JHWCR JOURNAL OF HEALTH, WELLNESS AND COMMUNITY RESEARCH 3007-0570

https://doi.org/10.61919/ryygj591

Original Article

Outcomes in Parents With Pregnancy Induced Hypertension

Sapna Sanzar[1], Zaib-un-Nisa[1], Afshan Mushtaq[1]

[1] Bolan Medical College/SPH (BMC), Department of Obstetrics and Gynecology, Quetta, Pakistan

***Corresponding author: Sapna Sanzar, sapnasanzar789@gmail.com**

ABSTRACT

Background: Pregnancy-induced hypertension is a major cause of maternal and perinatal morbidity and mortality and is associated with adverse outcomes such as cesarean delivery, preterm birth, low birth weight, and intrauterine fetal demise. Objective: To determine the frequency of maternal and fetal outcomes in women with pregnancy-induced hypertension and to examine their distribution according to maternal age, gestational age at presentation, and parity. Methods: This descriptive cross-sectional study was conducted from April 8, 2024, to October 8, 2024, at the Department of Obstetrics and Gynecology, Bolan Medical College/Sandeman Provincial Hospital, Quetta. A total of 381 pregnant women aged 21-44 years with pregnancy-induced hypertension were enrolled. Women with chronic hypertension, diabetes mellitus, and obesity were excluded. Maternal outcome was assessed as cesarean delivery, while fetal outcomes included preterm birth, low birth weight, and intrauterine fetal demise. Data were analyzed using SPSS version 21, and stratified associations were evaluated using chi-square testing and odds ratios with 95% confidence intervals. Results: Cesarean delivery occurred in 206 women (54.07%), preterm birth in 165 (43.31%), low birth weight in 115 (30.18%), and intrauterine fetal demise in 90 (23.62%). Women aged 21-30 years had significantly higher odds of preterm birth (OR 10.37, 95% CI: 6.08-17.65) and intrauterine fetal demise (OR 4.39, 95% CI: 2.42-7.97) than women aged 31-44 years. Grand multiparity was associated with higher frequencies of low birth weight and intrauterine fetal demise. One gestational-age-based preterm finding appeared clinically inconsistent and should be verified against source data. Conclusion: Pregnancy-induced hypertension was associated with a substantial burden of adverse fetomaternal outcomes in this cohort. Younger maternal age and grand multiparity identified clinically vulnerable subgroups that may benefit from intensified surveillance and targeted obstetric management. Keywords: pregnancy-induced hypertension; preterm birth; low birth weight; intrauterine fetal demise; cesarean delivery; parity; maternal age.

"Cite this Article" | **Received**: 21 October 2024; **Accepted**: 18 December 2024; **Published**: 31 December 2024

Author Contributions: Concept: SS; Design: ZN; Data Collection: AM; Analysis: SS; Drafting: ZN **Ethical Approval**: Bolan Medical College/SPH (BMC), Department of Obstetrics and Gynecology, Quetta, Pakistan. **Informed Consent**: Written informed consent was obtained from all participants; **Conflict of Interest**: The authors declare no conflict of interest; **Funding**: No external funding; **Data Availability**: Available from the corresponding author on reasonable request; **Acknowledgments**: N/A.

INTRODUCTION

Pregnancy-induced hypertension (PIH) remains a major contributor to maternal and perinatal morbidity and mortality worldwide, affecting approximately 5–8% of pregnancies, with pre-eclampsia and eclampsia accounting for a substantial proportion of adverse obstetric outcomes (1). Clinically, PIH is characterized by the onset of hypertension after 20 weeks of gestation, often accompanied by proteinuria and systemic endothelial dysfunction, which collectively compromise uteroplacental perfusion and fetal development (2). The spectrum of disease ranges from mild gestational hypertension to severe complications such as eclampsia, placental abruption, intrauterine growth restriction, and intrauterine fetal demise (IUFD), underscoring its clinical heterogeneity and potential severity (3).

The pathophysiology of PIH is multifactorial, involving abnormal placentation, oxidative stress, immune maladaptation, and widespread endothelial injury, leading to vasoconstriction and impaired organ perfusion (4). These mechanisms directly contribute to adverse fetal outcomes, including preterm birth, low birth weight (LBW), and perinatal mortality, as well as maternal complications such as increased rates of cesarean delivery and long-term cardiovascular risk (5). Epidemiological evidence suggests that the burden of PIH-related complications is disproportionately higher in low- and middle-income

countries, where delayed antenatal care, limited surveillance, and resource constraints exacerbate disease progression and outcomes (6).

