

Relation of thyroid function and gestational hypertension

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Abstract

Background: Gestational hypertension is associated with multiple maternal and fetal complications. This study aimed to find the relation between thyroid function and gestational hypertension.

Methodology: A case control study that included 100 patients, (47) normotensive compared with (53) hypertensive pregnant women.

Results: Most of those with blood group (-O) had hypertension 3(75%), followed by those with blood group (-A) 2(66.7). Mother age was significantly lower among healthy women 28 ± 6 , than those who had, mild HT 31.4 ± 6 , moderate HT 34.6 ± 6 , and severe HT 32.8 ± 3.8 .

Increasing parity was significantly associated with gestational HT, 1.5 ± 1.5 for healthy compared with 1.9 ± 1.6 , 3.5 ± 2.1 , 2.8 ± 1.8 for mild, moderate and severe gestational HT. TSH level was significantly higher 2.8 ± 4 , 1.5 ± 0.8 , 2.8 ± 2.1 among those who had mild, moderate, and severe HT, as compared with normotensive pregnant ladies 1.7 ± 1 .

S. FT3 level 3.7 ± 1.7 , 3.3 ± 1.7 , 3.1 ± 1.1 was significantly decreased among those with mild, moderate and severe gestational HT respectively compared with normotensive (4.4 ± 1.6).

Conclusion: Gestational hypertension was significantly associated with increased levels of TSH, and decreased FT3 levels, and non-significantly with increased S.FT4.

Key words: gestational hypertension, hypothyroidism. Thyroid dysfunction, preeclampsia

Introduction

In a physiological process like pregnancy, there will be an alteration in different organ systems to supply adequate nutrition to the fetus and these include circulatory, metabolic, and hormonal alterations [1]. In pregnancy there is an increased metabolic need, for which thyroid hormone changes in economy and in hypothalamic - pituitary -thyroid axis regulation [2, 3]. Although there is well-documented knowledge about thyroid functions during normal pregnancy, there is a scant information regarding thyroid functions in complicated pregnancy.[4] Hypertension is one of the pregnancy physiological adaptations that develop various natures of disorders. Gestational hypertension has its onset from 20 weeks of gestation. Regarding clinical presentation may be as the followings; only hypertension (gestational non-proteinuric hypertension) or preeclampsia (hypertension with proteinuria and multiorgan dysfunction); and eclampsia if seizures in addition to preeclampsia there are seizures. [5,6] WHO, and American College of Obstetricians and Gynecologists provided the definition of gestational hypertension that also recommended by the National High Blood Pressure Education Program Working Group on High Blood Pressure in Pregnancy.[7–9]

Around 10% of all pregnancies complicated by Hypertension that cause an elevated risk of adverse fetal, neonatal and maternal outcomes including prematurity, perinatal death, intrauterine growth restriction, acute hepatic or renal hepatic failure, haemorrhage weather antepartum or postpartum type and maternal death. [10,11] Globally, hypertensive disorders are complicating 5–20% of pregnancies and its incidence differs from 2-8% of pregnancies in developed world reaching more than 10% in developing countries.[9,12] Gestational hypertension is the 3rd leading cause of maternal death globally. In pregnancy, there is an elevated thyroid hormone demand and an elevated iodine uptake and thyroid hormones synthesis. Activity of thyroid gland in normal pregnancy undergoes many changes. [4]There is mild hyperthyroxinemia in normal pregnancy, while in gestational hypertension (GH) females have an elevated incidence of hypothyroidism, (an elevated TSH). [13,14]

According to a lot of reports there is proved that gestational hypertension is found to affect levels of thyroid hormones.[15,16] Normal thyroid hormones pattern during pregnancy is very essential for fetal tissues differentiations. Thyroid hormones pattern may be disorderly affected in hypertensive pregnancy in gestational hypertension.[17,18]

There are a number of adverse outcomes associated with maternal thyroid dysfunction during pregnancy. [19]Increases maternal TSH levels has been associated with an elevated risk of prematurity, abruption of placental, fetal death, and impaired development of neurological system in the child.[15,20] Still there is need to study the thyroid hormone changes specifically that accompanying gestational hypertension have yet to be worked

out in Iraqi population. The current study was done to assess thyroid functions in GH, because it will enable to understand and address thyroid crises in GH for still better management.

