

Evaluation of Second Trimester Serum Beta-Human Chorionic Gonadotropin (β -hCG) Levels as a Predictive Marker for Pregnancy-Induced Hypertension

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Abstract:

Background & Objectives: Hypertensive disorders of pregnancy, particularly pregnancy-induced hypertension (PIH) and preeclampsia, remain leading causes of maternal and perinatal morbidity and mortality worldwide. Abnormal placentation occurring between 8 to 18 weeks of gestation triggers hypoxic trophoblastic stress and hypersecretion of human chorionic gonadotropin (hCG). This study aimed to evaluate the clinical efficacy and predictive accuracy of second-trimester serum beta-human chorionic gonadotropin (β -hCG) levels (at 16–20 weeks of gestation) for the subsequent development and severity of PIH.

Methods: A prospective observational study was conducted involving 100 pregnant women with singleton pregnancies between 16–20 weeks of gestation attending the antenatal clinic. Baseline clinical evaluations and serum β -hCG estimations via Chemiluminescent Immunometric Assay (CLIA) were performed at booking. Patients were monitored prospectively until delivery and 12 weeks postpartum. Diagnostic performance was calculated using a cut-off threshold of 35,000 mIU/mL (equivalent to >2.0 Multiple of Median [MoM]). Correlation with disease severity was evaluated using Pearson's correlation coefficient.

Results: Of the 100 enrolled participants, 91 completed complete follow-up till delivery. PIH developed in 12 cases (13.19%), while 79 (86.81%) remained normotensive. Mean second-trimester serum β -hCG levels were significantly higher in women who subsequently developed PIH compared to normotensive controls ($52,144.75 \pm 21,561.62$ mIU/mL vs. $27,477.35 \pm 12,011.03$ mIU/mL, $p < 0.001$). Using a cut-off of 35,000 mIU/mL, elevated β -hCG yielded a sensitivity of 75.0%, specificity of 77.22%, positive predictive value (PPV) of 33.33%, negative predictive value (NPV) of 95.31%, and overall diagnostic accuracy of 76.92%. Bivariate correlation revealed a statistically significant moderate positive relationship between mid-trimester serum β -hCG levels and subsequent PIH severity ($r = 0.578$, $p < 0.05$).

Conclusion: Second-trimester serum β -hCG is an effective, reliable, and non-invasive biochemical predictor for PIH. Early identification of high-risk gravidae using serum β -hCG allows for targeted clinical surveillance, timely prophylactic interventions (such as low-dose aspirin and calcium), and mitigation of severe maternal-fetal complications.

Keywords: Pregnancy-Induced Hypertension, Preeclampsia, Beta-Human Chorionic Gonadotropin (β -hCG), Predictive Biomarkers, Trophoblastic Hypoxia, Second Trimester Screening.

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Introduction

Hypertensive disorders of pregnancy constitute one of the most critical challenges in contemporary obstetrics, affecting 5% to 10% of all pregnancies globally. Maternal mortality directly attributed to pregnancy-induced hypertension (PIH) and preeclampsia is estimated at 16%, outstripping other major obstetric complications including

hemorrhage (15%), unsafe abortion (8%), and puerperal sepsis (2%). According to estimates by the World Health Organization (WHO), a woman dies every 7 minutes due to complications associated with PIH in developing countries. The epidemiological burden of hypertensive disorders in pregnancy has seen a steady rise over the past

decade. This rise is primarily driven by societal and lifestyle changes, such as increased pre-pregnancy body mass index (BMI), delayed maternal age at first pregnancy, and sedentary lifestyles. In developing nations like India, maternal and perinatal morbidity and mortality remain disproportionately elevated due to social deprivation, delayed referral to tertiary care facilities, lack of access to skilled birth attendants, and inadequate antenatal screening.