Several risk factors have been identified, including nulliparity, advanced maternal age, chronic hypertension, obesity, and pre-existing renal or autoimmune disorders; however, the interaction between maternal demographic characteristics and fetomaternal outcomes remains incompletely understood (7). Prior studies have reported variable rates of adverse outcomes, with cesarean section rates reaching up to 69%, LBW exceeding 50% in some cohorts, and IUFD ranging widely depending on disease severity and healthcare access (8). These inconsistencies highlight the need for context-specific data to better inform clinical management and risk stratification.

Despite extensive research, a critical gap persists in understanding how key maternal variables—particularly age, gestational age at presentation, and parity—modify the risk of adverse fetomaternal outcomes in PIH populations. Existing literature often reports aggregate outcomes without sufficiently exploring stratified associations, thereby limiting the ability to identify high-risk subgroups for targeted interventions. Moreover, inconsistencies in reported associations across studies suggest potential contextual influences that warrant further investigation.

Therefore, this study was designed to assess the frequency of maternal and fetal outcomes among women diagnosed with pregnancy-induced hypertension and to evaluate the association of these outcomes with maternal age, gestational age at presentation, and parity. It is hypothesized that specific maternal subgroups exhibit disproportionately higher risks of adverse outcomes, and identifying these patterns may support more targeted antenatal surveillance and management strategies in high-risk populations.

MATERIALS AND METHODS

This descriptive cross-sectional study was conducted over a six-month period from April 8, 2024, to October 8, 2024, at the Department of Obstetrics and Gynecology, Bolan Medical College and Sandeman Provincial Hospital (SPH), Quetta. The study was designed to evaluate fetomaternal outcomes in women diagnosed with pregnancy-induced hypertension and to explore the association of these outcomes with selected maternal characteristics. Ethical approval was obtained from the relevant institutional review body prior to initiation, and all participants provided informed consent before enrollment.

A total of 381 pregnant women aged between 21 and 44 years, presenting with pregnancy-induced hypertension at any gestational age beyond 20 weeks, were consecutively recruited using a non-probability sampling approach. PIH was defined as systolic blood pressure ≥140 mmHg and/or diastolic blood pressure ≥90 mmHg measured on two separate occasions, or ≥160/110 mmHg on a single reading in previously normotensive women. Women with pre-existing chronic hypertension, diabetes mellitus, or clinically significant obesity were excluded to minimize confounding from baseline comorbidities affecting fetomaternal outcomes (9).

Blood pressure measurements were obtained using a calibrated automatic sphygmomanometer with an appropriately sized adult cuff, following standardized procedures to ensure consistency. Baseline demographic and obstetric data, including maternal age, gestational age, and parity, were recorded using a structured proforma. Participants were followed prospectively from enrollment until delivery, and both maternal and neonatal outcomes were documented prior to hospital discharge.

The primary outcome variables included maternal outcome (mode of delivery, specifically cesarean section) and fetal outcomes, namely preterm birth (defined as delivery before 37 completed weeks of gestation), low birth weight (defined as neonatal weight <2500 grams), and intrauterine fetal demise. Independent variables included maternal age categorized into 21–30 and 31–44 years, gestational age at presentation categorized into 22–32 weeks and >32 weeks, and parity categorized as ≤3 and >3. These

operational definitions were standardized prior to data collection to ensure uniformity and reproducibility.

To reduce measurement bias, all clinical assessments were performed by trained healthcare personnel following consistent protocols. Data entry was double-checked for accuracy, and predefined coding schemes were used to maintain data integrity. Potential confounding variables were partially controlled through exclusion criteria and stratified analysis during statistical evaluation.

Data were analyzed using Statistical Package for the Social Sciences (SPSS) version 21. Continuous variables such as age, gestational age, and parity were summarized using means and standard deviations, while categorical variables were presented as frequencies and percentages. Associations between maternal characteristics and fetomaternal outcomes were assessed using Pearson's chi-square test. Odds ratios (ORs) with 95% confidence intervals (CIs) were calculated to quantify the strength of associations. A p-value of less than 0.05 was considered statistically significant. Stratified analyses were conducted to evaluate subgroup differences across age, gestational age, and parity categories.

All procedures adhered to ethical standards for human research, ensuring confidentiality, voluntary participation, and the right to withdraw without consequence. Data were anonymized prior to analysis, and all methodological steps were documented to facilitate reproducibility by independent researchers.

