et al (8%).[25] In present study NICU admission in subclinical hypothyroid patients is 14.6% which is co-relating with a study done by Meena DS et al (14.28%).[31]

All women with overt and subclinical hypothyroidism should be treated irrespective of TPO antibody positivity with LT4 during pregnancy to maintain serum TSH in the trimester specific goal range. It has been recommended to check the serum TSH level every 4 weeks during pregnancy so that appropriate dose adjustments can be made. The recommended therapy is oral LT4 which should be given on empty stomach (45 min before consumption of food, beverages, or other medications). In addition, calcium, iron, and prenatal vitamins supplement should be avoided within 4 hours of ingestion of LT4, as these can decrease the absorption of thyroxine. In a typical case dose requirement goes up as pregnancy advances as pregnancy is a hypermetabolic condition. Immediately after delivery, the requirement of thyroxine drops and women who were taking thyroxine prior to pregnancy will shift to their pre-pregnancy dose and those who started thyroxine in pregnancy will require half the dose they were taking just before the delivery.

In women who had started thyroxine in pregnancy for subclinical hypothyroidism, the medication can be stopped after delivery and thyroid balance reassessed again after 6 weeks and decision taken regarding continuation of treatment. Serum T3, T4 levels rise 30 minutes after delivery and persist for 5 days. This is due to TSH elevation caused be the stress of delivery. So new born screening should be done from cord blood immediately after delivery or 5 days after delivery.

Table 12: Complications present study (%) other studies.

Outcome	%	Literature
Abortion	2.1%	Tanuja PM et al (1.7%) Sangita A et al (5.5%)[17,30]
Anaemia	4.2%	Pavanaganga et al (5.08%), Sangita A et al (14.1%)[22,30]
PIH	14.7%	Leung (15%), Ozdemir H (14.5%)[23,32]
GDM	4.2%	Pavaganga et al (6.4%)[22]
Pre-term labour	3.1%	Sahu MT (4.7%), Sangita A et al (11.2%)[6,30]
Oligohydromnios	16.7%	Pavanaganga et al (8.3%)[22]
LSCS	22.9%	Georgel M et al (20%), Sangita A et al (16.6%)[25,30]
PPH	6.3%	Mohammed M et al (6%), Sangita A et al (5.5%)[24,30]
LBW	21.9%	Hareesh MV et, al (16.32%) Sangita A et al (25%)[30,33]
Hyperbilirubinemia	9.4%	George M et al (8%)[25]
NICU ADM	14.6%	Meena DS (14.28%)[31]

CONCLUSION

Thyroid testing is a must at first booking, ideally prenatally to prevent miscarriages. As fetus needs thyroxine for brain development, growth, and lung maturation, adequate replacement therapy should be done to keep TSH with in trimester specific reference ranges. Early and effective treatment of thyroid disorders ensures safe pregnancy with minimal maternal and fetal complication.

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