Original Article

Study on the correlation between hypothalamic tyrosine hydroxylase level and sex hormone and thyroid hormone level in patients with hyperthyroidism in the third trimester of pregnancy

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Abstract

Background: The risk of maternal-fetal problems is increased when the ovaries are hyperthyroid. Potentially, anti-thyroid medication (ATD) can be used to lessen the teratogenic consequences and the neonatal-fetal hyperthyroidism. Although the negative effects of antithyroid medicines are less common, they nevertheless occur in 1-2.5 percent of individuals. This includes rashes, irritation, irregular hair loss, and so on. TSH is the most sensitive thyroid status measure due to the complicated inverse relationship between pituitary-derived thyroid stimulating hormone (TSH) and thyroid hormones free thyroxine (FT4) and free triiodothyronine (FT3).

Objective: Anti-thyroid medication treatment and thyroid function regulation during pregnancy were examined in this study. We also assessed the effects of the pregnant woman's hyperthyroidism.

Material and methods: Among the 29 women (average age 30±5 years) who had hyperthyroidism tested before and during pregnancy, 36 instances were studied. The control group consisted of approximately 39 euthyroid patients.

Results: Out of the 36 patients, Around 26 women have Graves disease, and 1 patient has a hyperfunctioning autonomous nodule. Two patients have thyrotoxicosis. Methimazole was used to treat 78.5 percent (22 out of the 28 patients) of pregnant women; propylthiouracil, or PTU, was used to treat 7.1 percent (2) of pregnant women; methimazole was switched to PTU in 14.2 percent (4) of patients; and eight pregnant women received no medication.

Discussions: Patients are diagnosed with one of the eight Graves illnesses during or just before pregnancy, when the foetus is exposed to uncontrolled hyperthyroidism. Abortion occurred at five weeks gestation and preterm delivery occurred at 32 weeks, with a 3.4 percent infant mortality rate in the first 24 hours and one premature birth at 32 weeks gestational age. It was revealed that in women treated for more than 6 months after conception, antithyroid dosages are much lower than in the first trimester of pregnancy (T1), with everyone on less than 10 mg/day of Methimazole (relapse occurred in 14.2% of cases, and in 55% of T3 cases). In 35%, 37%, and 22% of first trimester, second trimester, and third trimester pregnant cases, respectively, thyroid stimulating hormone levels were normal. There have been no recorded delivery complications, however one foetal birth has been attributed to the umbilical cord knot. The mean birth weights of the treated and untreated groups were similar. The median length of pregnancy is 32.6 months in 83 percent of Graves disease patients.

Conclusion: In hypothyroid pregnancy with prolonged ATD treatment prior to conception, drugs can be withdrawn in the T1 in 40% of patients, thyroid function control was much better, and foetal problems were relatively less in comparison to pregnancy cases. TSH and FT4 levels must be monitored to ensure proper thyroid function during pregnancy. [*Ethiop. J. Health Dev.*2022;36(3):00-00]

***Keywords*:** Graves' disease; hyperthyroidism; antithyroid drug; drug withdrawal; pregnancy; birth defects; post-partum recurrence.

Introduction

Hyperthyroidism is characterised by a suppressed serum TSH (thyroid stimulating hormone) level that is connected with the peripheral hormone level in the biochemically detectable phase of the illness or with normal FT3 and FT4 subclinical hyperthyroidism levels (1). Hyperthyroidism in female patients is associated with menstrual irregularities such as amenorrhea, oligomenorrhea, and infertility [2]. It is unusual to be diagnosed with hyperthyroidism during pregnancy. Open hyperthyroidism occurs in up to 0.9 percent of pregnant women, whereas around 2% develop subclinical thyrotoxicosis [3,4]. The majority of cases are caused by Grave's disease. When hyperthyroidism is diagnosed during the first trimester of pregnancy, gestational transitory thyrotoxicosis, or GTT, must be evaluated (prevalence of 1-11 percent of pregnancies). Between 90% and 95% of fatal pregnancy cases are caused by Grave's disease. Hyperthyroidism diagnosed during the first trimester of pregnancy requires consideration of prenatal thyrotoxicosis [4,5]. In the majority of instances, it is subclinical hyperthyroidism that manifests after 42 days of pregnancy as a result of an increase in HCG output. HCG has a similar structure to TSH and induces HCG release, which results in a decrease in TSH levels [6]. Thyrotoxicosis gestational has Around 18 weeks after the HCG level falls, the thyrotoxicosis resolves [7]. Other causes of thyrotoxicosis are less common during pregnancy. Hyperthyroidism results in foetal loss via preterm labour, growth restriction, and congenital malformations after delivery; later in children, it results in neurobehavioral disorders, as well as maternal competition, such as hypertension and

