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PREVALENCE OF THYROID DYSFUNCTION THYROID ANTIBODY POSITIVITY AND OBSTETRIC MPLICATIONS

Dr. Reshita[1] Dr. Meena Samant[2]
[1]Senior Resident, Department of Obs. and Gynae, PMCH, Patna
[2]Consultant and HOD, Department of Obs. and Gynae, Kurji Holy Family Hospital, Patna

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Corresponding author: Dr. Reshita
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Abstract
Background: Thyroid disorders are common among women of childbearing years. The prevalence of overt hyperthyroidism complicating pregnancy has been reported to range between 0.4% and 1.7%,and an estimated 2% to 3% of women are hypothyroid during pregnancy. Thyroid dysfunction is often overlooked in pregnant women, however, because of nonspecific symptoms and the hypermetabolic state of pregnancy**.**
Material and methods: This prospective study was carried out on outpatients and in patients of Department of Obstetrics and Gynaecology at Patna medical college and Hospital, Patna. 550women irrespective of their period of gestation on their first antenatal visit were studied and followed up till the time of delivery.
Conclusion: All the quantitative data such as age, gestational period, TSH, free T4, is expressed in terms of descriptive statistics for both group of women, namelywomen with thyroid disorders and women without such disorders.
Keywords: TSH, hyperthyroidism, pregnancy.

Introduction

Thyroid disorders are common among women of childbearing years. The prevalence of overt hyperthyroidism complicating pregnancy has been reported to range between 0.4% and 1.7%,and an estimated 2% to 3% of women are hypothyroid during pregnancy. Thyroid dysfunction is often overlooked in pregnant women, however, because of nonspecific symptoms and the hypermetabolic state of pregnancy.[1] Compounding the diagnosis further is the alteration in thyroid physiology that normally occurs during gestation. A clinician first must consider thyroid dysfunction a possibility, then differentiate normal physiologic changes from the true disease. Abnormalities in maternal thyroid function can adversely affect the fetus directly by the way of transplacental passage of abnormal maternal hormone concentrations, thyroid stimulating hormone (TSH) receptor antibodies, or prescribed antithyroid medications and indirectly by the way of altered maternal gravid physiology. The thyroid gland synthesises and releases thyroxine (T_4) and tri-iodothyronine(T_3) in a ratio of 4:1. Most circulating hormone is in the form of T4, with 99.7% being protein bound. Normal thyroid gland is able to compensate for increase in the thyroid hormone demands by increasing their secretion and maintaining the serum levels of free hormones within normal limits. However, in those situations in which there is a subtle pathologic abnormality of thyroid gland, such as autoimmune thyroiditis, normal increase in the production of thyroid hormone is not met. As a consequence, pregnant woman develop biochemical markers of hypothyroidism. Hypothyroidism is endemic in many parts of northern India. Hypothyroidism is defined as any elevation in serum TSH above 5mIU/l but recent studies have suggested lower TSH values in pregnancy, with upper limit of 2.5mU/L.[1] Normal TSH concentrations are dependent on gestational age and it is lower in first trimester as compared with second and third trimester of pregnancy. The prevalence of thyroid autoimmunity among euthyroid women of child bearing age is quite high. In a metaanalysis done by Prummel MF and Wiersinga WM, a clear association between the presence of thyroid antibodies i.e. thyroperoxidase antibody (TPO Ab) and miscarriages was found in both case control and longitudinal studies. thyroid abnormalities during pregnancy suggest that screening for thyroid dysfunction should be strongly considered. Hence, this study is being undertaken to find the prevalence of thyroid dysfunction and thyroid antibody positivity among pregnant women and to assess maternal and foetal outcome in the above group.

Objectives

To determine prevalence of thyroid dysfunction in pregnant women.To determine thyroid antibody positivity in pregnant women with hypothyroidism, To determine maternal and perinatal outcome in women with thyroid dysfunction and thyroid antibody positivity.

Review of Literature

Thyroid disorders are the second most common cause of endocrine dysfunction in women of childbearing age diabetes being the first. The diagnosis and management can be challenging as many of the symptoms of the disease are