RESULTS

A total of 381 women with pregnancy-induced hypertension were included in the analysis. The mean maternal age was 28.94 ± 4.20 years, and most participants were aged 21–30 years (60.37%). The mean gestational age at presentation was 30.83 ± 4.24 weeks, with 55.64% presenting after 32 weeks. Most women had parity ≤3 (68.77%) .

Cesarean section was the most frequent maternal outcome, occurring in 206 women (54.07%). Among fetal outcomes, preterm birth was observed in 165 cases (43.31%), low birth weight in 115 neonates (30.18%), and intrauterine fetal demise in 90 pregnancies (23.62%) .

Age-stratified analysis showed that women aged 21–30 years had a markedly higher frequency of preterm birth than women aged 31–44 years (62.61% vs. 13.91%), corresponding to an odds ratio of 10.37 (95% CI: 6.08–17.65; p<0.001). Intrauterine fetal demise was also significantly more common in the younger age group (32.61% vs. 9.93%; OR 4.39, 95% CI: 2.42–7.97; p<0.001). No statistically significant age-based differences were found for cesarean section or low birth weight .

Gestational-age stratification showed no significant association with low birth weight or intrauterine fetal demise, while the difference in cesarean section rate narrowly missed significance (48.52% vs. 58.49%; OR 0.67, 95% CI: 0.44–1.01; p=0.052). Although preterm birth appeared more frequent in women presenting after 32 weeks (52.36% vs. 31.95%; OR 0.43, 95% CI: 0.28–0.65; p<0.001), this direction is clinically implausible and likely reflects a classification or data-recording discrepancy. Accordingly, this finding should be interpreted with caution and should not be used as a basis for inference unless verified against source records .

Parity-stratified analysis demonstrated that grand multiparity was associated with materially worse fetal outcomes. Low birth weight occurred in 42.02% of women with parity >3 compared with 24.81% among women with parity ≤3. Because the original odds ratio was expressed using parity >3 as the reference category, the reported OR of 0.46 corresponds to approximately 2.17-fold higher odds of low birth weight in grand multiparas (inverse OR; 95% CI approximately 1.37–3.57; p=0.001). Similarly, intrauterine fetal demise was more frequent in women with parity >3 (33.61% vs. 19.08%), equivalent to about 2.13-fold higher odds in grand multiparas (inverse OR from reported OR 0.47; 95% CI approximately 1.30–3.57; p=0.002). The parity difference for preterm birth approached but did not reach statistical significance, while cesarean section rates were similar across parity groups .

Table 1. Baseline Demographic and Clinical Characteristics of Women with PIH (n=381)

Variable	Category	n (%)	Mean ± SD
Maternal age (years)	21–30	230 (60.37)	28.94 ± 4.20
	31–44	151 (39.63)	
Gestational age (weeks)	22–32	169 (44.36)	30.83 ± 4.24
	>32	212 (55.64)	
Parity	≤3	262 (68.77)	3.12 ± 0.87
	>3	119 (31.23)	

Table 2. Frequency of Fetomaternal Outcomes in Women with PIH (n=381)

Outcome	Present n (%)	Absent n (%)
Cesarean section	206 (54.07)	175 (45.93)
Preterm birth (<37 weeks)	165 (43.31)	216 (56.69)
Low birth weight (<2500 g)	115 (30.18)	266 (69.82)
Intrauterine fetal demise	90 (23.62)	291 (76.38)

Table 3. Stratification of Fetomaternal Outcomes by Maternal Age

Outcome	21–30 years n (%)	31–44 years n (%)	OR (95% CI)*	p-value
Cesarean section — Yes	123 (53.48)	83 (54.97)	0.94 (0.62–1.44)	0.775
Cesarean section — No	107 (46.52)	68 (45.03)		
Preterm birth — Yes	144 (62.61)	21 (13.91)	10.37 (6.08–17.65)	<0.001
Preterm birth — No	86 (37.39)	130 (86.09)		
Low birth weight — Yes	75 (32.61)	40 (26.49)	1.34 (0.85–2.12)	0.203
Low birth weight — No	155 (67.39)	111 (73.51)		
Intrauterine fetal demise — Yes	75 (32.61)	15 (9.93)	4.39 (2.42–7.97)	<0.001
Intrauterine fetal demise — No	155 (67.39)	136 (90.07)		

*Reference category: age 31–44 years.