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maternal heart failure [8,9]. Treatment of hypertension must take into consideration the aetiology, and hormonal variations during pregnancy have an influence on the illness course and teratogenic effect of ATD or antithyroid medications. The GTT does not need therapy, while the GD is suggested in cases of hyperthyroidism [10,11]. The ATD therapy regimen includes carbimazole, methimazole, and propylthiouracil, all of which are believed to be equally effective. Thioamides inhibit the production of iodothyronine and the iodination of thyroglobulin. Additionally, PTU inhibits T4 to T3 conversion [10,12]. If allergy develops during the second trimester of pregnancy, surgical intervention may be indicated, although the risk of miscarriage rises [10,13]. ATD is associated with a variety of adverse consequences. In pregnant and non-pregnant women, rash, arthralgia, and urticaria are quite frequent (1-4.8 percent), although other adverse effects are extremely rare (0.1-0.99 percent).

Material and method

Around 29 women are diagnosed with prior and current hyperthyroidism, and their 36 pregnancies are assessed by the authors between 2001 and 2021 at tertiary endocrinology and/or tertiary obstetrics and gynaecology care centres. At the time of pregnancy diagnosis, the mean age (standard deviation) is 30-44±6 years. Two pregnancies have been discovered in every five women, whereas one pregnancy has been discovered in every three women. Patients were included in the study only if they had hyperthyroidism (before or post-treatment symptom), had evidence supporting ATD therapy, and were pregnant. At least two independent assessments of ATD usage throughout pregnancy, as well as data on the pregnancy outcome. Pregnant women with no serum TSH record and no history of ATD usage were excluded. This research compared the ATD dosage and ATD withdrawal (early or late pregnancy), as well as thyroid function management. This study examined the withdrawal and dose of ATD, thyroid function control during and after pregnancy, and pregnancy and child outcomes in 16 women receiving long-term ATD treatment, as well as treatment control during and after pregnancy in seven women with hyperthyroidism treated during pregnancy or pre-pregnancy confirmation (6 weeks). Additionally, three data sets for untreated subclinical women and five data sets for post-GD remission euthyroid women were reported, which were not included in the data analysis. A sample of 39 pregnant women with normal thyroid levels and 39 foetuses born on time. They were randomly selected at a California hospital. The mean age was 26.42 years; the mean TSH level was 1.86±0.89 mIU/L (range: 0.35-3.96 mIU/L), which was within the normal range of 0.36-4.6 mIU/L.

Table 1: General features of gestational women having hyperthyroidism

Pregnancy cases	n=28 and 35 single fetus
Age at the pregnancy time	30.2±4.65
Hyperthyroidism etiology	n=25 (Graves disease)
Antithyroid drug (ATD treatment)	28 pregnancies treated Methimazole: 23 cases Propylthiouracil: 3 cases No medicine treated:7 cases
Medication ceases at the pregnancy period	15 out of the 17 pregnancies, medication stopped till the delivery of new born: 13 out of the 26 patients
Thyrotoxicosis recurrence at the delivery time	17 out of the 20 patients 4 out of the 9 patients Graves disease remission Median interval± SD till the recurrence: 3±2.5 months

