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RELATIONSHIP OF HYPERTHYROIDISM AND HYPEREMESIS GRAVIDARUM IN EARLY GESTATION

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Abstract

Objective:
During early pregnancy, determining the relationship between biochemical hyperthyroidism and hyperemesis gravidarum.

Study Design:
It's a case-control study.

Place and Duration of study:
Department of Gynecology and Obstetrics at Khyber Teaching Hospital, Peshawar, from March 2025 to July 2025, wherein the aim was to define the relationship between hyperthyroidism and HG in early pregnancy.

Methodology:
84 primigravidae in the first trimester were recruited, consisting of 42 with HG and 42 gestational-age-matched controls. The level of serum thyroid-stimulating hormone (TSH) was tested, and hyperthyroidism was operationally defined as TSH <0.3mIU/L.

Results:
The statistical analyses were done in SPSS version 25.0 and Chi-square or Fisher exact testing, odds ratios calculation, and logistic regression modelling. The prevalence of hyperthyroidism was found to be 23.8% in HG compared to 2.4% in controls (p=0.004), and the presence of HG was found to significantly increase the risk of hyperthyroidism (OR=0.078; 95% CI=0.009-0.642). HG was proven to be an independent predictor of suppressed TSH levels by multivariate regression.

Conclusion:
These findings support a strong linkage of HG and biochemical hyperthyroidism during early pregnancy and support the hypothesis of thyroid-stimulation mediated by hCG, and promote regular TSH screening of individuals with HG.

Introduction

The most extreme form of nausea and vomiting in pregnancy is hyperemesis gravidarum (HG), which remains one of the most significant obstetric problems in the world. Whereas mild nausea and vomiting are typical of pregnant women, HG represents a condition of great severity, persistence, and the extensive metabolic upheavals it causes.[1] Women with HG experience repeated vomiting, which often leads to dehydration, electrolyte imbalances, weight loss, nutrient deficiencies, and severe dysfunction of daily functioning.[2] HG is, therefore, not just a condition but a disorder that has far-reaching

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consequences to maternal health, developing fetuses, and daily functions.[3]

The mechanism of the pathophysiology of HG is multifactorial. Initial speculations were more focused on gastrointestinal, hormonal, and psychosomatic pathways, but modern-day discovery is greatly suggesting placental signaling pathways as major causal factors. A possible pathway is the growth differentiation factor-15 (GDF-15), which is a placental protein that becomes immensely elevated during early pregnancy.[4] Recent findings indicate that high levels of GDF-15 are highly correlated with the intensity of nausea and vomiting, which can have a mechanistic role in the pathogenesis of HG, so vulnerable subjects can develop an acute illness, whereas some have mild symptoms.[5] These findings demonstrate the biological diversity of HG and the necessity to have more effective treatment and diagnostic methods.[6]

The pregnancy is characterized by a significant endocrinological alteration of the thyroid axis. Estrogen boosts the thyroid-binding globulin and human chorionic gonadotropin (hCG), which has a similar structure to thyroid-stimulating hormone (TSH), and has a stimulatory effect on the thyroid gland; thus, transient changes in the levels of thyroid hormone are typical, particularly during early gestation when the concentration of hCG is high.[7] Although these changes are generally physiological in nature, at times they may trigger explicit thyroid dysfunction.[8] Hyperthyroidism in pregnancy is a disease that is subject to intense scrutiny due to the presence of serious maternal risks, such as miscarriage, preeclampsia, heart failure, and thyroid storm, and the presence of fetal complications and outcomes such as growth retardation and premature birth.[9] With such risks, hyperthyroidism is the second most common endocrine disorder in pregnancy, and it requires special attention and monitoring, and treatment regimens.[10]

The connection between HG and hyperthyroidism is not a relatively new phenomenon, as millions of years ago, both diseases shared a common path in the number of hormones.[11] Not only does the pronounced increase in hCG in early pregnancy cause nausea and vomiting, but the thyroid gland is also stimulated by a TSH-like effect that produces temporary, transient thyrotoxicosis, also known as gestational transient thyrotoxicosis (GTT), which may also coexist or mimic HG.[12] The interaction between these conditions is especially dramatic in gestational trophoblastic disease (GTD), where levels of hCG can be at levels many times higher than those seen in normal pregnancy.[13] These extreme levels of hormone exposure may be sufficient to elicit clinical effects of palpitations, tremors, and weight loss, which are significantly overlapping with HG.[14]