Hypertensive disorders of pregnancy encompass a spectrum of clinical conditions including gestational hypertension, preeclampsia, eclampsia, and chronic hypertension with superimposed preeclampsia. Preeclampsia is fundamentally a multisystem disorder characterized by new-onset hypertension (systolic BP ≥ 140 mmHg and/or diastolic BP ≥ 90 mmHg) and proteinuria or systemic end-organ dysfunction after 20 weeks of gestation. Maternal morbidities include eclamptic seizures, placental abruption, acute kidney injury, pulmonary edema, HELLP syndrome (hemolysis, elevated liver enzymes, low platelets), and cerebrovascular accidents. Fetal and neonatal consequences are equally severe, manifesting as intrauterine growth restriction (IUGR), oligohydramnios, premature delivery, low Apgar scores, and intrauterine fetal demise.

Although clinical signs and symptoms of PIH typically present during the third trimester, the pathophysiological cascade originates much earlier, during the late first and early second trimesters (8 to 18 weeks of gestation). Normal pregnancy is characterized by endovascular trophoblastic invasion into the uterine spiral arteries, converting them from narrow, high-resistance muscular vessels into wide, low-resistance, high-capacitance conduits. In pregnancies destined to develop PIH, defective extravillous cytotrophoblastic invasion results in persistent high vascular resistance, hypoperfusion, and placental ischemia.

In response to local hypoxia and ischemia, placental cytotrophoblasts undergo compensatory transformation into syncytiotrophoblasts, leading to hyperplacentosis and a hypersecretory state. Human chorionic gonadotropin (hCG), a glycoprotein hormone secreted by syncytiotrophoblasts, is markedly elevated in this hypoxic environment.

Therefore, measuring second-trimester serum β -hCG levels (between 16 and 20 weeks) provides a logical biochemical window to identify asymptomatic women at high risk of developing PIH before clinical onset.

Objectives

1. To evaluate the association between elevated mid-trimester (16–20 weeks) serum β -hCG

levels and the subsequent development of Pregnancy-Induced Hypertension (PIH).

2. To determine the diagnostic efficacy (sensitivity, specificity, positive predictive value, negative predictive value, and accuracy) of serum β -hCG as a predictive screening tool.
3. To analyze the correlation between mid-trimester serum β -hCG concentration and the clinical severity of PIH.

Materials and Methods

Study Design and Setting: This prospective clinical cohort study was conducted in the Department of Obstetrics and Gynaecology in collaboration with the Department of General Medicine at tertiary care teaching hospitals affiliated with J.J.M. Medical College, Davangere, over a two-year period (October 2019 to October 2021). Institutional Ethical Committee clearance was obtained prior to study initiation, and written informed consent was obtained from all participating women.

Study Population & Eligibility Criteria: A total of 100 pregnant women presenting for routine antenatal care between 16 and 20 weeks of gestation were consecutively screened and enrolled based on strict eligibility criteria.

Inclusion Criteria:

- Pregnant women with singleton gestations.
- Confirmed gestational age between 16 and 20 weeks (verified by reliable menstrual dates and early first-trimester crown-rump length ultrasound).
- Maternal age between 20 and 35 years.
- Normal pre-pregnancy body mass index (BMI < 24.9 kg/m²).

Exclusion Criteria:

- Multifetal gestations (twins, triplets).
- History of pre-existing chronic hypertension, pre-gestational diabetes mellitus, renal disease, or vascular disorders.
- History of recurrent pregnancy loss, molar pregnancy, or preeclampsia in previous pregnancies.
- Rh-negative blood group status, autoimmune diseases (e.g., APLA, SLE), or active malignancy.
- Conception via assisted reproductive techniques (IVF/ICSI).
- Documented major fetal congenital malformations on ultrasound screening.

Clinical Protocol & Laboratory Procedure: At initial enrollment (16–20 weeks), comprehensive demographic data, obstetric history, medical history, baseline physical examination, and body mass index (BMI) were recorded.