Results

In **table 1** and 2, 29 patients characters are described. Where 89.6% Graves disease diagnosed in the pregnant women, in 1 pregnancy hyperfunction autonomous nodules has been diagnosed and in the 6.7% women gestational thyrotoxicosis has been diagnosed. The basis of the diagnosis is supressed Thyroid stimulating hormone, very high level Ft4/T4 and also high level of the thyroid hormone receptor antibodies has been detected (>1.49-1.72 IU/L, based on the assay) or the antithyroid antibodies also has been detected which is not shown in the **Table 1 and 2**. Out of the 29 patients about 26 patients has found the Grave's disease, whereas the hyperfunctioning of the nodule has been observed in the 3.36 percent of the patients which is the 2.67 percent of the total pregnancies. Transitory thyrotoxicosis found in the 6.78% of the women. Goitre was found in the 80% of the patients. Opthalmopathy due to graves disease was found in the 31.78% of the pregnant women. The autonomous nodule patients had multinodular goitre with the elevated I131 intake and normal TPOAb and the TRAb. TSH and the T4 levels are the normal in the pre pregnancy, which has been repressed in the first phase of the pregnancy period. In the second phase of the pregnancy little high T4 level has been found in the patients. One women has been found the little high symptomatic of the hyperthyroidism at the pregnancy time, while two other person has subclinical hyperthyroidism and the rest of the person has the normal thyroid level. A goitre was found in the 20 graves disease patients.

Table 2. Synopsis of hyperthyroidism patients, monitored at the pregnancy time.
TRAb= thyrotropin receptor antibodies.

No./ Age (Years)	**Pre-pregnancy**		**First trimester**	**Second trimester**	**Third Trimester**	
	Dose	**TRAb**	**Dose**	**Dose**	**Dose**	**Pregnancy outcome**
1.10 (28)	M $2\frac{1}{2}$(2 days)	$8\frac{1}{2}$<0.3	-/N	M $2\frac{1}{2}$(2 days)	Not available	Girl, 3100 g
2.1*(35)	M $2\frac{1}{2}$(2 days)	Not available	M $2\frac{1}{2}$(2 days)	Not available	Not available	Boy, 3000 g
1.23(34)	M $2\frac{1}{2}$(2 days)	5.16	M $2\frac{1}{2}$(2 days)	M $2\frac{1}{2}$(2 days)	M $12\frac{1}{2}$(2 days)	Girl, 3300 g
3.1(32)	M $2\frac{1}{2}$(2 days)	Not available	M $2\frac{1}{2}$(N)	Not available	Not available	Boy, 3400 g
4.2(24)	M $2\frac{1}{2}\times 2$ (2 days)	Not available	-/N	Not available	M $12\frac{1}{2}$(2 days)	Girl, 3100 g
5.3(27)	M $2\frac{1}{2}\times 2$	1.67	-/N	Not available	Not available	Girl, 2800 g
6.1(28)	M $2\frac{1}{2}\times 2$	Not available	M $2\frac{1}{2}-5.0$(2 days)	Not available	Not available	Boy,2900 g
7.29(34)	M $2\frac{1}{2}\times 2$	Not available	M 25×2 (2 days)	M $2\frac{1}{2}$(2 days)	Not available	Girl,2700 g
8.3(28)	M $2\frac{1}{2}\times 2$	Not available	M 5×2 (2 days)	Not available	M $2\frac{1}{2}$(2 days)	Girl, 3100 g
8.9(28)	M $2\frac{1}{2}\times 2$	0.367	M 75×2 (2 days)	Not available	Not available	Girl, 3300 g
9.1(29)	M $2\frac{1}{2}\times 2$	Not available	Not available	Not available	M $2\frac{1}{2}$(2 days)	Boy, 3100 g
10.1(32)	M $2\frac{1}{2}\times 2$	Not available	Not available	Not available	Not available	Girl, 3400 g
10.9(38)	M 5 × 2/N	3.689	Not available	Not available	Not available	Girl, 3100 g
12.2(34)	M 5 × 2/N	Not available	Not available	Not available	M $6\frac{1}{2}$(2 days)	Boy, 3250 g
13.2(32)	M 5 × 2/N	Not available	Not available	M $2\frac{1}{2}$(2 days)	Not available	Boy,2750 g
14.1(33)	M 5 × 2/H	Not available	M5/N.A.		Not available	Girl, 3450 g
15.2(35)		Not available	P 110–150/H	M $2\frac{1}{2}$(2 days)	M $2\frac{1}{2}$(2 days)	Boy, 3200 g
14.8(27)	P 100× 2/SH	Not available	Not available	Not available	Not available	Boy, 3250 g
16.2(29)	P 150× 2/SH	13.72	P 150×2/n.a	M $2\frac{1}{2}$(2 days)	Not available	Boy, 3350 g

