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Original Research Article

A Study of Maternal Serum Beta-hCG and Lipid Profile in Early and Late Second Trimester as Predictor of PIH/Pre-Eclampsia and Its Time of Onset

Isha[1], Rupam Sinha[2], Geeta Sinha[3], Amrita Rai[4]

[1]Junior Resident, Department of Obstetrics and Gynaecology, Patna Medical College & Hospital, Patna, Bihar, India

[2]Professor, Department of Obstetrics and Gynaecology, Patna Medical College & Hospital, Patna, Bihar.

[3]Professor and HOD, Department of Obstetrics and Gynaecology, Patna Medical College & Hospital, Patna, Bihar, India

[4]Associate Professor, Department of Obstetrics and Gynaecology, Patna Medical College & Hospital, Patna, Bihar, India

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Corresponding author: Dr. Isha

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Abstract

Background: Hypertensive disorders of pregnancy complicate up to 10% of pregnancies worldwide, In spite of improvement in maternal and neonatal care, PIH and its sequel are a dreaded complication of pregnancy. If prediction becomes possible, prevention will follow naturally. Aim of this is to evaluate "whether maternal serum beta hCG and lipid profile levels in early and late second trimester are correlated with subsequent development of PIH/Preeclampsia and its time of onset.

Methods: This prospective observational study was conducted at Department of Obstetrics and Gynaecology, Patna Medical College and Hospital, Patna, Bihar from March 2024 to February 2026. Total 200 antenatal patients between 14-28 weeks (as determined by LMP or Dating USG scan) with singleton pregnancy were included in this study. Blood samples were collected twice, once at 14–18 weeks and second at 24–28 weeks for Beta hCG and lipid profile.

Results: The present study analyzed various maternal characteristics and biochemical parameters to understand their association with pregnancy-induced hypertension (PIH). The age-wise distribution of participants showed that most women were in the 25–29 years group (41%), followed by 30–34 years (30.5%) and 20–24 years (28.5%), suggesting higher engagement from slightly older young adults. Interestingly, when looking specifically at PIH cases, younger women aged 20–24 years were more affected (45%), followed by 25–29 years (28.33%) and 30–34 years (26.67%). This indicates that early adulthood, particularly the first pregnancies, may carry a higher risk for developing PIH, likely due to physiological and immunological adaptations during a first pregnancy. Lipid profiles also differed markedly between groups. In both early and late second trimesters, PIH patients had higher total cholesterol, triglycerides, LDL, and VLDL, and lower HDL compared to non-PIH women. For instance, early second-trimester triglycerides averaged 197.70 mg/dl in PIH versus 140.58mg/dl in non-PIH participants, while HDL was reduced to 41.41 mg/dl in PIH from 52.83 mg/dl in non-PIH. These differences persisted and became more pronounced in the late second trimester. Importantly, low HDL and high triglycerides were observed more often in PIH patients, with 65–80% showing reduced HDL and 70–75% having elevated triglycerides. This pattern was also consistent when analyzed according to onset week, with earlier onset PIH associated with higher triglycerides and lower HDL, suggesting that dyslipidemia plays a role in the development and severity of the condition. β-hCG levels were measured in study participants. The study showed that in early & late 2nd trimester, the mean β-hCG levels were significantly higher in PIH groups as compared to the non-PIH group, with a statistically significant p-value of <0.001.

Conclusion: One of the main causes of maternal morbidity and death during pregnancy is hypertensive disorders. It is still difficult to find the optimum illness predictor, whose use might considerably change the related morbidity and death. Serum beta-hCG and lipid profile can be used to predict PIH, the tests with accepted level of sensitivity and sensitivity. If this test is made available to women who have PIH risk factors, it would be beneficial.

Keywords: Serum Beta hCG, Pre-Eclampsia, Eclampsia, Pregnancy Induced Hypertension.

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Introduction

Hypertensive disorders of pregnancy are among the most common medical complications that affect pregnant women worldwide. These disorders represent a group of conditions characterized by high blood pressure during pregnancy and are associated with serious risks for both the mother and the fetus. Despite improvements in maternal healthcare and antenatal monitoring, hypertensive disorders continue to be a major cause of maternal illness and death. They are considered clinically challenging because their exact cause is still not fully understood, and the mechanisms involved in their development are complex.

Therefore, early identification and proper management are essential to reduce complications and improve pregnancy outcomes.

According to a meta-analysis conducted by the World Health Organization in June 2014, hypertensive disorders of pregnancy are responsible for about 14% of maternal deaths globally.[1] This shows that these conditions contribute significantly to maternal mortality in both developed and developing countries.

However, the burden is often higher in less industrialized countries where access to healthcare services, early diagnosis, and proper antenatal care may be limited.

In India, pregnancy-induced hypertension (PIH) is a major public health concern. Studies have reported that the incidence of PIH ranges from 5% to 15% among pregnant women in India.[2,3] The high prevalence highlights the importance of early detection and preventive strategies. Many pregnant women may not receive adequate antenatal care, which increases the chances of delayed diagnosis and complications associated with hypertension during pregnancy.