Severe cases of thyrotoxicosis, resulting in multi-system complications and life-threatening deterioration, have been reported in clinical cases of a hydatidiform mole precipitating severe thyroid dysfunction instead of merely being a biochemical abnormality.[15] In such cases, thyroid dysfunction is more than a biochemical abnormality; it is a significant determinant of morbidity. In exceptional yet life-threatening situations, HG has also been linked to thyroid storm, which is a hypermetabolic condition associated with fever, tachycardia, agitation, and an unstable hemodynamic condition.[16] Thyroid storm is a medical emergency, which highlights the imperative of early identification of thyroid disorders in women who show severe vomiting.[17]

Another major challenge is diagnosing the conditions since they have many similar symptoms. Both HG and hyperthyroidism have symptoms that overlap (tachycardia, nausea, vomiting, fatigue, and weight loss), which can delay the diagnosis of hyperthyroidism or lead treated inappropriate treatment.[18] On the other hand, the targeted study of thyroid dysfunction can result in the disregard of the nutritional and metabolic complications related to HG.[19] Thus, to make a proper diagnosis, a well-recognized clinical examination with the help of biochemical tests is critical.[20]

Hyperthyroidism is not typical in other instances. Cerebrovascular-like symptoms and other rare neurologic manifestations have also been described to make the clinical picture even more difficult and require an immediate assessment to rule out life-threatening conditions.[21] Also, long-

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term vomiting can change the metabolic parameters, including electrolytes and hepatic enzymes, which in their turn can confuse the meaning of thyroid function tests.[14]

The nutrition status is a determinant of the severity and progression of HG. The prolonged vomiting can result in a deficiency of both macro- and micronutrients, which can result in weakness, metabolic acidosis, and a poor immune response in women, as well as potentially lead to cardiac or neurological complications in the developing fetus, as the fetus depends on maternal nutrition [10]

In addition to biological factors, the social determinants also play a major role in the expression and treatment of HG.[8] There is variability in the severity of diseases due to socioeconomic status, education, access to healthcare, and environmental exposures, among which women in resource-limited settings may be late, poorly treated, have discontinuity of care, and consequently have increased risks of developing complications.[3] Hormonal dysregulation has also been identified to be caused by environmental endocrine disruptors, which may predispose to severe nausea and vomiting.[5]

The hyperthyroidism in the course of pregnancy bears significant implications as well in terms of the fetus's development. Excessive thyroid hormone levels have also been linked to miscarriage, placental malfunction, and fetal growth retardation.[12] On the other hand, overtreatment with antithyroid medication may cause fetal hypothyroidism or goiter, and this means that choices made by therapists require balancing the benefit of the mother and the child.[7] Propylthiouracil or methimazole are often used as antithyroid medications, but they should be used carefully as under- and over-suppression of thyroid activity can be dangerous.[4]

Mothers whose thyroid dysfunction is long-term have long-lasting effects beyond pregnancy. New evidence indicates that even the mild form of maternal thyroid hormonal abnormalities can impact neuro-development in the child, such as mental performance and behavioral changes.[21] These facts support the role of early diagnosis and proper control of maternal thyroid abnormalities during pregnancy.[9] Due to this, a number of predictive models have been suggested that can be used to discriminate high-risk women of thyroid dysfunction during the first trimester of pregnancy by combining clinical, biochemical, and demographic variables to enhance the effectiveness of the screening process and to provide specific intervention.[10]

Although the association between HG and thyroid abnormalities is accepted worldwide, local studies are still limited, especially among South Asians.[2] There are genetic predispositions, nutritional status, environmental exposures, and access to healthcare, which differ widely across different regions and can affect the disease prevalence and outcomes.[4] Local information is thus necessary in order to come up with context-specific guidelines and offer the best care to pregnant women [8]

In Pakistan, where the use of antenatal care is not homogeneous, and early presentation is not necessarily ensured, HG might never be diagnosed or treated properly.[10] In several centers, thyroid function testing is not done regularly, especially in the case of women who present with severe vomiting.[6] Therefore, hyperthyroidism can be diagnosed late when it becomes complicated. The relationship between HG and hyperthyroidism in the local population is hence important to help in enhancing clinical practice and preventing maternal and fetal complications that are preventable.[9]

Objective:

The study identified the relationship between hyperthyroidism and hyperemesis gravidarum during early pregnancy in women who reported to a Khyber Teaching Hospital, Peshawar.