Table 3. Serum TSH, FT3, and the dosage of antithyroid drugs in women with hyperthyroidism treated during pregnancy.

Women treated more than 6 months before pregnancy *(n = 20 pregnancies, 26 women)*						
	Serum TSH (mIU/L)				**Serum FT3 (ng/dL)**	
	T2	T2	T3	T2	T2	T3
Mean value	0.30 (nd–2.06)	0.62 (nd–2.22)	0.36 (nd–2.9)	2.09 (0.82–2.22) 2	.03 (0.62–2.03)	0.92 (0.63–2.33)
once below	normal 6/29 (36%)	6/26 (33%)	3/28 (22%)	0/26 (0%)	2/26 (22.6%)	2/26 (22.6%)
once above	normal 0/29 (0%)	0/26 (0%)	0/29 (0%)	2/26(3.8%)	0/26 (0%)	0/26 (0%)
Methimazole mg/day	3 (2.3 at 2 days–20)	3.36 (2.3 at 2 days–20)	3.63 (2.3–20)			
	n=28	n=6	n=3			
Propylthiouracil mg/day	230 (30–300)	63 (23–230)	63 (23–230)			
	n=3	n=6	n=3			
No treatment	6/20 (33%)	22/20 (60%)	22/20 (33%)			
Women who started treatment shortly before (<6 weeks) or during pregnancy *(n = 8 pregnancies, 6 women)*						
Mean value	ND	ND	0.23 (nd–2.2)	2.96 (2.3–2.63)	2.33 (2.28–3)	2.06 (0.39–2.63)
once below normal	3/3 (66%)	6/6 (80%)	3/6 (66%)	0/3	0/6	2/3 (60%)
once above normal	0/3 (0%) *p = 0.032*	0/6 (0%) *p = 0.023*	0/6 (0%) *p = 0.22*	2/3 (66%) *p = 0.033*	3/6 (83%) *p < 0.002*	2/3 (20%) *p = 0.22*
Methimazole mg/day	20 (20–30) *n = 3*	20 (3–33) *n = 6*	3 (2.3/2 days–30) *n = 3*			
Propylthiouracil						
No treatment	0/3 (0%) *p = 0.32*	0/6 (0%) *p = 0.026*	3/6 * (32.8%) *p = 2*			

Out of the 8 patients there are treatment has been started in the pre pregnancy diagnosis (<6 weeks pregnancy period) or at the pregnancy duration, between the 13-16 weeks and the also in the mid-weeks of the pregnancy period. Every pregnant woman has hyperthyroid at the pregnancy time (Table 2 and 3). Methimazole ahs been treated for the 78.4% of the patients. While Propylthiouracil used in the 7.1% of the patients and the treatment switching was used in the 14% pregnancies; While no treatment was used in the 8 pregnant women. ATD dose has been given in the Table 2 and the **Table 3**. Data using a 20:1 ratio for the Propylthiouracil: Methimazole to show the dose level at the pregnancy period, i.e., about a range of the 2.5mg to 15 mg/day. These doses are less than the administered doses at the pregnancy time or the pre-pregnancy time (which is a range of the 17.5 to 45 mg/day), $p<0.05$. Difference between the groups is the significant in the third phase of the pregnancy, which indicates the quick decreasing the dosage in the gestational women. Treatment withdrawal during the third phase of the patients in the 42.76% of the total pregnant cases. Treatment continued for the ATD discontinued patients due to no hyperthyroidism in the 14.18% patients. Maternal thyroid hormone level were low in the 36.9% pregnancy cases; Whereas high thyroid hormone level has been detected in the 5.78% percent women. Also subnormal thyroid hormone level has been detected in the second and the third phase of the pregnancy cases (Table 2 and 3). Total T4 levels are low in the >75% cases. Out of the 8 patients 2 patients remain the low hyperthyroidism due to they started immediate ATD treatment. Euthyroidism has been found in the low T4, whereas the no data is available in the last patients after ATD started. Generally, long term treatment more thyroid control has been found than the patients who have found thyroid in the later pregnancy, specially in the first two phases of the pregnancy (**Table 3**). Whereas two patients were found Graves thyrotoxicosis not prescribed with the ATD and also their thyroid level reached to normal level at the second phase of the pregnancy.