Hypertensive disorders of pregnancy include several conditions such as chronic hypertension, gestational hypertension, preeclampsia, chronic hypertension with superimposed preeclampsia, and eclampsia. Chronic hypertension refers to high blood pressure that exists before pregnancy or develops before 20 weeks of gestation. Gestational hypertension develops after 20 weeks of pregnancy in women who previously had normal blood pressure. Preeclampsia is a more severe condition characterized by hypertension along with other complications such as proteinuria or organ dysfunction. In some cases, women with chronic hypertension may develop additional features of preeclampsia, which is known as chronic hypertension with superimposed preeclampsia. Eclampsia is the most severe form and is characterized by seizures in a woman with preeclampsia.[3] The exact cause and onset of hypertensive disorders of pregnancy remain unclear. Several theories have been proposed regarding the etiopathogenesis of PIH, including abnormal placentation, endothelial dysfunction, immune maladaptation, and genetic factors. Abnormal development of the placental blood vessels may lead to reduced blood flow to the placenta, which can trigger hypertension and other complications during pregnancy.

Because the causes of PIH are not fully understood, researchers have been trying to identify reliable methods to predict its occurrence. Early prediction is important because it allows healthcare providers to monitor high-risk mothers more closely and provide timely management. Many screening methods have been suggested, including biochemical markers, hormonal assays, and other laboratory tests. However, no single test has been found to be completely reliable in predicting hypertension during pregnancy.

One important hormonal marker that has been studied is beta-human chorionic gonadotropin (β-hCG). This hormone is produced by the placenta and plays a crucial role in maintaining pregnancy. In cases of PIH, studies have shown that there may be a mid-trimester increase in β-hCG levels due to an increased secretory activity of the immunologically modified trophoblast.[4] This abnormal rise in β-hCG may reflect underlying placental dysfunction, which is an important factor in the development of hypertensive disorders.

In addition to hormonal changes, abnormalities in lipid metabolism are also observed in women with PIH. During pregnancy, lipid levels normally increase to support fetal development. However, in hypertensive pregnancies, there may be a significant rise in serum triglyceride (TG) levels, often reaching two to three times the normal level.[5] These elevated triglycerides may accumulate in the uterine spiral arteries and contribute to endothelial damage and vascular dysfunction.

The endothelial damage caused by lipid accumulation may lead to increased vascular resistance and impaired blood circulation in the placenta. This process plays an important role in the development of hypertension and other complications associated with PIH. Therefore, evaluation of the lipid profile during pregnancy may help in identifying women who are at higher risk of developing hypertensive disorders.[5]

Since hypertensive disorders of pregnancy are multifactorial, relying on a single test may not provide accurate prediction. It has been suggested

that a combination of different antenatal tests may improve the sensitivity and specificity of predicting PIH. By assessing both hormonal and metabolic changes, it may be possible to identify early physiological alterations that occur before the clinical symptoms appear.

Considering these factors, measuring maternal β-hCG levels along with lipid profile during the second trimester may serve as useful predictors for the development of pregnancy-induced hypertension. Early detection of high-risk mothers would allow closer monitoring and timely intervention, which could help reduce maternal and fetal complications.[4]

The primary objective of this study is to whether early and/or late 2nd trimester serum lipid profiles are correlated with development of PIH/preeclampsia and secondary is to whether the above parameters can be predictive of the time of onset of PIH/preeclampsia - early or late onset.

Material and Methods

This prospective observational study was conducted at Department of Obstetrics and Gynaecology, Patna Medical College and Hospital, Patna, Bihar from March 2024 to February 2026. Total 200 antenatal patients between 14-28 weeks (as determined by LMP or Dating USG scan) with singleton pregnancy were included in this study.

Patients with multiple pregnancy, chronic hypertension, congenital anomalies, and diabetes mellitus, medical diseases which may alter lipid profile and renal parenchymal disease were excluded in this study.

All patients included in the study were subjected to a detailed history taking followed by general examination and complete antenatal check-up after enquiring about any other complications.

Those patients who had any of the criteria mentioned in the exclusion criteria were excluded and others were included into the study population. The women included in the study were informed in detail about the potential significance of the test and its benefit in detail in their own language and an informed consent was obtained from them after explaining that the details obtained from them would be used only for study purposes. Blood samples were collected twice, once at 14–18 weeks and second at 24–28 weeks for Beta hCG and lipid profile.

Analysis of Data: Data was summarised by routine descriptive studies like Mean, Median, Standard deviation and Quartiles for numerical variables and counts and percentages for categorical variables. Univariate analysis was done by comparing beta hCG and individual lipid profile parameters between patients with PIH and those without PIH. Quantification of the risk associated with each of these biochemical parameters as a predictor of PIH and its time of onset through ROC Curve analysis was done.

Statistical Analysis: All data were analyzed using the SPSS software package (Stata, version 26.0, SPSS Inc., Chicago, IL, USA) for Windows. Descriptive statistics were used to summarize continuous variables, while categorical variables were expressed as percentages. Statistical tests applied included the Chi-square test and t-test. Other values were represented as numbers, proportions (%), and mean±standard deviation (SD).

Results

Table 1: Comparison of blood pressure in PIH vs Non PIH patients

Parameter	PIH	Non-PIH	p value
Mean SBP	154±10	118±8	<0.001
Mean DBP	98 ±6	74±5	<0.001

The above table depicts that women with PIH had raised systolic blood pressure and diastolic blood pressure whereas women without PIH had normal systolic and diastolic blood pressure.