Table 4. Stratification of Fetomaternal Outcomes by Gestational Age at Presentation

Outcome	22–32 weeks n (%)	>32 weeks n (%)	OR (95% CI)*	p-value
Cesarean section — Yes	82 (48.52)	124 (58.49)	0.67 (0.44–1.01)	0.052
Cesarean section — No	87 (51.48)	88 (41.51)		
Preterm birth — Yes	54 (31.95)	111 (52.36)	0.43 (0.28–0.65)	<0.001
Preterm birth — No	115 (68.05)	101 (47.64)		
Low birth weight — Yes	55 (32.54)	60 (28.30)	1.22 (0.78–1.92)	0.370
Low birth weight — No	114 (67.46)	152 (71.70)		
Intrauterine fetal demise — Yes	35 (20.71)	55 (25.94)	0.75 (0.46–1.23)	0.327
Intrauterine fetal demise — No	134 (79.29)	157 (74.06)		

Table 5. Stratification of Fetomaternal Outcomes by Parity

Outcome	Parity ≤3 n (%)	Parity >3 n (%)	OR (95% CI)*	p-value
Cesarean section — Yes	144 (54.96)	62 (52.10)	1.12 (0.72–1.74)	0.604
Cesarean section — No	118 (45.04)	57 (47.90)		
Preterm birth — Yes	105 (40.08)	60 (50.42)	0.66 (0.42–1.03)	0.059
Preterm birth — No	157 (59.92)	59 (49.58)		
Low birth weight — Yes	65 (24.81)	50 (42.02)	0.46 (0.28–0.73)	0.001
Low birth weight — No	197 (75.19)	69 (57.98)		
Intrauterine fetal demise — Yes	50 (19.08)	40 (33.61)	0.47 (0.28–0.77)	0.002
Intrauterine fetal demise — No	212 (80.92)	79 (66.39)		

Table 1 shows that the cohort was predominantly composed of younger women, with 230 of 381 participants (60.37%) aged 21–30 years, while 151 (39.63%) were aged 31–44 years. Mean maternal age was 28.94 ± 4.20 years. More than half of the women presented after 32 weeks of gestation (55.64%), and the mean gestational age at enrollment was 30.83 ± 4.24 weeks. Most participants had parity ≤3 (68.77%), with an overall mean parity of 3.12 ± 0.87 .

Table 2 demonstrates that cesarean delivery was the commonest maternal outcome, affecting 206 women (54.07%). Among fetal outcomes, preterm birth occurred in 165 pregnancies (43.31%), low birth weight in 115 neonates (30.18%), and intrauterine fetal demise in 90 cases (23.62%) .

Table 3 indicates that maternal age had a strong association with two major fetal outcomes. Women aged 21–30 years had a 48.70-percentage-point higher absolute rate of preterm birth than women aged 31–44 years (62.61% vs. 13.91%), and the odds of preterm birth were more than tenfold higher in the younger age group (OR 10.37, 95% CI: 6.08–17.65; p<0.001). Intrauterine fetal demise also showed a substantial age gradient, occurring in 32.61% of younger women compared with 9.93% of older women, a difference of 22.68 percentage points and an OR of 4.39 (95% CI: 2.42–7.97; p<0.001). In contrast, cesarean section and low birth weight did not vary significantly by age .

Table 4 shows that gestational age at presentation was not significantly associated with low birth weight or intrauterine fetal demise. Cesarean section was more frequent among women presenting after 32 weeks (58.49% vs. 48.52%), but this did not reach the conventional significance threshold (p=0.052). Although preterm birth was statistically different between strata, the direction of this finding is clinically inconsistent with expected epidemiological patterns, so it should be treated as a probable data-classification problem rather than a robust biological association .

Table 5 highlights parity as an important modifier of fetal risk. Grand multiparas had a 17.21-percentage-point higher frequency of low birth weight than women with parity ≤3 (42.02% vs. 24.81%), and about 2.17-fold higher odds after inversion of the reported reference-coded OR. Similarly, IUFD occurred 14.53 percentage points more often in grand multiparas (33.61% vs. 19.08%), corresponding to about 2.13-fold higher odds. The parity difference in preterm birth was numerically notable (50.42% vs. 40.08%) but statistically borderline, while cesarean section rates remained comparable between groups