Blood pressure was measured in a comfortable seated position with back supported after 10 minutes of rest, using a standardized, calibrated mercury sphygmomanometer with an appropriate arm cuff size. A 3 mL venous blood sample was drawn under aseptic precautions into a plain tube (red top) for serum β -hCG estimation. Serum was separated by centrifugation at 3000 rpm for 10 minutes and processed using the Chemiluminescent Immunometric Assay (CLIA) technique. A threshold value of 35,000 mIU/mL (corresponding to >2.0 Multiple of Median [MoM] for 16–20 weeks) was pre-defined as an elevated β -hCG level based on receiver operating characteristic analysis and literature standard cut-offs.

Follow-up and Clinical Endpoints: All enrolled subjects were followed prospectively throughout antenatal visits: every 4 weeks until 28 weeks, fortnightly until 34 weeks, and weekly thereafter until delivery, extending up to 12 weeks postpartum. At each visit, participants underwent clinical evaluation, blood pressure recording, and clean-catch dipstick urinalysis for proteinuria.

PIH was defined as new-onset systolic blood pressure ≥ 140 mmHg and/or diastolic blood pressure ≥ 90 mmHg on two separate measurements at least 4 hours apart after 20 weeks of gestation in a previously normotensive woman. Non-severe PIH was defined as BP 140–159/90–109 mmHg without end-organ injury. Severe PIH / preeclampsia was defined as BP $\geq 160/110$ mmHg, significant proteinuria ($\geq 2+$ dipstick or ≥ 300 mg/24 hours), or systemic end-organ dysfunction (thrombocytopenia $< 100,000/\text{mm}^3$, serum creatinine > 1.1 mg/dL, elevated transaminases twice normal, persistent severe headache, visual scotomata, or epigastric pain).

Statistical Analysis: Data were entered into Microsoft Excel and analyzed using SPSS version 25.0. Continuous variables were expressed as Mean $\pm$ Standard Deviation (SD) and categorical

variables as frequencies and percentages. Continuous baseline parameters between PIH and normotensive groups were compared using the Student's independent t-test. Categorical outcomes were evaluated using the Chi-square test or Fisher's exact test. Diagnostic performance parameters (sensitivity, specificity, PPV, NPV, and diagnostic accuracy) were derived using 2x2 contingency matrix analysis. Bivariate Pearson's correlation coefficient (r) was computed to assess the correlation between serum β -hCG levels and disease severity. A p -value < 0.05 was considered statistically significant, and $p < 0.001$ was considered highly significant.

Results

Out of 100 enrolled pregnant women, 9 (9.0%) were lost to follow-up due to relocation or delivery elsewhere. Thus, 91 participants completed the full follow-up protocol until delivery and post-partum evaluation.

Among the 91 completed cases, 12 women (13.19%) developed Pregnancy-Induced Hypertension (PIH), while 79 women (86.81%) remained normotensive throughout pregnancy.

Baseline Demographic and Clinical Characteristics: The mean maternal age in the PIH group was 25.58 ± 3.57 years (range: 20–32 years) compared to 24.40 ± 3.33 years (range: 19–36 years) in the normotensive group ($p = 0.261$). The majority of subjects were aged 20–24 years (48.35%), followed by 25–29 years (39.56%). Mean BMI was 22.48 ± 1.83 kg/m² in the PIH group and 22.29 ± 1.90 kg/m² in the normotensive group ($p = 0.742$). Parity distribution was comparable between groups: the PIH group comprised 7 primigravidae (58.33%) and 5 multigravidae (41.67%), while the normotensive group comprised 39 primigravidae (49.37%) and 40 multigravidae (50.63%). There were no statistically significant differences in age, BMI, or parity, confirming baseline comparability.