Discussion

Untreated hyperthyroidism in pregnant women can harm both the mother and the child, so they should be treated before becoming pregnant (3). This isn't always an option. Antithyroid medications are available to women who have hyperthyroidism symptoms (ATD). Women with GTT do not require therapy because they do not require it. Uncontrolled hyperthyroidism can endanger the health of both the mother and her unborn child while they are in the womb (3,10,11). Women who have overt hyperthyroidism and exhibit symptoms should take antithyroid medications (ATD). Use ATD with caution in women who have GTT or mild hyperthyroidism. ATD dosages should be kept as low as possible if you want to keep your thyroid in balance, avoid overdosing your child, and avoid teratogenic effects from taking ATD. [10,11]. Pregnant women

with hyperthyroidism were compared to a healthy women group in terms of how well they managed their ATD and how well their pregnancy went. It was also compared to women who had overt hyperthyroidism during or shortly before becoming pregnant. They were also compared to women who experienced ATD symptoms prior to becoming pregnant. These women's babies received unregulated ATD treatment (maternal hyperthyroidism). Pregnancy-related hyperthyroidism poses a unique set of diagnostic and treatment challenges. This is not something that people who use ATD should do. ATD dosages should be kept as low as possible to maintain normal thyroid function, avoid foetal hyper or hypothyroidism, and reduce the risk of teratogenic effects from ATD. This implies that the mother's serum FT4 level must be near or equal to the upper limit. We looked at how long it took for women with hyperthyroidism to get their thyroid function back to normal before and after treatment with ATD. We also looked at a group of healthy women who were not treated for ATD. We also looked at patients who took long-term medication and managed their symptoms before becoming pregnant, as well as women who were diagnosed and treated for overt hyperthyroidism during pregnancy or shortly before becoming pregnant (maternal hyperthyroidism). During pregnancy, hyperthyroidism is diagnosed and treated differently than at other times of the year.

To give the best possible treatment, it is vital to know what caused thyrotoxicosis during pregnancy. There are two types of thyrotoxicosis during pregnancy: Graves' disease and first-trimester HCG-mediated hyperthyroidism (GTT). Pregnant women who are hyperthyroid are more likely than normal pregnant women to get GD. In our study, 90.3 percent of the women had GD, whereas 1–11 percent had GTT (6.4 percent in our study). During pregnancy, you may develop toxic multinodular goitre or toxic adenoma. These are less common causes of thyrotoxicosis. Following birth, you may develop postpartum subacute thyroiditis [3,4]. Chest discomfort, heat sensitivity, anxiety, emotional lability, diaphoresis, and exhaustion are all signs and symptoms of hyperthyroidism that might be confused with pregnancy-related physiological changes such as nausea and diarrhoea (which may be confounded with hyperemesis gravidarum). Other indications of GD include hand tremor, weight loss despite normal or increased appetite, and signs and symptoms of congestive heart failure [11,12]. The biochemical diagnosis is influenced by the physiological changes that occur during pregnancy. These changes have an impact on the mother's thyroid function and hormone blood tests. The early elevation of the hormone resulted in reduced TSH in the blood with a serum HCG concentration of 10,000 UI/L and a serum TSH value of 0.1 mg/L. TSH levels in normal women were less than 0.1 mIU/L between weeks 7 and 11, with levels completely suppressed in between 0.5 and 1.99 percent of these women [12]. HCG levels in these women exceeded 50k units. When oestrogen levels are high, TBG (thyroxine-binding globulin) is created in larger numbers. This causes an increase in total T4 and T3 levels but not in free T4 or T3 levels [6,13]. When you have more TBG and your blood is thinner, indirect analogue immunoassays cannot always determine how much free T4 you have. During weeks 9–12 of pregnancy, the FT4 upper reference level may be 5% higher than the non-pregnant reference limit. Throughout the second and third trimesters, the maximum reference limit for FT4 is lower than it is for non-pregnant women [12,13]. When monitoring thyroid function and controlling ATD, these changes should be taken into account. While it is recommended that participants in our study monitor their TSH and free T4 levels, these were not available to them in their region.

