Original Research Article

Pregnancy with hypothyroidism and its adverse pregnancy outcomes

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Abstract

Background: Thyroid disorders are among the common endocrine disorders in pregnant woman after diabetes mellitus. Pregnancy is a stress test of maternal thyroid function. Several changes are observed in maternal thyroid function during pregnancy and failure to adapt to these physiological changes results in thyroid dysfunction. Subclinical hypothyroidism occurs in 10% of all pregnancies. Hypothyroidism has adverse effects on mother and fetus like anemia, pre eclampsia, preterm delivery, low birth weight and mental retardation of neonate. Decreased availability of thyroid hormones also impair neurological and intellectual development of the fetus. The relevance of this study is to document the association of hypothyroidism and its adverse effects on mother and fetus.

Materials and methods: This prospective observational study was carried out during the period January 2020 to December 2020 (12 months) at Government General Hospital (GGH), Rangaraya Medical College, Kakinada, Andhra Pradesh, India. Subjects of this study were 170 antenatal women in third trimester with singleton pregnancy admitted in the obstetric ward, and informed consent was obtained. Women were chosen irrespective of age, parity, residence and socioeconomic status. Women with multiple pregnancy, a known case of thyroid disorder, or any pre-existing medical disorder were excluded. Routine hematological parameters and estimation of T3, T4 and TSH was conducted.

Results: In this study out of 170 cases, anemia was seen in-15.29%, pre eclampsia in-11.77%, GDM in-4.11%, Oligohydramnios with IUGR in-5.88%, preterm labor in-3.53%, LSCS in-35.29% and IUFD in-0.59% of case and low birth weight (8.24%), Hyperbilirubinemia (3.53%) and NICU admissions (11.76%).

Conclusion: This Study concluded that hypothyroidism in pregnancy leads to preeclampsia, preterm labour, increases caesarean delivery, low birth weight and hyperbilirubinemia in neonates. Hence

effective treatment of hypothyroidism ensures safe pregnancy with minimal maternal and foetal complications.

Key words

Hypothyroidism, TSH, Maternal complications, Fetal complications, Cesarean sections.

Introduction

In women of child bearing age thyroid dysfunction is the 2nd most common cause of endocrine dysfunction after diabetes mellitus [1-3].

Development of maternal thyroid dysfunction during pregnancy can influence pregnancy outcome and fetal development. Thyroid physiology is perceptibly modified during normal pregnancy. These alterations take place throughout gestation help to prepare the maternal thyroid gland to cope up with the metabolic demands of the pregnancy.

The most notable change is the increase in thyroxin binding globulin (TBG). This begins early in the first trimester, plateaus during mid gestation and persists until shortly after delivery. This is due to stimulation of TBG synthesis by elevated maternal estrogen levels and more importantly due to reduced hepatic clearance of TBG because of estrogen induced sialylation [4]. Maternal thyroid hormone synthesis is also increased due to an accelerated renal clearance of iodide resulting from the increased maternal GFR.

A large plasma volume and thus an altered distribution of thyroid hormone, increased thyroid hormone metabolism in 2nd and 3rd trimesters due to a rise in placental type 2 and 3 deiodinases, increased renal clearance of iodide and higher levels of hepatic production of thyroxin binding globulin in the hyper estrogenic state of pregnancy are responsible for higher thyroxin requirements in pregnancy.

All these changes lead to an increase in the thyroid gland size in 15% of pregnant women which returns to the normal in postpartum period.

In 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 [5].

Hypothyroidism is widely prevalent in pregnant woman. Iodine deficiency significantly raises the risk of still birth and abortion among pregnant women and leads to decreased availability of iodine to the fetus. It retards the neurological development in fetus and also impairs the cognitive development, there by leading to learning disability and lowered achievement motivation in later stages of childhood [6].

Hypothyroidism in pregnancy lead to maternal complications like miscarriage, anemia, preeclampsia, placental abruption, preterm delivery, oligohydramnios, increased rates of cesarean delivery and PPH.

Fetal outcomes resulting from thyroid dysfunction are preterm birth, neonatal respiratory distress syndrome, low birth weight, perinatal morbidity and mortality, increased NICU admissions, hyperbilirubinemia and neuropsychological and cognitive impairment.