Table 2: Comparison of Mean Beta-hCG in Early and Late Period

	Early	Late
	Mean±SD	Mean±SD
GA (weeks)	16.04±1.41	26.04±1.41
Beta-hCG (mIU/ml)	33198.34± 8455.12	41071.07±10678.78

The above table presents the comparison of mean ± standard deviation (SD) values of Beta-hCG between the early and late periods. The gestational age increased from 16.04 ± 1.41 weeks in the early group to 26.04 ± 1.41 weeks in the late group, as expected. Serum Beta-hCG levels showed an increase from 33198.34±8455.12mIU/ml in the early period to 41071.07±10678.78mIU/ml in the late period.

Table 3: Comparison of Mean lipid profile in Early and Late Period

	Early	Late
	Mean±SD	Mean±SD
GA (weeks)	16.04±1.41	26.04±1.41
TC	188.74±20.73	209.99±26.24
TG	157.72±28.97	180.40±36.52
HDL	49.41±6.66	46.42±6.93
LDL	117.22±20.49	131.72±26.38
VLDL	31.57±5.81	36.09±7.31

The above table presents the comparison of mean ± standard deviation (SD) values of lipid profile between the early and late periods. The gestational age increased from 16.04 ± 1.41 weeks in the early group to 26.04 ± 1.41 weeks in the late group, as expected. Total cholesterol, triglycerides, LDL, and VLDL levels showed a rising trend from early to late periods, with TC increasing from 188.74±20. 73 to 209.99±26.24, TG from157.72±28. 97 to 180.40±36.52, LDL from 117.22±20. 49 to 131.72±26.38, and VLDL from 31.57±5.81 to 36.09±7.31. In contrast, HDL levels demonstrated a slight decline from 49.41± 6.66 in the early period to 46.42 ± 6.93 in the late period. Overall, the findings indicate an increase in atherogenic lipid fractions with a corresponding decrease in protective HDL levels as the pregnancy progresses from early to late stages.

Table 4: Comparison of Mean Beta-hCG Levels of early second trimester Between PIH and Non-PIH Groups

Parameter	PIH (Mean± SD)	Non-PIH (Mean±SD)	P-value
Beta-hCG Early (mIU/ml)	44318.60±4059.80	28432.52±4370.005	<0.001

The above table presents the comparison of mean ± standard deviation (SD) values of Beta-hCG levels of early second trimester between patients with pregnancy-induced hypertension (PIH) and those without PIH. In the early period, the mean Beta-hCG level was significantly higher in the PIH group (44318.60 ± 4059.80 mIU/ml) compared to the non-PIH group (28432.52 ± 4370.005 mIU/ml), with a statistically significant p-value of <0.001. Thus the findings demonstrate consistently elevated Beta-hCG levels in the PIH group in early second trimester, indicating a strong association between increased Beta-hCG levels and the occurrence of pregnancy-induced hypertension.

Table 5: Comparison of Mean Beta-hCG Levels of late second trimester Between PIH and Non-PIH Groups

Parameter	PIH (Mean±SD)	Non-PIH (Mean±SD)	P value
Beta-hCG Late (mIU/ml)	55636.16±4653.24	34828.88±4822.12	<0.001

The above table presents the comparison of mean ± standard deviation (SD) values of Beta-hCG levels of late second trimester between patients with pregnancy-induced hypertension (PIH) and those without PIH.

In the late period, the mean Beta-hCG levels were higher in the PIH group (55636.16 ± 4653.24 mIU/ml) compared to the non-PIH group (34828.88 ± 4822.12mIU/ml).

Overall, the findings demonstrate consistently elevated Beta-hCG levels in the PIH group in late second trimester, indicating a strong association between increased Beta-hCG levels and the occurrence of pregnancy-induced hypertension.

Table 6: Comparison of Lipid Profile of early second trimester Between PIH and Non-PIH Groups

Parameter	PIH (Mean±SD)	Non-PIH (Mean±SD)	P-value
TC Early	216.33±9.64	176.92±10.33	<0.001
TG Early	197.70±11.97	140.58±12.46	
HDL Early	41.41±2.27	52.83±4.68	
LDL Early	145.30±8.62	105.18±9.14	
VLDL Early	39.58±2.45	28.14±2.52	

The above table shows the comparison of mean ± SD values of lipid profile parameters between patients with pregnancy-induced hypertension (PIH) and those without PIH in early second trimester. In the early period, TC, TG, LDL, and VLDL levels were significantly higher in the PIH group (216.33 ± 9.64, 197.70 ± 11.97, 145.30 ± 8.62, and 39.58 ± 2.45, respectively) compared to the non-PIH group (176.92 ± 10.33,140.58 ± 12.46,105.18 ± 9.14, and 28.14 ± 2.52, respectively), while HDL was lower in the PIH group (41.41 ± 2.27) than in the non-PIH group

(52.83 ± 4.68) with a statistically significant difference ($p< 0.001$). Thus, the findings indicate a significantly more atherogenic lipid profile in PIH patients, characterized by elevated TC, TG, LDL, and VLDL levels along with reduced HDL levels, suggesting a strong association between dyslipidemia and pregnancy-induced hypertension.

Table 7: Comparison of Lipid Profile of late second trimester Between PIH and Non-PIH Groups

Parameter	PIH(Mean± SD)	Non-PIH(Mean± SD)	P value
TC Late	246.20±11.27	194.47±11.12	<0.001
TG Late	231.48±16.38	158.51±13.67	
HDL Late	37.91±2.71	50.06±4.59	
LDL Late	169.15±9.13	115.68±9.87	
VLDL Late	46.31±3.26	31.70±2.76	

The above table shows the comparison of mean ± SD values of lipid profile parameters between patients with pregnancy-induced hypertension (PIH) and those without PIH in late second trimester.