METHODOLOGY

Study Setting

The research was conducted in the Department of Gynaecology and Obstetrics, Khyber Teaching Hospital, Peshawar.

Study Design

A Case-Control Study design was used.

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Duration of Study

The study was conducted from March 2025 to July 2025 after the synopsis is approved.

Sample Size

The sample will consist of 84 pregnant women who were recruited; this includes 42 women who are diagnosed with hyperemesis gravidarum and 42 in the control group. The calculation of the sample size was based on an estimated value of 24 % prevalence of hyperthyroidism among HG patients and 3 % prevalence among the normal pregnant population, with an 80 % power of a 95 % confidence interval using Statulator online software.

Sampling Technique

Consecutive non-probability sampling.

INCLUSION CRITERIA

Cases:

- Singleton pregnancy
- Age 18–40 years
- First trimester
- Diagnosed with hyperemesis gravidarum as per the operational definition

Controls:

- Singleton pregnancy
- Age 18–40 years
- First trimester
- No hyperemesis gravidarum

EXCLUSION CRITERIA

Pregnant women with:

- Gestational diabetes or hypertension
- Severe hepatic or renal dysfunction
- Thyroid disease
- Digestive system disease
- Malignant tumors
- Hematological system disease

Operational Definitions

- **Hyperthyroidism:** Diagnosed with serum TSH levels lower than the normal range (0.3-4.2mIU/L -1).
- **Hyperemesis Gravidarum:** Defined as persistent vomiting (≥3 times per day) during the first trimester of pregnancy, accompanied by ketonuria of at least 2+ (detected by urine routine examination), unrelated to other causes, and requiring hospital admission for severe vomiting causing dehydration (decreased BP, increased pulse, thirst, and decreased urine volume).

Data Collection Procedure

The College of Physicians and Surgeons Pakistan (CPSP) and the institutional ethics committee were consulted accordingly to provide ethical approval. The inclusion and exclusion criteria will be applied against women who visit the antenatal clinic during their first trimester. The eligible women who have hyperemesis gravidarum will be admitted, and written informed consent will be obtained. The same clinic will also be used to select an equal number of gestational-age-matched controls, and consent will also be taken. All participants were asked to fill in demographic information, including name, age, gravidity, location of residence, gestational weeks, employment status, and education levels. A 3mL venous blood sample was collected aseptically and forwarded to the hospital laboratory to have the serum TSH estimated. Hyperthyroidism will be coded according to the operational definition, and all the data gathered will be input into a tabular proforma (Annex 1).

Data Analysis Procedure

Analysis of the data was performed by SPSS 25.0. The quantitative variables (age, TSH level, gestational weeks, and gravidity) were presented as mean +/- standard deviation or median with the interquartile range, depending on the normality that will be measured using the Shapiro-Wilk test. Categorical variables (residence, employment status, education level, hyperthyroidism) will be calculated in terms of frequencies and percentages. The Chi-square test/Fisher's exact test was applied to test the relationship between hyperthyroidism and hyperemesis gravidarum, with a level of 0.05 being regarded as statistically significant. The odds ratio with a 95 per cent confidence interval will be used to measure the strength of association. Age, gravidity, and gestational weeks will be considered

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using post-stratification to ensure the mitigation of confounding. The findings were displayed in tabular and graphical formats. SPSS output sheets are attached as supplementary material.

RESULTS and DISCUSSION

The study involved 84 primigravidae during the first trimester, with 42 having hyperemesis gravidarum (HG) and 42 controls of similar gestation age.

Baseline Characteristics

Table 1 contains the socio-demographic and obstetric data of the participants of the study. There was no significant difference in the mean age of women (27.6 ± 3.3 years; p = 1.000) in the two groups. There were also no differences in the mean gestational age between the HG and the control group (9.5 ± 1.0 vs 9.4 ± 1.0 weeks; p = 0.650). There were no statistically significant differences in education, place of residence, or employment status between the two groups (p> 0.05), and this shows that there was adequate comparability of cases and controls.

Table I and Table VI demonstrate that the median serum TSH levels were numerically lower in the HG group than in controls, although this was not statistically significant (1.85 vs 1.95mIU/L; p= 0.103).