The objective of the study was to determine the maternal and fetal outcome in pregnant women with hypothyroidism.

Materials and methods

This was a prospective study involving antenatal women in the third trimester with hypothyroidism (TSH >=3.0 mIU /L) admitted in obstetric ward. Estimation for TSH was

conducted using the Enhanced Chemiluminescence method. Informed consent was obtained from all subjects. Subjects were chosen irrespective of age, parity, residence and socio economic status. Patients with deranged thyroid profile were subsequently assessed for maternal and fetal complications.

Study population

170 Antenatal women in the third trimester with hypothyroidism admitted in the obstetrics ward at Government General Hospital Kakinada, Andhra Pradesh.

Inclusion criteria:

1. Singleton pregnancy in third trimester admitted in the obstetrics ward
2. Primigravida/ Multigravida

Exclusion criteria:

1. Multiple pregnancy
2. Known Cases of hyperthyroidism
3. Pre existing medical disorders like Diabetes mellitus, cardiac and pulmonary disorder

Maternal outcomes are assessed by the following:

1. Anemia
2. Oligohydramnios
3. Preeclampcia
4. Preterm delivery

Fetal outcomes are assessed by:

1. Birth weight
2. NICU admission
3. Hyperbilirubinemia.
4. Low apgar score

Statistical analysis

The quantitative variables are presented by their frequency along with percentage.

Results

This study was conducted at Government General Hospital, Kakinada, Andhra Pradesh. A total of 170 antenatal women in the third trimester admitted in obstetrics ward that were identified to have hypothyroidism and were assessed for maternal and fetal outcome.

Our study had maximum number of pregnant women in the age group 26-30 years who had thyroid disorder i.e. 50% and least in the age group of <20 years i.e. 2.35% and more than 35 years i.e. 2.94%. 31.77% women were in the age group 20-25 years who had hypothyroid disorder (**Table – 1**).

Table - 1: Distribution of cases according to the age.

Age distribution	n=170	%
<20	4	2.35
20-25	54	31.77
26-30	85	50
31-35	22	12.94
>35	5	2.94

Table - 2: Distribution of cases according to the parity.

Parity	n=170	%
Primigravida	78	45.88
Multigravida	92	54.12

Table - 3: Distribution of cases according to mode of delivery.

Mode of delivery	No. of cases (n=170)	%
Vaginal route	102	60
LSCS	60	35.29
Forceps delivery	8	4.71

Table - 4: Cases affected by hypothyroidism in pregnancy.

Cases	No of cases (n=170)	%
Complicated cases	104	61.18
Uncomplicated cases	66	38.82

Our study had 45.88% of primigravida and 54.12% multigravida with hypothyroid disorder (**Table – 2**). LSCS was done in 35.29% of cases while 60% were vaginal delivery (**Table – 3**).

Table - 5: Distribution of cases according to Complications.

Complications	No. of cases (n=170)	%
Maternal complications	74	43.53
Fetal complications	30	17.65
Normal	66	38.82

Table - 6: Distribution of cases according to Maternal Complications seen in hypothyroidism.

Complications	No. of cases (n=170)	%
Anemia	26	15.29
Preeclampsia	20	11.77
GDM	07	4.11
Oligohydramnios with IUGR	10	5.88
Preterm Labor	06	3.53
GDM + Hypertension	03	1.76
PPH	01	0.59
IUFD	01	0.59
Normal	96	56.48

Table - 7: Distribution of cases according to fetal complications seen in hypothyroidism complicating pregnancy.

Complications	No. of cases (n=170)	%
Low Birth Weight	14	8.24
Hyperbilirubinemia	06	3.53
Low apgar	10	5.88
Normal	140	82.35

In our study, thyroid disorder complicating pregnancy was 61.18% and 38.82% of patients had uneventful pregnancy outcomes (**Table - 4**). Among hypothyroid complicating pregnancies, maternal complications were 43.53% and Fetal complications were 17.65% (**Table – 5**).

In this study, complications seen were anemia in 15.29% cases, Preeclampsia in 11.77% cases, GDM in 4.11% of cases, Oligohydramnios with IUGR in 5.88% cases, Preterm Labor in 3.53% cases, GDM + Hypertension in 1.76%, PPH in 0.59% and IUFD in 0.59 % Cases (**Table – 6**). Fetal complications were as per **Table – 7**.