Patients and method

This research was done in gynecology and obstetrics, Gynecology and Obstetrics Department / Azadi Teaching Hospital after taking permission from Kirkuk health directorate, committee of medical research and Gynecology and Obstetrics Department. The study involve 100 t pregnant women with no history of thyroid disease before and through pregnancy, aged 18–40 years, having diagnosed gestational hypertension, had systolic BP ≥ 140 mmHg and diastolic BP ≥ 90 mmHg in 3rd trimester of gestation on two occasions at least 6 hour apart. Assessment of gestational age was based on the date of last menstrual period, proved by early pelvic examination, and verified by first trimester or early second-trimester ultrasound. The following cases were excluded from the study; mothers with known history of chronic hypertension, renal disorders, cardiovascular diseases, diabetes, any metabolic disorder that may threat mother or fetus and history of any medication that might affect the thyroid function.

Forty seven (47) normotensive pregnant females were involved as controls compared with 53 pregnant women with gestational hypertension. The blood sample was immediately transferred to plane serum vials and kept for 30 min to clot, and centrifuged at 3,000 rpm for 10 min. The clear serum was pipetted out in three separate 1.5 ml vials. The processed samples were stored at -20°C until used for hormone assays. Laboratory work was performed at a proved private laboratory.

Patients from private clinics and hospital admission fulfilling the criteria of inclusion were included in this study after a written informed consent. Detailed history and examination was done. Data was recorded on a questionnaire and analyzed using SPSS-25. The significance of differences between the groups was analyzed by independent t-test and one way ANOVA, and $p < 0.05$ was considered statistically significant.

Results

There is no significant relation between mother job and gestational induced hypertension, as shown in Table 1, (P value > 0.05).

Most of the pregnant women with blood group (-O) had hypertension 3(75%), followed by those with blood group (-A) 2(66.7%), this relation was statistically not significant (p value > 0.05), as shown in Table 2.

There is significant increase in BP with increasing age of mother, healthy 28 ± 6 , mild HT 31.4 ± 6 , moderate HT 34.6 ± 6 , severe HT 32.8 ± 3.8 . Increasing gravidity also

Table 1. The relation between mother job and gestational induced hypertension

Job	Blood Pressure				Total
	Normal	Mild Hypertension	Moderate Hypertension	Severe Hypertension	
House Wife	41	13	17	15	86
	47.7%	15.1%	19.8%	17.4%	100.0%
Employed	6	3	3	2	14
	42.9%	21.4%	21.4%	14.3%	100.0%
Total	47	16	20	17	100
	47.0%	16.0%	20.0%	17.0%	100.0%

$\chi^2 = 0.44$, $df=3$, P value=0.93 (not significant)

Table 2: The relation between mother blood group and gestational induced hypertension

Blood Group	Blood Pressure				Total
	Normal BP	Mild Hypertension	Moderate Hypertension	Severe Hypertension	
A+	11	4	7	8	30
	36.7%	13.3%	23.3%	26.7%	100.0%
B+	11	5	1	1	18
	61.1%	27.8%	5.6%	5.6%	100.0%
AB+	6	2	1	0	9
	66.7%	22.2%	11.1%	0.0%	100.0%
O+	15	4	7	6	32
	46.9%	12.5%	21.9%	18.8%	100.0%
A-	1	0	0	2	3
	33.3%	0.0%	0.0%	66.7%	100.0%
B-	1	1	0	0	2
	50.0%	50.0%	0.0%	0.0%	100.0%
AB-	1	0	1	0	2
	50.0%	0.0%	50.0%	0.0%	100.0%
O-	1	0	3	0	4
	25.0%	0.0%	75.0%	0.0%	100.0%
Total	47	16	20	17	100
	47.0%	16.0%	20.0%	17.0%	100.0%

$\chi^2 = 28.5$, $df=21$, P value=0.126 (not significant)

Table 3: The relation of clinical and biochemical characteristic with gestational hypertension

	Normal Blood Pressure		Mild Hypertension		Moderate Hypertension		Severe Hypertension		ANOVA
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	
Age in years	28.0	6.0	31.4	6.0	34.6	6.0	32.8	3.8	0.000*
Gestational Age of Pregnancy/weeks	35.0	3.8	33.8	3.3	33.8	4.3	34.4	4.0	0.618
Gravida	2.7	1.6	2.9	1.6	5.2	2.7	4.3	1.8	0.000*
Parity	1.5	1.5	1.9	1.6	3.5	2.1	2.8	1.8	0.000*
Abortion	0.2	0.7	0.3	0.6	0.7	1.1	0.5	1.2	0.204
TSH Mic International Unit/ mL	1.7	1.0	2.8	4.0	1.5	0.8	2.8	2.1	0.047*
FT3 ng/dL	4.4	1.6	3.7	1.7	3.3	1.7	3.1	1.1	0.015*
FT4 ng/dL	11.9	2.05	11.7	1.6	12.8	3.3	12.5	2.3	0.373

