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Original Article

Comparison of Thyroid Dysfunction in Women with or Without Pregnancy-Induced Hypertension: A Comparative Cross-Sectional Study

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ABSTRACT

Pregnancy-induced hypertension has been linked with altered thyroid function, yet local comparative data remain limited. **Objective:** To compare the prevalence of thyroid dysfunction in women with or without pregnancy-induced hypertension. **Methods:** This comparative cross-sectional study was performed at the Department of Obstetrics and Gynecology, Combined Military Hospital, Multan, for six months. A total of 208 pregnant females, 18-45 years of age, with singleton pregnancies beyond 20 weeks of gestation were enrolled. Out of these, 104 had pregnancy-induced hypertension, and 104 were normotensive. Women with chronic hypertension or pre-existing thyroid disease were excluded. Serum free T3, T4, and TSH were measured from a single laboratory, and thyroid dysfunction was defined using standard biochemical cut-offs. Quantitative variables are summarized as median with interquartile ranges and compared using the Mann-Whitney U test. In contrast, categorical variables are compared using the chi-square test, with $p<0.05$ taken statistically significant. **Results:** The median age was 22.5 (21-39) years, and 65.4% (n=136) were below 30 years of age. Baseline characteristics, including age, gestational age, gravidity, and obesity, were comparable between the groups. Overall, thyroid dysfunction was identified in 15.9% (n=33) of the participants, with subclinical hypothyroidism 6.7% (n=14), being the most frequent abnormality. Women with pregnancy-induced hypertension had a significantly higher proportion of thyroid dysfunction compared to normotensive women (25.0% vs. 6.7%, $p<0.001$), and all categories of thyroid dysfunction were more frequent in the hypertensive group. **Conclusion:** Thyroid dysfunction is more prevalent among women with pregnancy-induced hypertension.

INTRODUCTION

High blood pressure and proteinuria are the hallmarks of pregnancy-induced hypertension (PIH), which typically manifests after 20 weeks of pregnancy [1]. Excreting more than 300 mg of protein in a urine sample collected for 24 hours, a constant protein level of 30 mg/dl in random urine samples, or a protein-creatinine ratio of at least 0.3 in random urine samples are all indicators of proteinuria [2]. PIH accounts for 5-14% of all maternal deaths and neonatal morbidity and mortality, and it complicates 2-8% of pregnancies [3]. During pregnancy, the thyroid gland experiences normal physiological changes; if these changes are incompatible, the thyroid gland becomes dysfunctional [4]. Maternal thyroid hormones are essential to maintain systemic homeostasis and have been linked with a range of health-associated impacts on mothers and infants [5]. The overall prevalence of overt or subclinical hyperthyroidism during pregnancy is between 0.1 and 0.4% [6]. Numerous detrimental effects on the mother and fetus are associated with untreated overt and subclinical hypothyroidism [7]. Preeclamptic pregnant women were shown to be more likely than normal pregnant women to have low T4 levels and elevated TSH [8]. The relationship between maternal thyroid function and gestational hypertension is not well studied. Eighty pregnant women

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with hypothyroidism between the ages of 18 and 40 years and gestational ages between 20 and 40 weeks were enrolled. Twenty (25.0%) of the pregnant women who presented with hypothyroidism had pregnancy-induced hypertension [9]. In a prospective cohort study, Lai H et al enrolled 1226 pregnant women between 9 and 13 weeks of gestation; 81 of them developed pregnancy-induced hypertension (with a rate of 6.6%). Of the patients, 159 (12.9%) had thyroid dysfunction. Thyroid dysfunction was seen in 28.4% of pregnant women with gestational hypertension (n/N = 23/81) and 11.9% of pregnant women without gestational hypertension (n/N = 136/1145). Gestational hypertension was more common in pregnant women with hypothyroidism (8.6% vs. 2.5%) and subclinical hypothyroidism (11.1% vs. 5.4%) than in euthyroid women [10]. The information this study collected from our local setting by this study was useful in determining how significant the burden of hypothyroidism exists in hypertensive pregnant women. Therefore, by early screening for thyroid dysfunction in pregnancy, we will be able to address the issue and avoid the later development of pregnancy-induced hypertension, which could ultimately lead to Unfavourable outcomes for both the mother and the newborn.