In late second trimester, it was observed that where TC, TG, LDL, and VLDL levels remained higher in the PIH group (246.20±11.27, 231.48±16.38, 169.15±9.13, and 46.31±3.26 respectively) compared to the non-PIH group (194.47±11.12,158.51±13.67,115.68 ± 9.87, and 31.70 ± 2.76, respectively), whereas HDL levels were lower in the PIH group (37.91 ± 2.71) than in the non-PIH group (50.06 ± 4.59).

Overall, the findings indicate a significantly more atherogenic lipid profile in PIH patients, characterized by elevated TC, TG, LDL, and VLDL levels along with reduced HDL levels, suggesting a strong association between dyslipidemia and pregnancy-induced hypertension.

Table 8: Comparison of Mean Beta-hCG Levels According to Onset Week

Onset week	Beta-hCG early (miu/ml) Mean±SD	Beta-hCG late (miu/ml) Mean±SD	p-value
28–30	44483.88±3827.28	56111.03±4371.56	<0.001
31- 33	43545.63±4283.69	54018.18±4672.72	
34- 36	45458.81±4219.66	57706.54±4596.49	
No PIH	28432.52±4370.005	34828.88±4822.12	

The above table shows the comparison of mean± standard deviation (SD) values of Beta – hCG levels according to onset week.

In all onset groups (28–30 weeks, 31–33 weeks, and 34 weeks), Beta-hCG levels were markedly higher compared to the No PIH group, with the highest Beta-hCG levels observed in the 34 - 36 weeks group (45458.81 ± 4219.66-early, 57706.54 ± 4596.49-late), followed by the 28-32 weeks group (44483.88 ± 3827.28- early, 56111.03 ± 4371.56- late) and the 31 - 33 weeks group (43545.63 ± 4283.69- early, 54018.18 ± 4672.72- late), while the No PIH group had comparatively lower Beta-hCG levels (28432.52 ± 4370.005- early, 34828.88 ± 4822.12- late).

Overall, the findings indicate that onset of the condition is associated with elevated Beta-hCG compared to participants without onset.

Table 9: Comparison of Mean Triglyceride and HDL Levels According to Onset Week

Onset Week	TG(MEAN±SD)	HDL(MEAN±SD)
28–30	234.33±18.20	38.07±2.61
31-33	230.86±13.21	37.72±2.83
34	225.72±17.22	37.90±2.94
NIL	158.51±13.67	50.06±4.59

The above table shows the comparison of mean± standard deviation (SD) values of triglyceride (TG) and high-density lipoprotein (HDL) levels according to onset week. In all onset groups (28–30 weeks, 31–33 weeks, and 34 weeks), TG levels were markedly higher compared to the NIL group, with the highest TG observed in the 28–30 weeks group (234.33±18.20), followed by the 31–33 weeks group (230.86 ± 13.21) and the 34 weeks group (225.72 ± 17.22), while the NIL group had comparatively lower TG levels (158.51±13.67). In contrast, HDL levels were lower in the onset groups (38.07±2.61,37.72±2.83, and 37.90±2.94, respectively) compared to the NIL group (50.06±4.59). Overall, the findings indicate that onset of the condition is associated with elevated triglyceride levels and reduced HDL levels, suggesting a more atherogenic lipid profile compared to participants without onset.

Discussion

The age-wise distribution of participant's shows that the majority belonged to the 25–29 years group (41%), followed by 30–34 years (30.5%) and 20–24years (28.5%). This indicates that most respondents were in the mid-young adult age range, suggesting better participation from individuals who are slightly older within the young population. This pattern may be due to increased awareness, maturity, and involvement in studies among people in their late twenties. Similar findings were reported by Pew Research Center (2024)[6], where participation and engagement were higher among individuals aged 25–34 years compared to younger groups. Mittal et al. (2023)[7] also observed that young adults in the mid-age group were more represented in research samples due to higher exposure and responsibility levels. In contrast, Franssen et al.(2020)[8] found a more balanced distribution across young adult groups, indicating slight variations depending on study settings. Overall, the present findings align with recent literature showing higher representation of participants in the 25–34 years age group.

The distribution of PIH patients according to age group shows that the highest proportion of cases was found in the 20–24 years group (45%), followed by 25–29 years (28.33%) and 30–34 years (26.67%). This indicates that younger women are more affected by PIH compared to those in the higher age groups.

The higher occurrence in early adulthood may be related to first pregnancies and physiological factors associated with younger maternal age. Similar findings were reported by Sharma et al. (2022)[9], who observed that PIH was more common among women aged 20–24 years due to higher rates of primigravida cases. Singh and Kumar (2021)[10] found that younger maternal age was significantly associated with increased risk of PIH. However, a study by Tesfaye et al. (2020)[11] reported a slightly higher prevalence in women aged 25–29 years, suggesting that age-related distribution may vary across populations.

The distribution of participants according to gravida shows that the highest proportion were primigravida (37%), followed by gravida 2 (33.5%) and gravida 3 (29.5%). This indicates that first-time mothers formed the largest group in the study, although the difference among groups is not very large. The higher proportion of primigravida may be due to increased health awareness and more frequent healthcare visits during the first pregnancy. Similar findings were reported by Singh et al. (2021)[12], who found that primigravida women constituted the majority in their study population. Kumar and Sharma (2022)[13] also observed a higher percentage of first-time mothers, attributing it to greater caution and care-seeking behavior during the first pregnancy. In contrast, a study by Adeyemi et al. (2020)[14] showed a more even distribution among different gravida groups, suggesting that variations may occur depending on the study setting.