Table I: Socio-Demographic and Obstetric Characteristics of Study Participants (n = 84)

Characteristic	Hyperemesis Gravidarum (n = 42)	Controls (n = 42)	*p-value*
Age (years), mean ± SD	27.6 ± 3.3	27.6 ± 3.3	1.000†
Gestational age (weeks), mean ± SD	9.5 ± 1.0	9.4 ± 1.0	0.650†
TSH (mIU/L), median (IQR)	1.85 (1.27–2.10)	1.95 (1.70–2.10)	0.103‡
Education, n (%)			0.827§
Educated	22 (52.4)	23 (54.8)	
Uneducated	20 (47.6)	19 (45.2)	
Residence, n (%)			1.000§
Rural	26 (61.9)	26 (61.9)	
Urban	16 (38.1)	16 (38.1)	
Employment status, n (%)			1.000§
Employed	5 (11.9)	5 (11.9)	
Unemployed	37 (88.1)	37 (88.1)	

† Independent-samples *t*-test
‡ Mann–Whitney *U* test
§ Chi-square test

Correlation Hyperemesis Gravidarum vs. Hyperthyroidism.

Table-II indicates the relationship between hyperemesis gravidarum and biochemical hyperthyroidism. The HG group was found to have 10 women (23.8%) with biochemical hyperthyroidism (TSH < 0.3mIU/L) and 1 woman with biochemical hyperthyroidism. This was statistically significant (χ^2 = 8.47; p = 0.004; Fisher's exact p = 0.007). Hyperthyroidism odds ratio in women having HG was 0.078 (95 %CI: 0.009-0.642).

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Table II: Association Between Hyperemesis Gravidarum and Biochemical Hyperthyroidism

Thyroid Status	Controls (n = 42)	HG Cases (n = 42)	Total
Hyperthyroid (TSH < 0.3 mIU/L)	1	10	11
Not hyperthyroid	41	32	73
Total	42	42	84

$\chi^2 = 8.47$, $p = 0.004$
Fisher's exact $p = 0.007$
Odds Ratio = 0.078 (95% CI: 0.009–0.642)

Stratified Analysis

Table III, Table IV, and Table V provide stratified analyses by age group, gravidity, and gestational age, respectively. Within all of the strata, the HG group was found to have a higher percentage of biochemical hyperthyroidism than controls. Association directions were similar across all strata, but some of the comparisons were not statistically significant because of small subgroups.

Table III: Stratified Analysis by Age Group

Age Group (years)	HG (HT/Total)	Control (HT/Total)	Odds Ratio (95% CI)	*p-value*
≤ 24	2 / 9	0 / 7	—	0.182
25–29	5 / 21	1 / 22	0.15 (0.02–1.44)	0.068
≥ 30	3 / 12	0 / 13	—	0.055
Total	10 / 42	1 / 42	0.078 (0.009–0.64)	0.004

Table IV: Stratified Analysis by Gravidity

Gravidity	HG (HT/Total)	Control (HT/Total)	Odds Ratio (95% CI)	*p-value*
G1	3 / 8	0 / 7	—	0.070
G2	5 / 17	1 / 17	0.15 (0.02–1.46)	0.072
G3+	2 / 17	0 / 18	—	0.134
Total	10 / 42	1 / 42	0.078 (0.009–0.64)	0.004

Table VII: Binary Logistic Regression Analysis for Hyperthyroidism

Predictor	B	Wald	*p-value*	Adjusted OR (95% CI)
Hyperemesis gravidarum	–2.83	6.30	0.012	0.059 (0.006–0.538)
Age ≥ 30 years	1.08	1.50	0.221	2.93 (0.53–16.35)
Gravidity ≥ G3	–1.67	4.40	0.036	0.188 (0.039–0.896)
Gestational age (11 vs 8 weeks)	0.33	0.45	0.503	1.39 (0.53–3.66)
Education (educated)	–0.65	0.43	0.514	0.53 (0.08–3.63)
Residence (urban)	–0.93	0.95	0.329	0.40 (0.06–2.54)