According to the available data, a potential correlation between thyroid dysfunction and pregnancy-induced hypertension (PIH) may exist, but the results are not consistent and context-dependent. In addition, we do not have any strong comparative data on the local population, especially in Pakistan, which looks at the variation of thyroid dysfunction in hypertensive and normotensive pregnant women. This will leave a research gap in knowledge of the actual burden and trend of thyroid abnormalities in PIH in this context, constraining the use of evidence-based screening and control measures. This study aimed to understand the prevalence of hypothyroidism in pregnant women with pregnancy-induced hypertension.

METHODS

This comparative cross-sectional study was performed at the Department of Obstetrics and Gynaecology, Combined Military Hospital, Multan. The study spanned a period from 1st July 2024 to 31st December 2024 after acquiring approval from the institutional ethics committee (ERC# 49/2024; dated 18th April 2024). A total of 208 pregnant females, singleton pregnancy, 18-45 years of age, > 20 weeks of gestation, presenting prior to delivery and visiting the antenatal clinic, were recruited in this research through non-probability consecutive sampling after taking informed consent. Women with diabetes, kidney or autoimmune disorder, chronic hypertension, and on treatment for thyroid disorder before pregnancy were excluded from the study. The participants included 104 women with pregnancy-induced hypertension and 104 without PIH. A sample size of 208 participants was calculated through online software, using the formula for the proportion difference between two groups, assuming thyroid dysfunction of 28.4% in women with PIH and 11.4% without PIH [10], at 85% power of the study and 4% significance level. $n = (Z_{\alpha/2} + Z_{\beta})^2 [p_1(1-p_1) + p_2(1-p_2)]/(p_1-p_2)^2$. Women with systolic BP of more than 140 mmHg and/or diastolic BP of more than 90 mmHg, on at least two occasions and at least four hours apart, without proteinuria (urinary dipstick test negative for proteins) and with normal blood pressure before 20 weeks of pregnancy were categorized as pregnancy-induced hypertension (PIH). Body mass index was assessed at the time of enrolment. Weight was measured in kilograms on a digital weighing scale and height in meters on a stadiometer. Women with a body mass index of ≥30 kg/m^2 were categorized as obese. Non-fasting Free T3, T4, and TSH were measured after a venous blood sample was obtained in a serum separator tube from a single laboratory. Reference ranges for second (Free T3: 0.50 – 1.60 ng/dL, T4: 6.5 – 15.0 µg/dL, and TSH: 0.2 – 3.0 mU/L) and third trimester (Free T3: 0.50 – 1.50 ng/dL, T4: 7.0 – 15.5 µg/dL, and TSH: 0.3 – 3.0 mU/L) of pregnancy were applied as per the assay available in hospital. Patients with TSH levels >5.4 mU/l and normal levels of T3 and T4 were categorized as subclinical hypothyroid, TSH values > 5.4 mU/l and low free T3 (< 0.52) and or T4 (<4.5) as hypothyroid, serum TSH levels <0.4 mU/l with normal T3 and T4 as subclinical hyperthyroid and TSH levels <0.4 mU/l, high T3 (>1.9) and T4 (>12) as hyperthyroid. Presence of any of the abnormalities was categorised as thyroid dysfunction positive.

The data were analysed through SPSS version 27.0. Normality of quantitative data was evaluated through the Shapiro-Wilk test. Age, T3, T4, and TSH were not normally distributed and reported as median and IQR. Categorical variables were reported as numbers and proportions. Age, T3, T4, and TSH between the groups were compared through the Mann-Whitney U test. Categorical variables are compared through the chi-square test. For all the comparisons, p-value <0.05 was taken as significant.

RESULTS

Median age of the women was 22.5 (21-39) years, and 136 (65.4%) were below 30-years of age. Median gestational age was 23 (22-24) weeks. In 55.8% (n=116) of cases, women were multigravida, and 108 (51.9%) of the participants were obese. Age, gestational age, gravida, and obesity were comparable between women with and without pregnancy-induced hypertension (Table 1).