Table 1: Baseline Demographic and Clinical Characteristics of Study Participants

Parameter	PIH Group (n=12)	Normotensive (n=79)	t-value / Statistical Test	p-value	Significance
Maternal Age (years)	25.58 ± 3.57	24.40 ± 3.33	$t = 1.130$	0.261	Not Significant (NS)
Body Mass Index (kg/m ²)	22.48 ± 1.83	22.29 ± 1.90	$t = 0.330$	0.742	Not Significant (NS)
Parity (Primi / Multi)	7 / 5 (58.3% / 41.7%)	39 / 40 (49.4% / 50.6%)	$\chi^2 = 0.342$	0.558	Not Significant (NS)

Blood Pressure Changes across Gestation: At initial booking (16–20 weeks), baseline systolic blood pressure (123.33 ± 5.54 mmHg vs. 121.22 ± 6.30 mmHg, $p = 0.274$) and diastolic blood pressure (77.67 ± 4.49 mmHg vs. 78.95 ± 4.91 mmHg, $p = 0.397$) showed no significant difference

between the PIH and normotensive groups. However, at delivery, systolic BP (162.50 ± 18.09 mmHg vs. 121.44 ± 5.99 mmHg, $t = 15.61$, $p < 0.001$) and diastolic BP (104.83 ± 9.12 mmHg vs. 78.73 ± 5.02 mmHg, $t = 14.79$, $p < 0.001$) were significantly higher in the PIH group.

Table 2: Comparison of Mean Systolic and Diastolic Blood Pressure (mmHg) Between Groups

Time point / Variable	PIH Group (n=12)	Normotensive (n=79)	t-value	p-value	Significance
Booking Systolic BP (mmHg)	123.33 ± 5.54	121.22 ± 6.30	1.10	0.274	Not Significant (NS)
Booking Diastolic BP (mmHg)	77.67 ± 4.49	78.95 ± 4.91	-0.85	0.397	Not Significant (NS)
Delivery Systolic BP (mmHg)	162.50 ± 18.09	121.44 ± 5.99	15.61	< 0.001	Highly Significant (HS)
Delivery Diastolic BP (mmHg)	104.83 ± 9.12	78.73 ± 5.02	14.79	< 0.001	Highly Significant (HS)

Second-Trimester Serum β -hCG Levels and Predictive Accuracy: Mean mid-trimester serum β -hCG levels were significantly elevated in women who subsequently developed PIH compared to those who remained normotensive (52,144.75 ± 21,561.62 mIU/mL vs. 27,477.35 ± 12,011.03 mIU/mL; $t = 5.871$, $p < 0.001$).

Table 3: Comparison of Second-Trimester Serum β -hCG Levels (mIU/mL) Between Groups

Biomarker	PIH Group (n=12)	Normotensive (n=79)	t-value	p-value	Significance
Serum β -hCG (mIU/mL)	52,144.75 ± 21,561.62	27,477.35 ± 12,011.03	5.871	< 0.001	Highly Significant (HS)

When categorizing cases based on the cut-off threshold of 35,000 mIU/mL, 27 women had elevated β -hCG (>35,000 mIU/mL), of whom 9 (33.33%) developed PIH. Conversely, among 64 women with normal β -hCG (<35,000 mIU/mL), only 3 (4.69%) developed PIH (Chi-square = 13.82, $p = 0.001$).

Table 4: Distribution of Cases According to Serum β -hCG Threshold (>35,000 mIU/mL)

Serum β -hCG Level	PIH Developed (n=12)	Normotensive (n=79)	Total (n=91)
> 35,000 mIU/mL (Elevated)	9 (75.0%)	18 (22.8%)	27
< 35,000 mIU/mL (Normal)	3 (25.0%)	61 (77.2%)	64
Total	12 (100.0%)	79 (100.0%)	91

Table 5: Stratified Distribution of Serum β -hCG Levels and Hypertensive Status

Serum β -hCG Range (mIU/mL)	PIH Group (n=12)	Normotensive Group (n=79)	Total Subjects (n=91)
< 20,000	1 (8.3%)	23 (29.1%)	24
20,000 – 30,000	2 (16.7%)	24 (30.4%)	26
30,000 – 40,000	1 (8.3%)	22 (27.8%)	23
40,000 – 50,000	1 (8.3%)	9 (11.4%)	10
50,000 – 60,000	2 (16.7%)	0 (0.0%)	2
60,000 – 70,000	2 (16.7%)	0 (0.0%)	2
70,000 – 80,000	2 (16.7%)	1 (1.3%)	3
80,000 – 90,000	1 (8.3%)	0 (0.0%)	1
Total	12 (100.0%)	79 (100.0%)	91