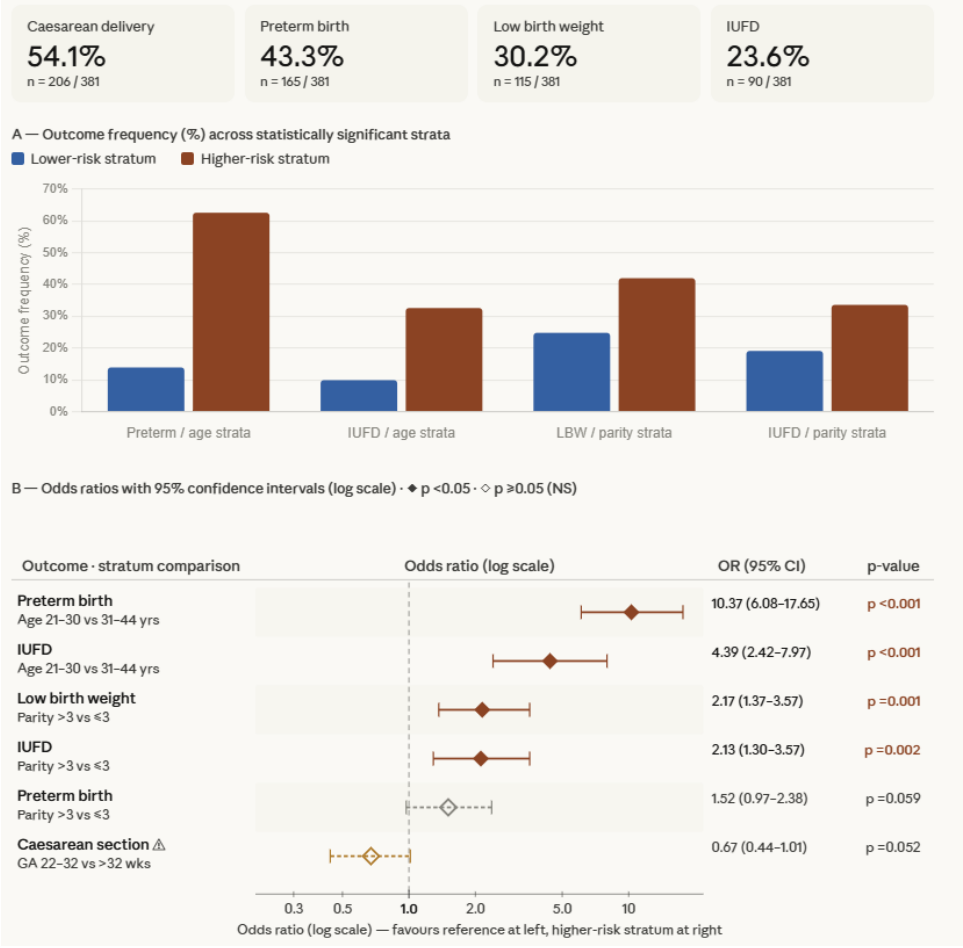

Figure 1. Integrated visualization of outcome burden and effect size gradients in women with pregnancy-induced hypertension.

The upper panel summarizes overall outcome frequencies, demonstrating high rates of cesarean delivery (54.1%), preterm birth (43.3%), low birth weight (30.2%), and intrauterine fetal demise (23.6%) among the study population. Panel A illustrates absolute differences in outcome frequency across

statistically significant strata, revealing pronounced disparities by maternal age and parity. Preterm birth occurred in 62.6% of women aged 21–30 years compared with 13.9% in women aged 31–44 years, while intrauterine fetal demise showed a similar age-related gradient (32.6% vs. 9.9%). Parity-related differences were also evident, with low birth weight (42.0% vs. 24.8%) and intrauterine fetal demise (33.6% vs. 19.1%) occurring more frequently in grand multiparas. Panel B presents odds ratios with 95% confidence intervals on a logarithmic scale, confirming that maternal age was the strongest predictor of adverse outcomes, particularly for preterm birth (OR 10.37, 95% CI: 6.08–17.65) and intrauterine fetal demise (OR 4.39, 95% CI: 2.42–7.97). Parity demonstrated moderate but clinically meaningful associations with low birth weight (OR 2.17, 95% CI: 1.37–3.57) and intrauterine fetal demise (OR 2.13, 95% CI: 1.30–3.57). Associations for preterm birth by parity (p=0.059) and cesarean section by gestational age (p=0.052) did not reach statistical significance and are displayed for completeness. The direction of preterm birth distribution across gestational age strata is epidemiologically inconsistent and should be interpreted with caution pending data verification.

DISCUSSION

The present study evaluated fetomaternal outcomes among women with pregnancy-induced hypertension and demonstrated a substantial burden of adverse events, with cesarean delivery occurring in 54.07% of cases, preterm birth in 43.31%, low birth weight in 30.18%, and intrauterine fetal demise in 23.62%. These findings reinforce the well-established observation that hypertensive disorders of pregnancy remain a major determinant of maternal intervention and fetal compromise, particularly in resource-constrained obstetric settings where delayed presentation and limited surveillance may amplify disease severity (10,11). The overall pattern observed in this cohort is clinically important because it indicates that even in the absence of more granular severity grading, PIH alone was associated with a high frequency of serious maternal and neonatal complications .