Model $\chi^2 = 16.47$, $p = 0.011$
Nagelkerke $R^2 = 0.33$

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FIGURES

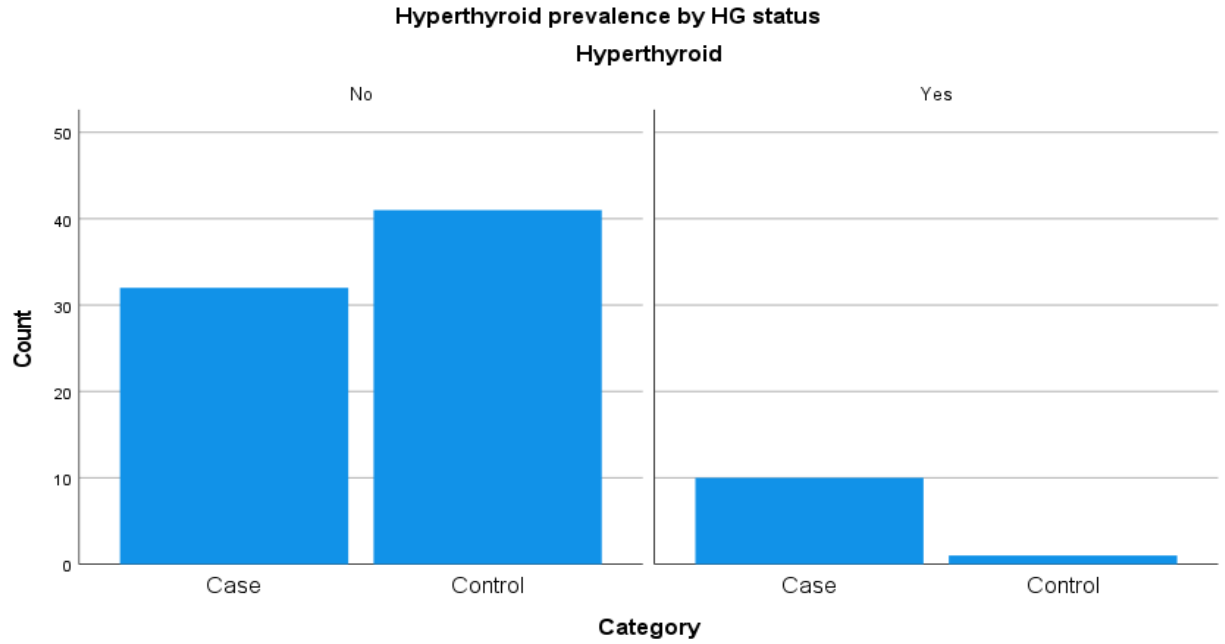

Figure I: Prevalence of biochemical hyperthyroidism among women with hyperemesis gravidarum and controls during early pregnancy

Prevalence: 23.8% (10/42) in HG vs 2.4% (1/42) in controls.

Table V: Stratified Analysis by Gestational Age

Gestational Age (weeks)	HG (HT/Total)	Control (HT/Total)	Odds Ratio (95% CI)	*p-value*
8	2 / 8	0 / 8	–	0.131
9	3 / 14	0 / 14	–	0.067
10	3 / 13	1 / 13	0.28 (0.03–3.10)	0.277
11	2 / 7	0 / 7	–	0.127
Total	10 / 42	1 / 42	0.078 (0.009–0.64)	0.004

Multivariate Analysis

Table VII represents a binary logistic regression analysis. Hyperemesis gravidarum was found to be independently related with biochemical hyperthyroidism after age, gravidity, gestational age, education, and residence (adjusted OR= 0.059; 95 %CI= 0.006- 0.538; *p*= 0.012). A statistically significant association was also found in gravidity, but other variables did not show any independent effect.

Table VI: Comparison of Median TSH Levels Between Groups

Group	Median (IQR) mIU/L	Mean Rank	Mann–Whitney U	*p-value*
Hyperemesis gravidarum	1.85 (1.27–2.10)	38.18	700.5	0.103
Control	1.95 (1.70–2.10)	46.82		

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DISCUSSION

The current case-control study proves a high degree of association between hyperemesis gravidarum and biochemical hyperthyroidism in early pregnancy. A very low percentage of healthy pregnant controls had suppressed levels of TSH as compared with nearly one-quarter of women with HG. This relationship remained even on the condition of the demographic and obstetric factors, which is why HG is an independent predictor of biochemical hyperthyroidism.