Correlation with Disease Severity: Among the 12 women who developed PIH, 5 (41.67%) had non-severe PIH and 7 (58.33%) developed severe PIH / preeclampsia. Bivariate Pearson correlation analysis demonstrated a statistically significant

moderate positive correlation between serum β -hCG levels and disease severity ($r = 0.578$, $p < 0.05$). Women with serum β -hCG >50,000 mIU/mL predominantly progressed to severe preeclampsia.

Table 6: Association Between Mid-Trimester Serum β -hCG Levels and PIH Clinical Severity

Serum β -hCG Range (mIU/mL)	Total PIH Cases (n=12)	Non-Severe PIH (n=5)	Severe PIH / Preeclampsia (n=7)
< 20,000	1	0	1
20,000 – 30,000	2	2	0
30,000 – 40,000	1	0	1
40,000 – 50,000	1	1	0
50,000 – 60,000	2	0	2
60,000 – 70,000	2	1	1
70,000 – 80,000	2	1	1
80,000 – 90,000	1	0	1
Total	12	5	7

Table 7: Diagnostic Efficacy Metrics of Second-Trimester Serum β -hCG (>35,000 mIU/mL)

Diagnostic Metric	Calculated Value (%)	95% Confidence Interval
Sensitivity	75.00%	42.81% – 94.51%
Specificity	77.22%	66.41% – 85.82%
Positive Predictive Value (PPV)	33.33%	21.68% – 47.40%
Negative Predictive Value (NPV)	95.31%	88.08% – 98.25%
Overall Diagnostic Accuracy	76.92%	66.93% – 85.14%

Discussion

Early prediction of pregnancy-induced hypertension remains a cornerstone of preventive modern obstetrics. The primary finding of this study is that elevated serum β -hCG levels (>35,000 mIU/mL or >2.0 MoM) measured during the second trimester (16–20 weeks of gestation) are strongly associated with the subsequent development and clinical severity of PIH. Our prospective evaluation demonstrated a sensitivity of 75.0%, specificity of 77.22%, negative predictive value of 95.31%, and overall diagnostic accuracy of 76.92%.

Pathophysiological Rationale of Serum β -hCG Elevation: The physiological mechanism governing β -hCG elevation in preeclampsia stems from defective endovascular trophoblastic invasion. In normal gestation, extravillous trophoblasts invade the uterine spiral arteries up to the inner third of the myometrium, transforming narrow, musculo-elastic vessels into high-flow, low-resistance vascular channels. In pregnancies destined for PIH, trophoblastic invasion is incomplete, restricted only to the decidual segment. Persistent high vessel resistance causes localized placental hypoperfusion, ischemia, and cellular hypoxia.

Hypoxia triggers severe cellular stress in the placenta, arresting cytotrophoblast differentiation and inducing compensatory differentiation into syncytiotrophoblasts (hyperplacentalosis). Syncytiotrophoblasts enter a hypersecretory state, releasing vast quantities of human chorionic gonadotropin into the maternal circulation. Furthermore, placental hypoxia impairs cellular clearance and increases syncytiotrophoblastic microparticle shedding into maternal systemic blood, amplifying maternal endothelial cell

activation, systemic inflammation, and oxidative stress.

Comparative Analysis with Existing Literature:

The incidence of PIH in our study cohort was 13.19% (12 out of 91 cases), which aligns closely with reported rates in South Asian pregnant populations. Vidyabati et al. reported a PIH incidence of 17.7% among 164 gravidae evaluated at 14–20 weeks. Similarly, Satyanarayan et al. observed an incidence of 10.8% (21/174), and Kaur et al. reported 12.35% (22/178).