common symptoms in pregnancy. Thyroid gland is a highly vascular organ, situated at the front and sides of the neck; it consists of right and left lobes connected across the middle line by a narrow portion, the isthmus. Its weight issomewhat variable, but is usually about 30 grams. It is slightly heavier in females, in whom itbecomes enlarged during menstruation and pregnancy. Thyroid gland develops from a median diverticulum which appears about the fourth week on the summit of the tuberculum impar. It grows downward and backward as a tubular duct, which bifurcates and subsequently subdivides into a series of cellular cords, from which the isthmus and lateral lobes of thyroid gland develop. The thyroid gland synthesises and releases thyroxine(T_4) and triiodothyronine(T_3) in a ratio of 4:1. [3] Production of thyroid hormones is controlled by serum thyrotropin (TSH) synthesised by the anterior pituitary gland and released in response to thyrotropin releasing hormone (TRH) secreted from neurons of paraventricular nucleus into the hypophyseal portalcirculation. In thyroid gland, iodide is converted back to iodine and is organified by binding to tyrosyl residues, which are a part of the glycoprotein, thyroglobulin. Binding of iodine to tyrosyl residues is dependent on the enzyme, thyroid peroxidase.[2] Binding of one iodine molecule to tyrosyl residue leads to the formation of monoiodotyrosine (MIT), whereas binding of two iodine molecule leads to diiodotyrosine (DIT). Coupling of two DITs yields T_4 and a DIT anda MIT forms T_3. Daily secretion rates of T_4 and T_3 are 90µg and 30µg, respectively. Half life of T_4 is 1 week and of T3 is 1 day. TRH and TSH synthesis and release are regulated by negative feedback of circulating thyroidhormones. Most circulating hormone is in the form of T4, with 99.7% being protein bound. The main protein involved is thyroxine-binding globulin (TBG). Hypothyroidism is defined as any elevation in serum TSH above 5mIU/l but recent studies have suggested lower TSH values in pregnancy, with upper limit of 2.5mU/L.[1] Normal TSH concentrations are dependent on gestational age and it is lower in first trimester as compared with second and third trimester of pregnancy.

Material and methods

This is a prospective study that involved, total 550 antenatal women, both outpatients and inpatients of Obstetrics and Gynaecology Department at Patna medical college and Hospital, Patna, Bihar. A detailed history with special reference to previous history of thyroid dysfunction and complete examination was conducted at first antenatal visit.

Inclusion Criteria

The study included antenatal women in any trimester on their first antenatal visit. General physical examination was done for all patients. Pelvic examination and ultrasound examination were done. Details of pregnancy regarding gestational age, weight gain, BMI and menstrual history were recorded.

Exclusion Criteria

Patients on treatment for thyroid dysfunction.

Blood sample was collected in bottles obtained from the biochemistry department. Under all aseptic precautions 5ml of venous sample was drawn from the antecubital vein. Sample was transported to biochemistry laboratory immediately. Serum was separated and used for processing thyroid function test and thyroperoxidase antibody.

Serum concentrations of TSH, free T_4 (FT_4) were measured by fully automated chemiluminescent immunoassay, run on the analyzer Roche Cobas 6000 : e - 601 (Roche Diagnostic Ltd.) Serum concentration of thyroperoxidase antibody (TPO Ab) was also measured by electrochemiluminiscent immunoassay on fully automated analyzer Roche Cobas 6000 : e - 601 (Roche Diagnostics Ltd.) results obtained by using trimester specific reference range were analysed. Various parameters- incidence of thyroid dysfunction, TPO positivity, mode of delivery, antenatal and perinatal complications have been compared.

Results

The study included 550 antenatal women in any period of gestation on their first visit . Details of pregnancy regarding gestational age, BMI and menstrual history were recorded. Features suggestive of thyroid disorder such family history of thyroid disorders (first degree relatives), previously diagnosed thyroid disease, patients on thyroid medication and presence of goitre was looked for. All these patients were tested for thyroid function test and thyroperoxidase antibody on their first visitin antenatal clinic. All the patients diagnosed to have thyroid dysfunction and thyroid antibody positivity were followed up jointly in endocrinology and antenatal clinic.

Table 1:

STATUS	N = (550 -12) 538	Percentage
Euthyroid	332	61.71
Hypothyroidism	203	37.73
Hyperthyroidism	3	0.56

Out of the 550 subjects, 12 were known hypothyroid. Out of the 538 subjects , 332 (61.71%) were euthyroid, 203 (37.73%) were hypothyroid, 3(0.56%) were hyperthyroid when trimester specific range for TSH was used.

Table 2:

TPO	Hypothyroid	
	No	Percentage
0 - 35	192	95.5
> 35	9	4.6
Mean	21.03	
SD	63.99	
p – value	0.760	

Out of the 201 subjects in the hypothyroid group, 192 (95.5%) were TPO negative, with value less than 35, 9 (4.6%) were TPO positive, with value morethan 35.