These results are in line with current knowledge that thyroid dysfunction, especially the suppressed TSH, is common in women experiencing severe nausea and vomiting during early pregnancy. In other studies, previous reports of suppression of TSH in a considerable proportion of HG cases support the idea of gestational transient thyrotoxicosis due to the elevated levels of human chorionic gonadotropin (hCG).[1,3] The prevalence in the current study corresponds to the range of those reports, which support the external validity of the findings.

The biological nature of this relationship is well known. hCG has structural homology with thyroid-stimulating hormone and is able to stimulate thyroid receptors, although temporarily, during the first trimester when hCG levels are maximum. This is the reason why there was no significant difference in median TSH levels across groups, whereas there was a particular subgroup of a HG patient population with a significant TSH inhibition, as observed in this paper.

Stratified analyses were done and showed that there was a consistent direction of association across age groups, gravidity, and gestational age. The overall trend was statistically significant in all subgroups, but the small size of the samples did not yield statistical significance; they had a biologically consistent relationship and thus were not by chance. The same patterns have been observed in previous research studies that have reported the influence of thyroid functioning in early gestation.[7-9].

The multivariate regression analysis also enhances the results since it shows that HG is still the most predictive independent variable of biochemical hyperthyroidism after addressing the possible confounder variables. The gravidity correlation could be due to adaptive endocrine or immune modifications during consecutive pregnancies, as indicated in the past literature.[10] The correlation should, however, be considered to be investigated further in larger studies.

The confusion of the symptoms in HG and hyperthyroidism, like vomiting, weight loss, tachycardia, and fatigue, can make it clinically difficult to detect thyroid dysfunction in women with severe vomiting, which can result in misinterpretation of the symptoms or improper practice. On the other hand, it is necessary to realise gestational transient thyrotoxicosis in order to prevent the inappropriate use of unnecessary antithyroid therapy that can be associated with risks to the fetus.[14]

The research contributes to the local findings in Pakistan, where thyroid screening in HG is not a uniform procedure and where limited evidence is available in the region. In light of possible maternal and fetal outcomes of thyroid malfunction during pregnancy, the results indicate the role of biochemical evaluation in a woman with severe vomiting at the initial stage of pregnancy.[17]

Certain shortcomings must be admitted. The single-center design can be a weakness of the generalizability, and no evaluation of free thyroxine levels or thyroid antibodies was performed, which inhibits the possibility to differentiate between transient thyrotoxicosis and underlying thyroid disease. Moreover, subgroup analyses were constrained due to the small sample size. Although such limitations exist, the uniformity of the results presented in the unadjusted, stratified, and adjusted analyses proves the strength of the observed association.

CONCLUSION

This study was done in an attempt to determine the correlation between hyperthyroidism and hyperemesis gravidarum (HG) in the first trimester of pregnancy. The statistics indicate that those individuals who present with HG during the first trimester have a significantly greater occurrence of thyroid dysfunction, specifically biochemical hyperthyroidism, as compared to individuals who

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do not have HG. The thyroid physiologic reaction to increased β-hCG levels seems to be one of the major contributory factors to these initial gestational alterations. The findings support the hypothesis that HG and hyperthyroidism are interrelated through common endocrine mechanisms, indicating the need to integrate the addition of thyroid testing in the diagnostic algorithm of women with severe vomiting during early pregnancy.

The paper highlights the importance of early diagnosis, targeted assessment, and proper treatment of thyroid diseases in pregnant women affected by HG. The management of gestational transient thyrotoxicosis versus primary hyperthyroid disease can be achieved by earlier identification of thyroid malfunction, which helps develop safe treatment regimens, thus alleviating maternal morbidity. Taken together, the facts support the idea of incorporating the thyroid profile evaluation into the routine of diagnostic algorithms used to diagnose HG, and that could potentially increase the accuracy of diagnosis and lead to a more positive clinical outcome.

ETHICAL APPROVAL

The Ethical Approval was obtained from the Institutional Ethics Committee of Khyber Teaching Hospital, Peshawar.

INFORMED CONSENT

All participants were informed of the study and signed a written informed consent before being enrolled.

COMPETING INTERESTS

The authors state that they do not have conflicting interests.

FUNDING

This study did not obtain any external funding.

AUTHOR CONTRIBUTIONS

The study was conceived by all the authors, data collected, analysed, and the manuscript written, and ultimately approved.

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The authors note that the cooperation of the study participants and the hospital laboratory staff is accepted.

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