Our finding of significantly higher mean serum β -hCG in PIH cases (52,144.75 $\pm$ 21,561.62 mIU/mL vs. 27,477.35 $\pm$ 12,011.03 mIU/mL, $p < 0.001$) corroborates multiple international studies. Vidyabati et al. demonstrated that 72.4% of gravidae who developed PIH had serum β -hCG levels >45,000 mIU/mL, calculating that every 1,000 mIU/mL increase in β -hCG raised PIH risk by 10.7%. In a landmark trial by Kaur et al., 20 out of 24 women (83.3%) with mid-trimester β -hCG >2 MoM developed PIH, yielding a sensitivity of 90.91% and specificity of 97.44%. Desai et al. similarly reported that 62 out of 90 gravidae (68.9%) with β -hCG >2 MoM developed PIH, noting that 95.1% of these women developed symptoms prior to 32 weeks, indicating early-onset severe disease.

Conversely, Satyanarayan et al. found no statistically significant correlation between mid-trimester hCG and PIH (mean hCG 15,660 mIU/mL in PIH vs. 17,112 mIU/mL in normotensives). This discrepancy across studies can be attributed to differences in gestational age at sampling (13 vs 18 weeks), varying assay methodologies (RIA vs CLIA), differing diagnostic cut-off definitions, and heterogeneous study population risk profiles.

Table 8: Comparative Efficacy of Serum β -hCG for Predicting PIH across Studies

Study / Author	Publication Year	Sample Size (n)	Sensitivity (%)	Specificity (%)	Positive Predictive Value (%)
Satyanarayan et al.	2001	174	14.29%	87.93%	12.50%
Kaur et al.	2012	178	90.91%	97.44%	89.33%
Pawar et al.	2017	440	75.00%	75.00%	31.20%
Present Study	2026	91	75.00%	77.22%	33.33%

Correlation with Clinical Severity & Clinical Utility: In our cohort, a moderate significant positive correlation was established between serum

β -hCG levels and disease severity ($r = 0.578$, $p < 0.05$). Gravidae presenting with serum β -hCG concentrations exceeding 50,000 mIU/mL

predominantly progressed to severe preeclampsia requiring early delivery. This aligns with findings by Murtoniemi et al., who confirmed that β -hCG levels are significantly higher in severe early-onset preeclampsia than in non-severe late-onset variants.

From a clinical management standpoint, mid-trimester serum β -hCG offers a high Negative Predictive Value (95.31%). This high NPV indicates that gravidae with normal β -hCG levels ($<35,000$ mIU/mL) can be reliably reassured of a low risk for severe PIH, avoiding unnecessary hospitalizations. For women testing positive ($>35,000$ mIU/mL or >2.0 MoM), low-dose aspirin (75–150 mg daily) initiated early, combined with calcium supplementation (1.0–1.5 g daily) and close blood pressure and biophysical monitoring, can significantly delay disease onset and attenuate adverse maternal-fetal sequelae.

Strengths and Limitations: The primary strength of this study is its prospective longitudinal design, well-defined inclusion/exclusion criteria, uniform chemiluminescent assay methodology, and complete clinical follow-up through the postpartum period. However, limitations include a relatively small sample size ($n=91$) from a single geographical region and the lack of combined uterine artery Doppler ultrasonography or additional angiogenic biomarkers (such as sFlt-1 or PlGF), which could further boost positive predictive value.

Conclusion

In conclusion, second-trimester maternal serum β -hCG estimation (at 16–20 weeks of gestation) serves as an effective, accessible, non-invasive biochemical predictor for pregnancy-induced hypertension. Elevated serum β -hCG levels ($>35,000$ mIU/mL or >2.0 MoM) significantly correlate with both the development and clinical severity of PIH, demonstrating high diagnostic accuracy (76.92%) and exceptional negative predictive value (95.31%). Incorporating serum β -hCG screening into routine second-trimester antenatal protocols can facilitate early risk stratification, timely prophylactic pharmacological administration, and improved maternal and perinatal health outcomes.

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