Discussion

Thyroid disorder is the most common endocrine disorder after diabetes in women of reproductive age and the most common disorder is subclinical hypothyroidism.

Due to complex hormonal changes in pregnancy it is important to remember thyroxine requirements are higher in pregnancy.

Maternal thyroid deficiency even subclinical hypothyroidism has been associated with adverse pregnancy outcome and maybe improved by thyroxine replacement [6].

Pregnancy causes increased thyroid gland vascularity, increased renal iodide clearance and iodide losses to the fetus [7, 8].

Fluctuations in the thyroxine metabolism that occurs during pregnancy may further impair maternal fetal transfer of thyroxine in spite of apparently optimal thyroid status [9].

In the present study GDM in hypothyroidism cases is 4.11% and study done by Pavanaganga, et al. it is 6.4% [10].

In the present study preeclampsia hypothyroidism cases is 11.77% where it is 14.5% in a study done by Ozdemir H, et al. [11].
Thyroid hormones potentiate beta adrenergic response by increasing the number of beta adrenergic receptors with an opposite action on alpha adrenergic receptors. In hypothyroid state, the density of α-1 adrenergic receptors increases while beta adrenergic receptors are reduced on vascular beds. Action of alpha adrenergic receptors mainly involves smooth muscle cell contraction causing vasoconstriction In the blood vessel.

In the present study, anemia is seen in 15.29% of cases complicated by hypothyroidism where as it is 5.08% in study done by Dr. Pavanaganaga, et al. [10].

In present study Oligohydramnios with IUGR is seen in 5.88% of hypothyroidism cases where as it is 8.35% in a study done by Dr. Pavanaganaga, et al. [10].

In present study preterm labor in hypothyroid patients is 3.53% where as it is 4.75% in study done by Shau MT [12].

In Current study primary LSCS in hypothyroidism cases is 35.29% where as it is 22.9% in a study done by Georgel M, et al. [13].

Fetal Outcome

Low birth weight (LBW) is associated with hypothyroidism due to its association with preeclampsia. Reduced fetal thyroxine may cause disruption to the development of the pituitary-thyroid axis of the new born, fetal pituitary growth hormone secretion, vascular responsiveness and maturation and cardiovascular homeostasis in utero [14-16].

In the present study LBW was 8.24% where it is 25% in a study conducted by Sangeeta A, et al. [17].

11.76% hypothyroid mothers had their babies admitted in NICU where as it is 14.28% in a study done by Meena DS, et al. [18].

In present study hyperbilirubinemia in hypothyroid patients is 3.53% where it is 8% in study done George M, et al. [13].

In present study low apgar was 5.88% where it is 21.1% in a study conducted by Kalpana Mahadik, et al. [19].

Children born to mothers with hypothyroidism had significantly increased risk of impairment in IQ score, neuropsychological developmental indices and learning abilities.

Children born to untreated hypothyroidism women had an IQ score that was 7 points below the mean IQ of children born to healthy women and women given thyroxine supplementation.

All women with overt and sub clinical hypothyroidism should be treated irrespective of thyroid peroxidase antibody positivity with levothyroxine during pregnancy to maintain serum TSH in trimester specific goal range.

The recommended therapy is oral levothyroxine which should be taken on an empty stomach (45 minutes before consumption of food, beverages or other medications). In addition calcium iron and prenatal vitamin supplements should be avoided within four hours of ingestion of levothyroxine as they can decrease the absorption of thyroxine. In a typical case the dose requirement goes up as pregnancy advances as pregnancy is a hyper metabolic condition. In women who had started their thyroxine in pregnancy for subclinical hypothyroidism the medication can be stopped after delivery and thyroid balance reassessed again after six weeks and decision taken regarding continuation of treatment.

Serum T_3 T_4 levels rise 30 minutes after delivery and persists for five days. This is due to TSH elevation caused by stress of delivery. So new born screening should be done from cord blood immediately after delivery or five days after delivery.

Conclusion

The study concludes that hypothyroidism in pregnancy leads to anemia preeclampsia, preterm labor, increased cesarean delivery in mother and low birth weight, respiratory distress syndrome and hyperbilirubinemia in neonates. Hence effective treatment of hypothyroidism ensures safe pregnancy with minimal maternal and fetal complications.

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