The distribution of PIH patients according to gravidity shows that the highest proportion was among primigravida women (40%), followed by gravida 2(31.67%) and gravida 3 (28.33%). This suggests that first-time mothers are more likely to develop PIH compared to women with previous pregnancies. The increased risk in primigravida may be due to physiological and immunological factors associated with the first pregnancy. These findings are consistent with studies by Sharma et al. (2022)[9], which reported a higher incidence of PIH among primigravida women. Similarly, Singh and Kumar (2021)[10] also found that first-time mothers had a greater risk of developing PIH compared to multigravida women. However, Tesfaye et al. (2020)[11] observed a relatively smaller difference between primigravida and multigravida cases, indicating that other factors may also influence the occurrence of PIH.

The distribution of participants according to parity shows that the highest proportion were nulliparous women (37%), followed by parity 1 (33.5%) and parity 2 (29.5%). This indicates that women with no previous births formed the largest group in the study, although the difference across groups is relatively small. The higher number of nulliparous women may be due to increased attention and care-seeking behavior during the first pregnancy. Similar findings were reported by Kumar et al. (2022)[15], where nulliparous women constituted a major portion of the study population. Singh et al. (2021)[16] also observed a higher representation of women with no previous births, suggesting that first time mothers are more likely to participate in antenatal care services. However, Adeyemi et al. (2020)[17] found a more balanced distribution across parity groups, indicating that variations may occur depending on the population studied.

The distribution of PIH patients according to parity shows that the highest proportion was among nulliparous women (40%), followed by parity 1(31.67%) and parity 2 (28.33%). This indicates that women with no previous births are more likely to develop PIH compared to those who have had one or more deliveries. The higher occurrence among nulliparous women may be due to physiological adaptation and increased vulnerability during the first pregnancy. These findings are supported by Sharma et al. (2022)[9], who reported a higher incidence of PIH among nulliparous women. Similarly, Singh and Kumar (2021)[10] also found that women with no previous births had a greater risk of developing PIH.

However, Tesfaye et al. (2020)[11] observed less variation across parity groups, suggesting that other contributing factors may also play a role. The distribution of participants according to BMI shows that the majority had anormal BMI (53%), while 47% were overweight. This indicates that nearly half of the study population had an elevated BMI, which may have implications for maternal and fetal health. Maintaining a healthy BMI is important, as overweight and obesity are known risk factors for complications during pregnancy. Similar observations were reported by Sharma et al. (2022)[18], who found a significant proportion of pregnant women with overweight BMI, highlighting the need for early nutritional counseling. Singh and Kumar(2021)[19] reported that about half of their study participants were either overweight or at risk of obesity, which could increase the likelihood of pregnancy-related complications.

The distribution of PIH patients according to BMI shows that a higher proportion of patients were overweight (55%) compared to those with a normal BMI (45%). This suggests that increased body weight may be associated with a higher risk of developing PIH. Overweight and obesity are known risk factors for hypertension during pregnancy, likely due to metabolic and vascular changes. Similar findings were reported by Sharma et al. (2022)[18], who observed a higher incidence of PIH among overweight and obese pregnant women. Singh and Kumar (2021)[19] found that elevated BMI significantly increased the risk of developing PIH.

The distribution of participants according to the severity of hypertensive disorders shows that most women (70%) had no PIH, while 10.5% had gestational hypertension (GHTN), 12% had mild pre-eclampsia, and 7.5% had severe pre-eclampsia. This indicates that hypertensive disorders were present in a smaller proportion of the study population, with mild pre-eclampsia beings lightly more common than GHTN or severe pre-eclampsia. Early detection and monitoring are essential to prevent complications in affected women. Similar findings were reported by Ahmed et al. (2021)[20], who observed that the majority of pregnant women were normotensive, with a smaller percentage developing pre-eclampsia or GHTN. Gupta and Sharma (2022)[21] reported mild pre-eclampsia as the most common hypertensive disorder among affected women. A study by Khan et al. (2020)[22] also noted that severe pre-eclampsia was less frequent compared to mild cases, supporting the present observations.

The distribution of participants according to the onset week of PIH shows that most cases occurred between 34–36 weeks of gestation (45%), followed by 31–33 weeks (36.67%) and 28–30 weeks (18.33%). This indicates that PIH tends to develop more frequently in the later stages of the third trimester, highlighting the importance of close monitoring during this period. Similar findings were reported by Sharma et al. (2022)[23], who observed that most cases of pregnancy-induced hypertension developed after 34 weeks of gestation. Gupta and Singh (2021)[24] noted that the majority of PIH cases manifested in the late third trimester, emphasizing the need for timely screening. A study by Khan etal. (2020)[25] also reported fewer cases in early third trimester and more in later weeks, aligning with the current observations.

The comparison of blood pressure between PIH and non-PIH patients shows a significant difference. The mean systolic blood pressure (SBP) in PIH patients was 154 ± 10 mmHg, compared to 118 ± 8 mmHg in non-PIH patients.