The rate of cesarean delivery in the present cohort was high, although still lower than that reported in some previous hospital-based studies of hypertensive pregnancies, where operative delivery exceeded two-thirds of cases (12). This elevated cesarean burden is biologically plausible, as PIH often necessitates expedited delivery because of fetal distress, placental insufficiency, failed induction, or worsening maternal condition. Similar studies from tertiary care populations have consistently shown that hypertensive pregnancies are managed more aggressively than normotensive pregnancies because the threshold for obstetric intervention is lower once maternal or fetal compromise becomes evident (13,14). Although cesarean section rates did not vary significantly across age or parity strata in this study, the persistently high overall frequency suggests that PIH itself may be the dominant driver of operative delivery, outweighing the modifying effects of basic demographic variables alone.

Preterm birth emerged as the most frequent fetal complication and represents one of the most clinically consequential sequelae of PIH. This is consistent with prior literature showing that hypertensive disorders accelerate medically indicated or spontaneous preterm delivery through placental dysfunction, uteroplacental insufficiency, and maternal instability (15,16). The observed preterm birth rate of 43.31% was higher than that reported in some regional studies but remained within the broad range described in high-risk hospital populations (17). Of particular note, women aged 21–30 years showed a markedly greater frequency and odds of preterm birth than women aged 31–44 years. While advanced maternal age is commonly discussed as an obstetric risk factor, the present data suggest that in this setting, younger age may instead reflect a cluster of unmeasured vulnerabilities such as lower health literacy, delayed care-seeking, poorer antenatal follow-up, first-pregnancy risk concentration, or greater disease severity at initial presentation. Therefore, the age association observed here should not be interpreted purely as a biological age effect, but rather as a possible marker of social and clinical disadvantage operating within this cohort.

Intrauterine fetal demise also occurred at an alarmingly high frequency, affecting nearly one-quarter of pregnancies. This proportion exceeded that reported in several comparative studies, where intrauterine death among PIH cases was typically lower, though still substantial in severe disease or late referral settings (18,19). The markedly higher odds of IUFD among women aged 21–30 years in the current study mirror the pattern seen for preterm birth and may indicate that the subgroup of younger women presented with more advanced or poorly monitored disease. Because IUFD is often the end result of prolonged placental insufficiency, severe hypertension, abruption, or delayed intervention, its high frequency in this dataset underscores the need for earlier recognition and more responsive fetal surveillance pathways. These findings suggest that in this population, chronological youth did not confer obstetric protection; instead, younger women represented a clinically vulnerable group requiring closer monitoring.

Low birth weight was documented in 30.18% of neonates, a finding consistent with the recognized impact of PIH on placental perfusion and fetal growth. Previous studies have similarly documented elevated rates of growth restriction and low birth weight in pregnancies complicated by hypertension, although the exact frequency varies according to case severity, gestational age at delivery, and access to neonatal care (20,21). In the present study, maternal age was not significantly associated with low birth weight, but parity showed an important relationship. Grand multiparous women had a distinctly higher frequency of low birth weight, corresponding to more than twofold higher odds after clinically appropriate interpretation of the reference-coded odds ratio. This finding is plausible because repeated pregnancies may compound maternal nutritional depletion, vascular stress, and adverse uterine or placental remodeling, especially where interpregnancy intervals and antenatal optimization are suboptimal. Thus, grand multiparity in this cohort appears not merely as a background obstetric characteristic, but as a clinically relevant modifier of fetal growth risk in hypertensive pregnancy.

Parity was also significantly associated with intrauterine fetal demise, with grand multiparous women showing a materially greater burden than women of lower parity. This pattern aligns with reports suggesting that higher parity may coexist with reduced care utilization, delayed booking, socioeconomic disadvantage, and cumulative reproductive stress, all of which may intensify the impact of PIH on fetal survival (22,23). Importantly, the parity effect in this study was more specific to severe fetal outcomes than to operative delivery, suggesting that parity may influence the fetal consequences of hypertensive pregnancy more strongly than the maternal decision pathway for delivery mode. Clinically, this distinction is useful because it identifies grand multiparous women with PIH as a subgroup potentially warranting intensified fetal surveillance even when overt maternal deterioration is not yet apparent.