Table 3: Distribution of Subjects According to Menstrual History

MenstrualHistory*	Euthyroid (n= 332)		Hypothyroid (n= 215)		Hyperthyroid (n= 3)	
	N	%	N	%	N	%
Normal	327	98.5	202	94	3	100
Irregular	5	1.5	13	6	-	-
p - value	**0.014**					

Out of the 332 subjects in the euthyroid group 327(98.5%) had normalmenstrual history and 5(1.5%) had irregular menstrual history.Out of the 215 subjects in the hypothyroid group 202(94%) had normalmenstrual history and 13(6%) had irregular menstrual history.Out of the 3 subjects in thehyperthyroid group all subjects (100%) had normal menstrual history.In the euthyroid group out of 332 subjects, 10(3%) delivered < 30 weeks of gestation, 45(13.6%) delivered between 30 - 35 weeks, 246(74.1%) delivered between 35 – 40 weeks of gestation and 30(9%) delivered at > 40 weeks of gestation. In the hypothyroid group out of the 215 subjects, 12(5.6%) delivered < 30 weeks of gestation, 25(11.6%) delivered between 30 – 35 weeks, 162(75.3%) delivered between35 – 40 weeks of gestation and 16(7.5%) delivered at > 40 weeks of gestation. In thehyperthyroid group out of 3 subjects, 2(66.7%) delivered between 30 – 35 weeks,1(33.3%) delivered between 35 – 40 weeks of gestation.

In the euthyroid group perinatal complications observed were 26(7.8%) had hyperbilirubinaemia, 20(6%) had foetal distress, 49(14.8%) had low birth weight ,31(9.3%) had MSAF, 13(3.9%) underwent IUFD and 1(0.3%) had septicaemia. In the hypothyroid group perinatal complications observed were 28(13%) had hyperbilirubinaemia, 27(12.6%) had foetal distress, 29(13.5%) had low birth weight, 20(9.3%) had MSAF, 14(6.5%) underwent IUFD.In the hyperthyroid group perinatal complications observed were 1(33.3%) had foetal distress.

Discussion

Thyroid dysfunction (hyper- and hypothyroidism) is recognized with increasing frequency in the 12 months following delivery, or following spontaneous or medically induced abortions. Most of these cases are due to intrinsic thyroid dysfunction. The clinical course is not uniform in majority of patients. In about one third of cases, mild symptoms of hyperthyroidism develop between 2 and 4 months postpartum. On physical examination goitre is felt which is firm and non tender. Thyroid tests are in hyperthyroid range and thyroid antibodies, anti TPO antibody titres, are elevated in most cases. Spontaneously, without specific therapy, hypothyroidism develops in a few months, with spontaneous recovery and return to euthyroid state by 7 to 12 months following delivery. It has been suggested that some ultrasonographic changes of thyroid gland, such as hypoechogenicity, are typical of postpartum thyroiditis and may aid in the diagnosis of syndrome. There are some features that may predict the development of postpartum thyroiditis- presenceof goitre and high titres of thyroid antibodies in the first half of pregnancy, episodes of postpartum thyroiditis in previous pregnancies, hypothyroidism antedating pregnancy, and a strong family history of autoimmune thyroid disease. An increased risk of developing Grave's hyperthyroidism in the postpartum period is recently reported, particularly in women from 35-39 years of age. The risk was estimated to be 5.6 times greater when compared with nulliparous women. Antithyroid peroxidise antibody is the best available screening tool for postpartum thyroiditis. Antithyroid peroxidise positive women should have a serum TSH performed at 6 and 9 months postpartum, After 24 weeks gestation : Fine needle aspiration biopsy may be postponed until after delivery, unless there is strong suspicion of malignancy. Suppressive therapy with thyroxine to prevent further growth of the lesion , although considered controversial by some, may be considered. Thyroid cancer discovered during pregnancy is not more aggressive, however, than that diagnosed in a similar aged group of non

pregnant women, leading experts to advocate postponing definitive surgery until after delivery in most patients. A retrospective study of pregnant women with differentiated thyroid cancer found no difference in either recurrence or survival rates between women operated on during or after their pregnancy. The concentrations of Thyroid Stimulating Immunoglobulin may remain elevated, in spite of maternal euthyroidism. The concentration of these IgG immunoglobulins is low early in normal pregnancy, reaching a level in the foetus similar to that of the mother around 30 weeks' gestation. Therefore, the symptoms of foetal hyperthyroidism are not evident until 22 to 24 weeks of gestation. When TSH levels are present in high concentrations, This complication should be easily avoidable with proper management of maternal hyperthyroidism. The importance of identifying subclinical hypothyroidism and thyroid autoimmunity in early pregnancy is thus obvious as it is likely to have a profound influence on the outcome of pregnancy. Thyroid autoimmunity not only doubles the incidence of pregnancy loss but alsoposes an increased risk for obstetric complications – intrauterine fetal demise, gestational hypertension, placental abruption and poorer perinatal outcome. Thyroid autoantibodies have therefore been suggested as one of the markers of at risk pregnancy.

Conclusion

All the quantitative data such as age, gestational period, TSH, free T4, is expressed in terms of descriptive statistics for both group of women, namely women with thyroid disorders and women without such disorders. In our study mode of delivery was affected by thyroid dysfunction diagnosed during pregnancy. Rate of caesarean section in hypothyroid group was 46.5% and it was found to be statistically significant , **(p – value 0.025),** the most common indication for caesarean section being foetal distress.

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