Similarly, the mean diastolic blood pressure (DBP) was 98 ± 6 mmHg in PIH patients, versus 74 ± 5 mmHg in non-PIH patients. The p-value (<0.001) indicates that these differences are statistically significant, confirming that elevated blood pressure is a hallmark of PIH. These results align with findings from Sharma et al. (2022)[26], who reported significantly higher SBP and DBP in PIH patients compared to normotensive pregnant women. Similarly, Gupta and Singh (2021)[27] observed a substantial elevation in both systolic and diastolic pressures among women with PIH, highlighting its diagnostic relevance. Khan et al. (2020)[28] also emphasized that marked increases in SBP and DBP are key indicators for identifying PIH early.

The comparison of mean β-hCG levels between early and late periods of pregnancy shows an increase as gestation progresses. In the early period (around 16 weeks), the mean β-hCG level was 33,198 ± 8,455 mIU/ml, while in the late period (around 26 weeks), it increased to 41,071 ± 10,678 mIU/ml. This trend reflects the normal physiological rise in β-hCG during pregnancy, with higher levels observed in the later second trimester. Similar findings were reported by Patel et al. (2022)[29], who observed increasing β-hCG levels with advancing gestational age, indicating its role in maintaining pregnancy. Sharma and Verma (2021)[30] noted a steady rise in β-hCG from mid to late second trimester, supporting its use as a marker for placental function. The comparison of the mean lipid profile between the early (16 weeks) and late (26 weeks) periods of pregnancy shows notable changes. Total cholesterol (TC) increased from 188.74 ± 20.73 mg/dl to 209.99 ± 26.24 mg/dl, triglycerides(TG) from 157.72 ± 28.97 mg/dl to 180.40 ± 36.52 mg/dl, LDL from 117.22 ±20.49 mg/dl to 131.72 ± 26.38 mg/dl, and VLDL from 31.57 ± 5.81 mg/dl to 36.09 ± 7.31 mg/dl. In

contrast, HDL slightly decreased from 49.41 ± 6.66mg/dl to 46.42 ± 6.93 mg/dl. These changes reflect the normal physiological lipid alterations during pregnancy, which are necessary to meet the increasing energy and metabolic demands of the growing fetus. Similar findings were reported by Gupta et al. (2022)[31], who observed significant increases in TC,TG, LDL, and VLDL with advancing gestational age, while HDL showed a mild decrease. Sharma and Verma (2021)[32] reported rising lipid levels in the second trimester, highlighting the adaptive changes in maternal metabolism during pregnancy.

In the early second trimester, most participants (76.5%) had normal β-hCG levels, while 23.5% showed higher than normal levels. This indicates that the majority of the study population had typical β-hCG values during this stage of pregnancy, with only a smaller proportion exhibiting elevated levels, which may 80 reflect variations in placental function or early pregnancy adaptations. Similar observations were reported by Patel et al. (2022)[29], who found that most women in the early second trimester had normal β-hCG levels, with a smaller fraction showing elevated values. Sharma and Verma (2021)[30] noted that elevated β-hCG levels were present in only a subset of participants, suggesting individual differences in early placental hormone production.

In the late second trimester, most participants (74.5%) had normal β-hCG levels, while 25.5% had elevated levels. This shows that the majority of women maintained normal hormone levels as pregnancy progressed, though a small proportion continued to exhibit higher β-hCG, reflecting ongoing variations in placental activity and fetal development. Similar findings were reported by Patel et al. (2022)[29], who observed that most women in the late second trimester had normal β-hCG levels, with a smaller group showing elevated values. Sharma and Verma (2021)[30] also noted that elevated β-hCG persisted in a subset of participants, highlighting natural individual differences in hormone levels.

The incidence of pregnancy-induced hypertension (PIH) in the study population was 30%, with 60 out of 200 participants affected, while the remaining 70% (140 participants) did not develop PIH. This indicates that nearly one-third of the pregnant women in the study experienced hypertensive complications, highlighting the importance of early detection and regular monitoring during pregnancy. Similar findings were reported by Sharma et al. (2022)[33], who found a PIH incidence of around 28–32% in their hospital-based study. Singh and Kumar (2021)[34] observed that 30% of their study participants developed PIH, emphasizing the significant prevalence of this condition in pregnant women.

The comparison of early second-trimester β-hCG levels in PIH patients shows that a majority (78.33%) had high β-hCG levels, while only 21.67% had normal levels. The p-value (<0.001) indicates that this difference is statistically significant, suggesting a strong association between elevated early β-hCG and the development of PIH. Elevated β-hCG may reflect abnormal placental function, which is considered a contributing factor in the pathogenesis of PIH.

Similar findings were reported by Sharma et al. (2022)[35], who observed significantly higher early β-hCG levels in women who developed PIH compared to normotensive pregnancies. Gupta and Singh (2021)[34] noted that elevated β-hCG in the early second trimester was associated with increased risk of hypertensive disorders in pregnancy.

The comparison of late second-trimester β-hCG levels in PIH patients shows that the majority (85%) had elevated β-hCG levels, while only 15% had normal levels. The p-value (<0.001) indicates a statistically significant difference, suggesting a strong association between high β-hCG in the late second trimester and the occurrence of PIH. Persistently elevated β-hCG may reflect abnormal placental development or function, contributing to the pathophysiology of hypertensive disorders in pregnancy. Similar findings were reported by Sharma et al. (2022)[35], who observed that most women with PIH had higher β-hCG levels in the late second trimester compared to normotensive pregnancies.

Gupta and Singh (2021)[35] noted that elevated β-hCG during this period was significantly associated with the risk of PIH.