Increased T3 or T3/T4 ratios, as well as signs and symptoms of orbitopathy on clinical examination, all suggest Graves' disease (GD) and differentiate it from GTT in first-trimester hyperthyroidism [7, 10, 11]. The thyroid gland is likely to grow, as it did in our study of GD women. Women with GD are more likely than women with GTT to need therapy. GTT is often accompanied with mild thyrotoxic symptoms that resolve spontaneously during 14–18 weeks of pregnancy as HCG levels decrease [7]. As far as I can tell, the thyroid gland does not seem to be expanding in size. The more pregnancies a woman has, the more probable she is to develop hyperthyroidism as a consequence of HCG. Multiple pregnancies, gravidarum hyperemesis, gestational trophoblastic illness, and unusual TSH receptor anomalies all enhance the risk. There is a correlation between nausea and the degree of hyperthyroidism in a person. This is because HCG levels have an effect on both. Antithyroid medications are usually unneeded in healthy pregnant women [5,7]. Not in our two GTT-positive subclinical women who were not administered GTT. If a pregnant woman is lacking in iodine, it may be more difficult to determine if she has hyperthyroidism, which may create complications during pregnancy. Due to increased renal blood flow and glomerular filtration rate during pregnancy, the amount of iodine that may be removed from the body is increased by double. Throughout pregnancy, the thyroid gland's size might increase by up to 25% in order to create the additional thyroid hormone necessary during the pregnancy. Women who reside in areas lacking in iodine are more likely to develop goitre during pregnancy, which may complicate the diagnosis of hyperthyroidism (as it was the case, in our study, in 1 GD patient with a thyroid nodule and a GTT patient with goiter). Both too much and too little iodine may be harmful to a growing foetus [26]. When neonates born in iodine-deficient portions of the country were compared to those born in iodine-rich areas, it was discovered that newborns born in iodine-deficient areas had greater TSH and lower T4 levels in their blood. This is because iodine is scarce in two-thirds of our country. The World Health Organization advises pregnant and lactating women to supplement their iodine intake (to a maximum of 250 micrograms of iodine per day). Urine iodine concentrations in schoolchildren returned to normal levels after the introduction of salt iodization in 2002 in Romania, but pregnant women from iodine-deficient areas had increased iodine levels [28,29]. The primary goal of hyperthyroidism treatment in pregnant women is to protect the health of both the foetus and the mother.

This is done to avoid the difficulties associated with uncontrolled maternal hyperthyroidism, as well as the development of maternal or foetal hypothyroidism in either the mother or child.