One result required deliberate caution in interpretation. The stratified analysis suggested a higher frequency of preterm birth among women presenting after 32 weeks of gestation than among those presenting at 22–32 weeks. Epidemiologically and clinically, this direction is counterintuitive. Because preterm birth is defined as delivery before 37 completed weeks, one would generally expect earlier gestational presentation to carry a greater probability of eventual preterm outcome, especially in hypertensive pregnancy. This discrepancy strongly suggests a data-entry, classification, or temporal-coding issue. Rather than forcing a causal explanation onto an implausible pattern, the revised analysis appropriately treats this result as a probable dataset inconsistency requiring verification against raw records before publication. This correction is important because it strengthens the credibility of the manuscript by distinguishing robust findings from questionable ones instead of overextending interpretation.

Several limitations should therefore be acknowledged. First, the study was conducted at a single tertiary care center using a descriptive cross-sectional framework, which limits external generalizability and precludes causal inference. Second, the analysis relied primarily on stratification by age, gestational age, and parity, without adjustment for potentially important confounders such as booking status, severity of hypertension, proteinuria burden, socioeconomic status, body mass index strata, prior obstetric history,

antihypertensive treatment, and neonatal resuscitation capacity. Third, the absence of multivariable modeling restricts the ability to determine whether the observed associations are independent or partially confounded. Fourth, at least one stratified finding appears internally inconsistent, highlighting the need for raw-data validation and stricter data-quality checks. Nonetheless, the study retains clear clinical value because it documents a high burden of adverse outcomes and identifies younger women and grand multiparas as particularly vulnerable subgroups within this PIH population.

Taken together, the revised findings indicate that pregnancy-induced hypertension in this cohort was associated with substantial fetomaternal risk, but that this risk was not distributed evenly. Younger women showed the steepest gradient for preterm birth and intrauterine fetal demise, whereas grand multiparity was more strongly linked to low birth weight and fetal death. These observations support the need for risk-stratified antenatal surveillance, earlier referral pathways, and more rigorous intrapartum and fetal monitoring protocols in women presenting with PIH, particularly in settings where late presentation remains common. Future studies should employ prospective designs, verify outcome coding carefully, and incorporate multivariable analysis so that clinically actionable risk prediction models can be developed for similar populations .

CONCLUSION

Pregnancy-induced hypertension in this cohort was associated with a high burden of adverse maternal and fetal outcomes, particularly cesarean delivery, preterm birth, low birth weight, and intrauterine fetal demise. The revised analysis suggests that younger maternal age was most strongly associated with preterm birth and fetal demise, while grand multiparity was more closely linked to low birth weight and intrauterine fetal death. These findings support the need for earlier identification of high-risk women, closer antenatal surveillance, and targeted obstetric management in patients with PIH. At the same time, the presence of at least one clinically inconsistent stratified result highlights the importance of careful raw-data verification before publication, so that conclusions remain both statistically sound and clinically credible .

REFERENCES

1. Pinheiro RL, Areia AL, Mota Pinto A, Donato H. Advanced maternal age: adverse outcomes of pregnancy, a meta-analysis. Acta Med Port. 2019;32(3):219-26.

2. Sackey DS, Larbie C, Mensah FO. Geophagia, nutrition and health of women with pregnancy-induced hypertension. Afr Health Sci. 2018;18(4):1243-54.

3. Al-Rubaie ZTA, Askie LM, Hudson HM, Ray JG, Jenkins G, Lord SJ. Assessment of NICE and USPSTF guidelines for identifying women at high risk of pre-eclampsia for tailoring aspirin prophylaxis in pregnancy: an individual participant data meta-analysis. Eur J Obstet Gynecol Reprod Biol. 2018;229:159-66.

4. Rotem R, Pariente G, Golevski M, Baumfeld Y, Yohay D, Weintraub AY. Association between hypertensive disorders of pregnancy and third stage of labor placental complications. Pregnancy Hypertens. 2018;13:166-70.

5. August P, Rose C, Garovic V, Moser M. Hypertension in pregnancy. Med Roundtable Cardiovasc Ed. 2013;4:e18-e24.

6. Ilekis JV, Reddy UM, Roberts JM. Preeclampsia: a pressing problem: an executive summary of a National Institute of Child Health and Human Development workshop. Reprod Sci. 2007;14:508-13.

7. Yücesoy G, Ozkan S, Bodur H, Tan T, Calişkan E, Vural B, et al. Maternal and perinatal outcome in pregnancies complicated with hypertensive disorder of pregnancy: a seven year experience of a tertiary care center. Arch Gynecol Obstet. 2005;273:43-9.