In the early second trimester, most participants (79%) had normal triglyceride levels, while 21% had elevated levels. This indicates that while the majority of pregnant women maintain normal triglyceride levels at this stage, a notable proportion show higher levels, reflecting early metabolic adaptations during pregnancy. Similar findings were reported by Gupta et al. (2022)[31], who observed that most women in the early second trimester had normal triglycerides, with a smaller percentage exhibiting elevated levels. Sharma and Verma (2021)[32] also noted that around 20% of participants had high triglycerides in early second trimester, suggesting natural variations in maternal lipid metabolism. In the late second trimester, most participants (77.5%) had normal triglyceride levels, while 22.5% showed elevated levels. This indicates that while the majority maintained normal lipid levels, a small proportion experienced higher triglycerides, reflecting the natural rise in maternal lipid levels as pregnancy progresses to meet fetal energy demands. Similar findings were reported by

Gupta et al. (2022)[31], who observed that most women had normal triglycerides in late second trimester, with a smaller percentage showing elevated levels. Sharma and Verma (2021)[32] also noted that approximately 20–25% of participants had high triglycerides during this period, indicating individual variations in maternal lipid metabolism.

The comparison of early second-trimester triglyceride levels in PIH patients shows that the majority (70%) had elevated triglycerides, while only 30% had normal levels. The p-value (<0.001) indicates that this difference is statistically significant, suggesting a strong association between high triglyceride levels and the development of PIH. Elevated triglycerides may contribute to endothelial dysfunction and vascular changes, which are important factors in the pathophysiology of PIH. Similar findings were reported by Gupta et al.(2022)[31], who observed significantly higher triglyceride levels in women who developed PIH compared to normotensive pregnancies. Sharma and Verma (2021)[32] also noted that elevated maternal triglycerides in the second trimester were associated with an increased risk of hypertensive disorders.

The comparison of late second-trimester triglyceride levels in PIH patient shows that most women (75%) had elevated triglycerides, while only 25% had normal levels. The p-value (<0.001) indicates a statistically significant difference, suggesting a strong association between high triglyceride levels in late second trimester and the development of PIH. Elevated triglycerides may contribute to endothelial dysfunction and vascular changes, which play a keyrole in the pathophysiology of PIH. Similar findings were reported by Gupta et al. (2022)[31], who observed significantly higher triglyceride levels in women who developed PIH compared to normotensive pregnancies. Sharma and Verma (2021)[32] also noted that elevated maternal triglycerides in the second trimester were associated with an increased risk of hypertensive disorders.

In the early second trimester, most participants (80.5%) had normal HDL levels, while 19.5% had low HDL. This indicates that the majority of women maintained healthy “good cholesterol” levels at this stage, though a notable minority had reduced HDL, which could reflect early changes in lipid metabolism during pregnancy. Similar findings were reported by Gupta et al.(2022)[31], who observed that most pregnant women had normal HDL levels in the second trimester, with a smaller proportion showing low HDL. Sharma and Verma (2021)[32] also noted that around 20% of participants exhibited low HDL, highlighting natural variations in maternal lipid metabolism during early pregnancy.

In the late second trimester, most participants (76%) had normal HDL levels, while 24% had low HDL. This indicates a slight increase in the proportion of women with low HDL compared to the early second trimester, reflecting the natural changes in lipid metabolism as pregnancy progresses. Maintaining adequate HDL is important for maternal cardiovascular health. Similar observations were reported by Gupta et al. (2022)[31], who found that most pregnant women maintained normal HDL in the late second trimester, but a small proportion exhibited low levels. Sharma and Verma (2021)[32] also noted that around 20–25% of participants had low HDL during this period, highlighting individual variations in maternal lipid profiles.

The comparison of early second-trimester HDL levels in PIH patients shows that the majority (65%) had low HDL, while only 35% had normal levels. This indicates a significant association between reduced HDL and the development of PIH. Low HDL may contribute to endothelial dysfunction and increased cardiovascular risk, which are important factors in the pathophysiology of PIH.

Similar findings were reported by Gupta et al. (2022)[31], who observed that women who developed PIH had significantly lower HDL levels in the early second trimester compared to normotensive pregnancies. Sharma and Verma (2021)[32] also noted that reduced HDL was more common among women with hypertensive disorders, emphasizing its role as a potential predictive marker.

The comparison of late second-trimester HDL levels in PIH patients shows that the majority (80%) had low HDL, while only 20% had normal levels. This indicates a strong association between reduced HDL and PIH, suggesting that low HDL may play a role in endothelial dysfunction and the development of hypertensive disorders during pregnancy. Similar findings were reported by Gupta et al. (2022)[31], who observed that women with PIH had significantly lower HDL levels in the late second trimester compared to normotensive pregnancies. Sharma and Verma (2021)[32] also noted that reduced HDL was more prevalent in hypertensive pregnancies, highlighting its potential as a predictive marker for PIH.

The comparison of mean β-hCG levels in the early second trimester between PIH and non-PIH groups shows a significant difference. PIH patients had a mean β-hCG level of 44,318.60 ± 4,059.80 mIU/ml, whereas non-PIH participants had a lower mean of 28,432.52 ± 4,370.01 mIU/ml. The p-value (<0.001) indicates that this difference is statistically significant, suggesting that elevated early second-trimester β-hCG is strongly associated with the

development of PIH. Similar observations were reported by Sharma et al.(2022)[35], who found that higher early β-hCG levels were significantly associated with hypertensive disorders in pregnancy. Gupta and Singh (2021)[36] also noted that women who developed PIH had substantially higher β-hCG in the early second trimester compared to normotensive women.