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Table 1: Characteristics of Pregnant women (9-13 weeks) visiting antenatal clinics of CMH (n=208)

Characteristics	All (n=208), n (%)	With PIH (n=104), n (%)	Without PIH (n=104), n (%)	p-value
Age (years)	22.5 (21-39)	22.5 (21.0 – 39.0)	22.5 (21.0 – 39.0)	>0.999
Age groups - < 30-year	136 (65.4%)	68 (50%)	68 (50%)	>0.999
≥ 30-year	72 (34.6%)	36 (50%)	36 (50%)	
Gestational age (week)	23 (22-24)	23 (22-24)	23 (22-24)	>0.999
Gravida - Primigravida	92 (44.2%)	48 (52.2%)	44 (47.8%)	0.577
Multigravida	116 (55.8%)	56 (48.3%)	60 (51.7%)	
Obesity - Yes	108 (51.9%)	57 (52.8%)	51 (47.2%)	0.405
No	100 (48.1%)	47 (47.0%)	53 (53.0%)	

PIH – Pregnancy Induced Hypertension, Age and Gestational Age Reported as Median (IQR). *Mann-Whitney U Test for Median Comparison, Chi-Square Test for Categorical Comparison

The median T3 (ng/dl), T4 (µg/dl), and TSH (mU/l) levels were 0.7 (0.5-1.1), 6.0 (5.2-6.9), and 1.6 (0.9-2.1), respectively. Thyroid dysfunction was identified in 33 (15.9%) of the participants. The most common abnormality was subclinical hypothyroidism in 14 (6.7%), followed by hypothyroidism in 9 (4.3%) and subclinical hyperthyroidism in 7 (3.4%). Mothers with PIH had a markedly high proportion of thyroid dysfunction in contrast to women without PIH (25% vs. 6.7%, p-value<0.001). Similarly, types of thyroid dysfunction were significantly more common in females having PIH compared to patients without PIH (Table 2).

Table 2: Thyroid Functions and Status of Pregnant Women (9-13 Weeks) Visiting Antenatal Clinics of CMH (N=208)

Characteristics	All (n=208), n (%)	With PIH (n=104), n (%)	Without PIH (n=104), n (%)	p-value
T3 (ng/dl)	0.7 (0.5-1.1)	0.6 (0.6-0.9)	0.7 (0.6 – 1.3)	0.028
T4 (µg/dl)	6.0 (5.2-6.9)	5.9 (4.9 – 7.0)	6.1 (5.5 – 6.8)	0.201
TSH (mU/l)	1.6 (0.9-2.1)	1.5 (0.8 – 2.1)	1.7 (1.0 – 2.2)	0.267
Thyroid Dysfunction - Yes	33 (15.9%)	26 (25.0%)	7 (6.7%)	<0.001
No	175 (84.1%)	78 (75.0%)	97 (93.3%)	
Hypothyroidism	9 (4.3%)	7 (6.7%)	2 (1.9%)	<0.001
Subclinical Hypothyroidism	14 (6.7%)	10 (9.6%)	4 (3.8%)	
Hyperthyroidism	3 (1.4%)	3 (2.9%)	0 (0.0%)	
Subclinical Hyperthyroidism	7 (3.4%)	6 (5.8%)	1 (1.0%)	

T3, T4, And TSH Were Reported as Median (IQR) And Compared Through the Mann-Whitney U Test. *Chi-Square Test for Categorical Comparison

DISCUSSION

Maternal thyroid hormone circulation has been linked to several unfavourable health outcomes in both pregnant women and young infants, and it is essential for maintaining systemic homeostasis. Hypertensive disorders are common in pregnant ladies having thyroid dysfunction. Variations in thyroid function can raise the risk of hypertensive pregnancy issues and have detrimental effects on several organ systems [11]. In the present study, thyroid dysfunction was identified in 15.9% of the participants. The most common abnormality was subclinical hypothyroidism in almost half of the cases, followed by hypothyroidism in 27.3% and subclinical hyperthyroidism in 21.2%. Patients with PIH had a markedly high proportion of thyroid dysfunction in contrast to women without PIH (p-value<0.001). Subclinical hypothyroidism during pregnancy was linked to higher chances of PIH (3.6% vs. 2.1%; OR, 1.5 [95% CI, 1.0 to 2.1], according to Begum *et al.* who conducted a large data meta-analysis. This supports our finding that women with PIH have high rates of thyroid dysfunction [12]. Found that at a TSH diagnostic threshold of <3.0 mIU/L, subclinical hypothyroidism during gestation was not linked to hypertension in pregnancy (p=0.077). Instead, whether the TSH diagnostic threshold is greater or less than 4 mIU/L, the risk of PIH rises [13]. In a systematic study, no statistically significant difference was found in the pooled OR of Unfavorable feto-maternal outcomes in pregnant females with subclinical hyperthyroidism and the euthyroid control group [14]. This contradicts our findings. These discrepancies may be because our study included all thyroid abnormalities, many of which (hypothyroid states) have stronger pathophysiological links to endothelial dysfunction and hypertensive disorders, while the meta-analysis only looked at subclinical hyperthyroidism, which frequently reflects normal gestational physiology and has minimal hypertensive impact. In 2023, a case-control investigation was carried out. In line with our increased thyroid dysfunction in PIH cases, they found that mean TSH