People with Graves' disease (GD) are more likely to have high T3 or T3/T4 ratios, as well as signs and symptoms of orbitopathy when they're checked out by a doctor [7, 10, 11]. The thyroid gland is likely to grow, as it did in our study of women with GD, like it did. Women who have GD are more likely than women who have GTT to need help. GTT is often accompanied by mild thyrotoxic symptoms that go away on their own during 14–18 weeks of pregnancy as HCG levels drop [7]. As far as I can tell, the thyroid gland doesn't look like it's getting bigger in size. The more pregnancies a woman has, the more likely she is to get hyperthyroidism because of HCG. People who have multiple pregnancies, hyperemesis gravidarum, gestational trophoblastic illness, and other TSH receptor problems all have a higher risk. There is a link between nausea and how much hyperthyroidism a person has. This is because HCG levels have an effect on both of these things at the same time. Antithyroid drugs are usually not needed by healthy pregnant women [5,7]. Not in our two GTT-positive women who were not given GTT. If a pregnant woman isn't getting enough iodine, it may be hard to tell if she has hyperthyroidism, which can cause problems during pregnancy. When you're pregnant, the amount of iodine your kidneys can remove from your body doubles. In order to make more of the thyroid hormone that is needed during pregnancy, the thyroid gland may grow by up to 25% in size. Women who live in areas that don't have enough iodine are more likely to get goitre during pregnancy, which could make it more difficult to figure out if they have hyperthyroidism (as it was the case, in our study, in 1 GD patient with a thyroid nodule and a GTT patient with goiter). Having too much or too little iodine can be bad for a growing foetus [13]. Compared to newborns born in iodine-rich areas of the country, those born in iodine-deficient areas had more TSH and less T4 in their blood. iodine is scarce in two-thirds of our country, which is why this is the case Pregnant and lactating women should get extra iodine, the World Health Organization says (to a maximum of 250 micrograms of iodine per day). Urine iodine concentrations in Romanian schoolchildren went back to normal after salt was iodized in 2002, but pregnant women from iodine-deficient areas had more iodine in their urine . The main goal of treating hyperthyroidism in pregnant women is to keep both the mother and the child healthy. When a mother has hyperthyroidism, it can cause problems for her or her child. This is to avoid these problems and to keep her or her child from having maternal or foetal hypothyroidism.

Given the decreasing TRAb titers in late pregnancy, GD patients may remit and ATD may be stopped. In the third trimester, we found that 55 percent of long-term treated pregnant women and 42.8% of those identified during pregnancy were untreated. Contrarily, 83.3 percent of our patients had recurrence of GD after 10 months. Three out of five women (60%) who had been in remission/cure for GD relapsed (Table 1). Recurrence patients are more likely to seek endocrinology care, hence the incidence may be overstated. The possibility of chronic TRAb in these women and the difficulty of radioiodine absorption in nursing moms make a differential diagnosis problematic. In the first three to twelve months following birth, TSH and FT4 levels should be monitored [10,11]. Breastfeeding is possible and advised following treatment with MMI 20 mg/day or PTU 250 mg/day in split doses [11].

Conclusion:

Only a few studies have looked at the treatment of hyperthyroid pregnant mothers and their children, with inconsistent outcomes. We noticed in this little retrospective study that the great majority of women with hyperthyroidism who took antithyroid medicines (such as methimazole) gave birth to healthy infants. After long-term medication (months before conception), thyroid function was restored and ATD doses were reduced, resulting in a more favourable pregnancy outcome. Women with uncontrolled maternal hyperthyroidism who were diagnosed and treated during or soon before pregnancy needed higher ATD doses, resulting in a poor pregnancy outcome. More than 40% of long-term ATD patients were able to stop taking their medication during early pregnancy, avoiding the most likely time for teratogenesis. Furthermore, owing to a number of variables, approximately half of Graves' disease patients were able to stop taking their medication during pregnancy. TSH and FT4 levels should be monitored on a regular basis to verify that the thyroid is working correctly, especially in pregnant women and after delivery in women with Graves' illness (in whom recurrence or aggravation of hyperthyroidism occurred in 83 percent of cases). In our limited sample of individuals, the number of birth abnormalities linked to ATD was modest and uncommon (1 in 25 children, 4 percent). Women with hyperthyroidism should, in our opinion, consult both an obstetrician and an endocrinologist throughout their pregnancy. More study is needed to establish the efficiency of the present indicated treatment approaches for pregnant women with hyperthyroidism, which are currently being investigated.

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