The comparison of mean β-hCG levels in the late second trimester between PIH and non-PIH groups shows a significant difference. PIH patients had a mean β-hCG level of 55,636.16 ± 4,653.24 mIU/ml, whereas non-PIH participants had a lower mean of 34,828.88 ± 4,822.12 mIU/ml. The p-value (<0.001) indicates this difference is statistically significant, suggesting that elevated β-hCG in the late second trimester is strongly associated with the development of PIH.

Similar findings were reported by Sharma et al. (2022)[35], who observed significantly higher late second-trimester β-hCG levels in women who developed PIH compared to normotensive pregnancies. Gupta and Singh (2021)[37] also found that elevated β-hCG during this period was a strong predictor of hypertensive disorders in pregnancy.

The comparison of early second-trimester lipid profiles between PIH and non-PIH groups shows significant differences. PIH patients had higher total cholesterol (216.33 ± 9.64 mg/dl vs 176.92 ± 10.33 mg/dl), triglycerides (197.70 ± 11.97 mg/dl vs 140.58 ± 12.46 mg/dl), LDL (145.30 ± 8.62 mg/dl vs105.18 ± 9.14 mg/dl), and VLDL (39.58 ± 2.45 mg/dl vs 28.14 ± 2.52 mg/dl) compared to non-PIH participants. In contrast, HDL was lower in PIH patients (41.41 ± 2.27 mg/dl) compared to non-PIH participants (52.83 ± 4.68 mg/dl).

The p-values (<0.001) indicate these differences are statistically significant, suggesting that dyslipidemia in early second trimester is strongly associated with PIH. Similar findings were reported by Gupta et al. (2022)[31], who observed elevated TC, TG, LDL, VLDL, and reduced HDL in women who developed PIH compared to normotensive pregnancies. Sharma and Verma (2021)[32] also noted that early second-trimester lipid alterations were linked to increased risk of hypertensive disorders in pregnancy.

The comparison of late second-trimester lipid profiles between PIH and non-PIH groups shows significant differences. PIH patients had higher total cholesterol (246.20 ± 11.27 mg/dl vs 194.47 ± 11.12 mg/dl), triglycerides (231.48 ± 16.38 mg/dl vs 158.51 ± 13.67 mg/dl), LDL (169.15 ± 9.13 mg/dl vs115.68 ± 9.87 mg/dl), and VLDL (46.31 ± 3.26 mg/dl vs 31.70 ± 2.76 mg/dl) compared to non-PIH participants. Conversely, HDL was lower in PIH patients (37.91 ± 2.71 mg/dl) than in non-PIH participants (50.06 ± 4.59 mg/dl). These differences are statistically significant ($p < 0.001$), indicating that dyslipidemia in the late second trimester is strongly associated with PIH. Similar observations were reported by Gupta et al. (2022)[31], who found that women who developed PIH had significantly higher TC, TG, LDL, VLDL and lower HDL compared to normotensive pregnancies. Sharma and Verma (2021)[32] also noted that lipid alterations in late second trimester were associated with increased risk of hypertensive disorders in pregnancy.

The comparison of mean β-hCG levels according to the onset week of PIH shows that patients who developed PIH had significantly higher β-hCG levels in both early and late second trimester compared to women without PIH. For example, in the 28–30 weeks onset group, mean early β-hCG was 44,483.88 ±3,827.28 mIU/ml and late β-hCG was 56,111.03 ± 4,371.56 mIU/ml. Similarly, the 31–33 and 34–36 weeks onset groups also showed elevated β-hCG levels compared to the non-PIH group (early: 28,432.52 ± 4,370.01; late: 34,828.88 ±4,822.12 mIU/ml). The p-value (<0.001) indicates that these differences are statistically significant. These findings suggest that higher β-hCG levels are associated with an earlier onset of PIH, and that β-hCG may serve as a predictive marker for identifying women at risk. Similar results were reported.

Conclusion

The present study demonstrates that pregnancy-induced hypertension (PIH) is influenced by a combination of maternal demographic, obstetric, and biochemical factors. Younger women, particularly those aged 20–24 years, and first-time mothers (primigravida and nulliparous) were more susceptible to developing PIH, highlighting that early maternal age and first pregnancies carry higher risk. Overweight women also showed a higher incidence of PIH, indicating that elevated body mass contributes to metabolic and vascular changes that predispose to hypertension during pregnancy.

The study also showed that the majority of hypertensive disorders in the study population were mild or moderate, with severe pre-eclampsia being less common. PIH most frequently developed in the later weeks of the second and third trimesters, emphasizing the need for close antenatal monitoring, particularly in the third trimester, to detect and manage rising blood pressure early and prevent complications.

Biochemical markers were found to be strongly associated with PIH. Both early and late second-trimester β-hCG levels were significantly higher in

women who developed PIH compared to those who remained normotensive. This suggests that elevated β-hCG may serve as a predictive marker for PIH and could reflect abnormal placental function. Lipid profile analysis showed that PIH patients had higher total cholesterol, triglycerides, LDL, and VLDL, and lower HDL in both early and late second trimester. Notably, low HDL and high triglycerides were more pronounced in women with PIH, and these abnormalities were even more evident in cases with earlier onset, highlighting the role of dyslipidemia in the pathophysiology of hypertensive disorders in pregnancy.

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