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was higher and FT4 was lower in patients with PIH compared to controls, and hypothyroidism was considerably more common in PIH (20 in PIH vs. 6 patients in controls) [15]. One hundred and thirty-three pregnant women were enrolled in case-control research by Aryee *et al.* In contrast to normotensive pregnancies, hypertensive pregnancies were more likely to have thyroid dysfunction (particularly increased TSH and reduced thyroid hormones), which is consistent with our findings of a significant link between PIH and thyroid dysfunction [16]. Discovered that thyroid dysfunction was more prevalent in patients with severe hypertension and contributed to Unfavorable outcomes (such as preterm delivery and low birth weight babies) in a retrospective cohort study carried out in Wuhan. This suggests that the correlation between thyroid abnormalities and PIH extends to the severity of complications [17]. Examined 200 pregnant women, 100 of whom had pre-eclampsia and 100 of whom had normal blood pressure. Compared to women with normotension, pre-eclamptic women had lower levels of free T3 and free T4 and high levels of TSH. The pre-eclampsia group had a considerably higher prevalence of both overt and subclinical hypothyroidism. Furthermore, the severity of PIH was found to be significantly correlated with worsening thyroid dysfunction, with severe cases linked to a large incidence of thyroid abnormalities [18]. Through common mechanisms of endothelial failure, abnormal placentation, and altered vascular control, PIH and thyroid dysfunction are closely linked. Hypertension in pregnancy is more frequently associated with hypothyroidism and subclinical hypothyroidism, which lower nitric oxide availability, raise systemic vascular resistance, and encourage arterial stiffness [19]. Additionally, trophoblastic invasion and spiral artery remodelling are may be affected by thyroid hormone insufficiency, which results in placental ischemia, oxidative stress, and the release of anti-angiogenic substances that are associated with worsened PIH [20]. Furthermore, dyslipidaemia and low-grade inflammation are linked to hypothyroid conditions, which worsen endothelial damage and hypertensive states during pregnancy. The strengths of this study were that thyroid dysfunction in pregnancy-induced hypertensive and normotensive women were clearly compared in equal group allocation. Confounding and misclassification were reduced by strictly excluding pre-existing thyroid conditions and hypertension. Analytical variability was decreased by uniform laboratory evaluation of free T3, free T4, and TSH from a single laboratory. Internal validity and repeatability were improved by using established biochemical cut-offs.

A few limitations of the present study were due to its cross-sectional design; a temporal correlation between thyroid dysfunction and PIH could not be determined. Pre-pregnancy thyroid status was not available, making it impossible to determine whether dysfunction preceded or resulted from PIH. Thyroid autoantibodies (anti-TPO, anti-TG) were not measured, limiting insight into autoimmune contributions. Because the study was only carried out at one tertiary care facility, its applicability to larger populations may be limited. In the future, prospective longitudinal studies are suggested to determine whether thyroid dysfunction precedes or follows the development of PIH. It would be easier to understand the underlying mechanisms if thyroid autoantibodies and iodine status were included. Research should be done to determine how early thyroid screening and treatment affect the severity of PIH and the results of pregnancies. To increase external validity, multi-centre research with a large population should be conducted.

CONCLUSIONS

Thyroid dysfunction, particularly subclinical hypothyroidism, was significantly more common among women with PIH in contrast to normotensive pregnancies. There should be targeted thyroid screening in hypertensive pregnancies to optimize maternal care.

Authors' Contribution

Conceptualization: AB
Methodology: AB, LN, SM, WQKN
Formal analysis: NY, SM,
Writing and Drafting: AB, LN, SM, WQKN
Review and Editing: AB, NY, LN, SM, WQKN

All authors approved the final manuscript and take responsibility for the integrity of the work

Conflict of Interest

All the authors declare no conflict of interest.

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