*significant

significantly associated with gestational HT, 2.7±1.6 for healthy compared with 2.9±1.6, 5.2±2.7, 4.3±1.8 for mild, moderate and severe gestational HT, this relation was statistically significant as shown in table (3). Increasing parity was significantly associated with gestational HT, 1.5±1.5 for healthy compared with 1.9±1.6, 3.5±2.1, 2.8±1.8 for mild, moderate and severe gestational HT, this relation was statistically significant. Increasing TSH level was associated with gestational HT significantly, as compared with normotensive pregnant ladies 1.7±1 for healthy compared with 2.8±4, 1.5±0.8, 2.8±2.1 for mild. Decreased S. FT3 level associated significantly with gestational HT, 4.4±1.6 for healthy compared with 3.7±1.7, 3.3±1.7, 3.1±1.1 for mild, moderate and severe gestational HT, this relation was statistically significant, as shown in Table 3.

Discussion

Overt or subclinical thyroid dysfunction is associated with miscarriage, gestational hypertension, placental abruption, postpartum hemorrhage anemia and increased fetal morbidity and mortality. [21, 22, 23]

In this study there was significant difference in age of study groups, the hypertensive group older than the normotensive group. This goes with what find previously by Abdulslam K. and Yahaya IA, who found significantly increased mother age among those with gestational hypertension. [24]

Increasing TSH level was significantly higher among mothers with gestational Hypertension, as compared with normotensive pregnant ladies 1.7±1 for healthy compared with 2.8±4, 1.5±0.8, 2.8±2.1 for mild. This goes with a study In India done by Pasupathi P. et al found a significant

increase in S.TSH level among preeclampsia pregnant women 5.24 ± 2.58 compared to normal pregnant women 3.89 ± 2.32 .[25]

In Nigeria Abdulslam K. and Yahaya IA also found a significant increase in TSH among hypertensive group 2.1 ± 1.7 as compared with normotensive group 1.6±1. [15] Saki F. and et al in Iran found that 75% of the pregnant women with hypothyroidism (clinical or sub-clinical hypothyroidism) had preeclampsia. [26]

Decreased S. FT3 level associated significantly with gestational HT, healthy pregnant women mean S.FT3 was 4.4±1.6 compared with 3.7±1.7, 3.3±1.7, 3.1±1.1 for mild, moderate and sever gestational HT. this finding supported by previous studies, Pasupathi P. et al 3.57 ± 1.21 normotensive pregnant, 2.72 ± 1.15for preeclampsia women was significantly lower in preeclampsia than in normally pregnant women.[25]

In Nigeria reported non- significant decrease from 6.9±2.6 among normotensive to 6.4±2.7 among hypertensive group.[24]

Pasupathi P. et al found that FT4 was non significantly higher among preeclampsia women 2.42 ± 0.75 than normal pregnant 2.38 ± 0.99.[25]

Reduced extra-thyroidal conversion of T4 to T3 may be the cause of the higher T4 levels and lower T3 levels in preeclampsia.[25]

Johns L E and et al found that FT4 was inversely associated with fetal weight and growth, [27] gestational hyper tension associated with decreased fetal growth and weight, therefore we are in need of research to know the

relation between the gestational hypertension fetal growth and FT4 level.

Preeclampsia is pregnancy-induced autointoxication with multisystem disorders; the most affected organs are brain, liver, and kidneys. Functional disorders in these organ systems are evident in preeclampsia [13] However, the liver and kidneys are the most important organs in peripheral deiodination (conversion of T4 to T3) and in the maintenance of normal blood levels of T4 and T3. This is why involvement of liver and kidneys in preeclampsia is likely to change serum T4 and T3 levels. In some other studies, investigators have observed that preeclamptic women may be affected by a variety of conditions. These include systemic illnesses, protein-energy malnutrition, starvation, anorexia nervosa, Cushing's syndrome, and excessive steroid therapy. When the women have developed such systemic disorders, the extra-thyroidal deiodination of T4 to T3 has been reduced. Due to wide range of normal limits, however, the differences in T4 and T3 usually neither exceed normal limits nor produce significant metabolic changes. [28]

In previous studies an association between hypothyroidism and preeclampsia was found, hypothyroidism may be the cause of reversible hypertension in the pregnant and non-pregnant population. [29,30] Hypothyroidism can cause vascular smooth muscle contraction both in systemic and renal vessels, which leads to increased diastolic hypertension, peripheral vascular resistance, and decreased tissue perfusion. [30, 31]

In rare cases, proteinuria may be severe enough to cause thyroxine and thyroid binding globulin loss in a way that couldn't be compensated by the body, resulting in thyroid dysfunction. [32, 33]

Conclusion

Gestational hypertension was associated with increased levels of TSH, and decreased FT3 levels significantly, while FT4 was none significantly increased, even though it stile within border normal ranges.

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