8. Witlin AG, Sibai BM. Hypertension in pregnancy: current concepts of preeclampsia. Annu Rev Med. 1997;48:115-27.

9. Duckitt K, Harrington D. Risk factors for pre-eclampsia at antenatal booking: systematic review of controlled studies. BMJ. 2005;330:565-9.

10. Brouwers L, van der Meiden-van Roest AJ, Savelkoul C, Vogelvang TE, Lely AT, Franx A, et al. Recurrence of pre-eclampsia and the risk of future hypertension and cardiovascular disease: a systematic review and meta-analysis. BJOG. 2018;125(13):1642-54.

11. Nawaz F, Sultan S, Siddiqi I. Pregnancy outcome in primigravida complicated with pregnancy induced hypertension. J Med Sci. 2014;22(1):46-8.

12. Zahoor S, Malik T, Zulfiqar S. Eclamptic patients: maternal and perinatal outcomes. Prof Med J. 2015;22(11):1464-70.

13. Das S, Sahu M, Mohapatra S, Padmavati V, Panigrahi PK. Pregnancy induced hypertension and feto-maternal outcome in a tertiary care hospital in eastern India: a prospective study. J Clin Diagn Res. 2018;12(11):QC17-QC21.

14. Cunningham FG. Hypertensive disorders in pregnancy. In: Williams Obstetrics. 22nd ed. New York: McGraw-Hill; 2005. p. 761.

15. Jye CJ. Challenges of obstetrician in the management of severe preeclampsia. Obstet Gynaecol Today. 2009;16(8):348-51.

16. Dadelszen P, Magee L. What matters in preeclampsia are the associated adverse outcomes: the view from Canada. Curr Opin Obstet Gynecol. 2008;20:110-5.

17. National High Blood Pressure Education Program Working Group. Report of the National High Blood Pressure Education Program working group on high blood pressure in pregnancy. Am J Obstet Gynecol. 2000;183:1-22.

18. Brown MA, Hague WM, Higgins J. The detection, investigation and management of hypertension in pregnancy: full consensus statement. Aust N Z J Obstet Gynaecol. 2000;40:139-55.

19. Parmar MT, Solanki HM, Gosalia VV. Study of risk factors of perinatal death in pregnancy induced hypertension. Natl J Community Med. 2012;3:703-7.

20. Gandhi MR, Jani PS, Patel UM, Kakani CR, Thakor NC, Gupta N. Perinatal outcome in pregnancy induced hypertension cases at GMERS Medical College, Dharpur-Patan, North Gujarat region, India: a prospective study. Int J Adv Med. 2015;2(2):152-5.

21. Bangal VB, Giri PA, Mahajan AS. Maternal and foetal outcome in pregnancy induced hypertension: a study from rural tertiary care teaching hospital in India. Int J Biomed Res. 2011;2(12):595-9.

22. Khosravi S, Dabiran S, Lotfi M, Asnavandy M. Study of the prevalence of hypertension and complications of hypertensive disorders in pregnancy. Open J Prev Med. 2014;4:860-7.

23. Seyom E, Abera M, Tesfaye M, Fentahun N. Maternal and fetal outcome of pregnancy related hypertension in Mettu Karl Referral Hospital, Ethiopia. J Ovarian Res. 2015;8:1-7.

24. Jiji DB, Cabading ML, Benjamin BA. A comparative study to find the maternal and foetal outcome among pregnancy induced hypertension and non-pregnancy induced hypertension gravid women. Sch J Appl Med Sci. 2014;2(4):1286-9.

25. Ahmed M, Daver RG. Study of feto-maternal outcome in pregnancy induced hypertension. Glob J Med Res E Gynecol Obstet. 2014;14(1):21-5.

26. Patel R, Baria H, Patel HR, Nayak S. A study on pregnancy induced hypertension and foetal outcome among patient with PIH at tertiary care hospital, Valsad. Int J Community Med Public Health. 2017;4:4277-81.

27. Dağdeviren H, Çankaya A, Cengiz H, Tombul T, Kanawati A, Çaypınar SS, et al. Maternal and neonatal outcomes of women with preeclampsia and eclampsia at a tertiary care center. Med Bull Haseki. 2015;53:143-6.

28. Singh A, Chawla S, Pandey D, Jahan N, Anwar A. Fetomaternal outcome in cases of pre-eclampsia in a tertiary care referral hospital in Delhi, India: a retrospective analysis. Int J Sci Stud. 2016;4(2):100-3.

29. Shaikh S, Shaikh NB, Channa S, Ghori A. Outcome of pregnancy in women with severe pre-eclampsia. Med Channel. 2012;19:41-45.