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Association of Gestational Hypertension with Neonatal Cardiovascular Physiology

Sanodia Afridi[1], Imran Khan[2*], Asfa Mumtaz[3], Shabnam Bibi[4], Haji Gul[5] and Khalil Ahmad[6]

[1]Department of Obstetrics and Gynaecology, Health Net Hospital, Hayatabad, Pakistan
[2]Department of Paediatrics, Gomal Medical College, Dera Ismail Khan, Pakistan
[3]Department of Community Medicine, Khyber Medical College, Peshawar, Pakistan
[4]Department of Obstetrics and Gynaecology, Women and Children Hospital, Kohat, Pakistan
[5]Department of Paediatrics, Gajju Khan Medical College, Swabi, Pakistan
[6]Department of Paediatrics, Bacha Khan Medical College, Mardan, Pakistan

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***Corresponding Author:**
Imran Khan
Department of Paediatrics, Gomal Medical College, Dera Ismail Khan, Pakistan
drimranbettani2017@gmail.com

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ABSTRACT

Gestational hypertension (GH) is a common hypertensive disorder of pregnancy associated with increased maternal and neonatal risks. While its impact on maternal cardiovascular health is well-established, its effects on neonatal cardiovascular physiology remain insufficiently explored. **Objective:** To examine the association between GH severity and neonatal cardiovascular outcomes. **Methods:** A cross-sectional observational study was conducted at Health Net Hospital, Peshawar, including 150 mother-neonate pairs diagnosed with GH. Participants were categorized into mild, moderate, and severe GH groups per ACOG criteria. Neonatal cardiovascular parameters heart rate, blood pressure, pulmonary artery pressure, LVEF, and CHD, were assessed. One-way ANOVA and Chi-square tests analyzed group differences, while logistic regression identified independent predictors of NICU admission. **Results:** Of the 150 neonates, 34% required NICU admission, with all severe GH cases admitted ($p<0.001$, Cramer's $V=0.638$). One-way ANOVA showed no significant differences in heart rate, blood pressure, or LVEF across GH groups ($p>0.05$), though LVEF showed a borderline trend ($p=0.059$). Logistic regression confirmed GH severity as an independent predictor of NICU admission (OR: 0.181, 95% CI: 0.097–0.339, $p<0.001$), while birth weight was non-significant ($p=0.575$). **Conclusions:** It was concluded that the severity of gestational hypertension is significantly associated with adverse neonatal cardiovascular outcomes, particularly NICU admission. GH severity independently predicted NICU requirement, emphasizing the need for close monitoring and early intervention in pregnancies complicated by moderate to severe GH.

INTRODUCTION

Gestational hypertension (GH), defined as new-onset hypertension after 20 weeks of gestation without proteinuria, affects approximately 1% to 6% of pregnancies in Western countries [1]. GH poses notable risks to both maternal and fetal health, including preterm delivery, fetal growth restriction, and placental abruption [2, 3]. Although the long-term cardiovascular risks for women with GH chronic hypertension and cardiovascular disease (CVD) are well documented, the immediate cardiovascular effects on neonates remain underexplored. Most existing literature has focused on preeclampsia, leaving the impact of GH itself, particularly across its severity spectrum, insufficiently studied [4, 5]. Given the rising incidence of hypertensive disorders in pregnancy, understanding how GH severity affects neonatal cardiovascular parameters is essential. While a few studies have touched on neonatal outcomes in hypertensive pregnancies [6], data specifically examining GH severity and its correlation with neonatal cardiovascular function, including parameters like heart rate, blood pressure (BP), left ventricular ejection

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fraction (LVEF), and Neonatal Intensive Care Unit (NICU) admission, are scarce. This study addresses the identified gap by evaluating the association between GH severity (mild, moderate, severe) and detailed neonatal cardiovascular outcomes.

This study aims to determine whether increasing GH severity independently predicts adverse neonatal cardiac parameters and NICU admission, thereby contributing evidence to guide perinatal care and risk stratification.

METHODS

This cross-sectional observational study was conducted at Health Net Hospital, Peshawar, from October 2023 to October 2024, after obtaining ethical approval from the Ethics Review Committee (Approval Ref No: 3060/HNH/HR). The study aimed to assess the association between gestational hypertension (GH) and neonatal cardiovascular physiology. A total of 150 mother-neonate pairs were enrolled using non-probability consecutive sampling. The sample size was determined using Open Epi version 3.01. A total of 150 mother-neonate pairs was calculated based on a 95% confidence interval, 5% margin of error, and an expected NICU admission prevalence of 40% in gestational hypertension cases. The calculation used the standard formula for single population proportion: $n = [Z^2 \times P(1-P)] / d^2$, where Z=1.96, p=0.40, and d=0.05. This prevalence was derived from a prior study by Khan *et al.*, (2022) [7], which highlighted neonatal complications associated with GH. This ensured sufficient statistical power and external validity for our analysis. This sampling method was chosen for its feasibility, although it may limit generalizability. Inclusion Criteria: pregnant women aged 18–45 years, Singleton pregnancies diagnosed with GH after 20 weeks of gestation, classified as mild, moderate, or severe per ACOG guidelines, Deliveries at ≥32 weeks of gestation, and Live-born neonates with complete cardiovascular assessment data. Exclusion criteria: chronic hypertension, preeclampsia, or eclampsia, Multiple gestations, Neonates with congenital anomalies or requiring immediate surgery, and Incomplete maternal or neonatal records. Data were collected using a structured proforma from patient records and direct assessments. Maternal variables included age, gravida, parity, BMI, mode of delivery, onset and severity of GH, antihypertensive use, proteinuria, and antenatal booking. Neonatal data included gender, birth weight, Apgar scores (1 min, 5 min), gestational age (term/preterm), NICU admission, and resuscitation status. Cardiovascular variables assessed were heart rate, systolic/diastolic BP, oxygen saturation, pulmonary artery pressure, LVEF, patent ductus arteriosus (PDA), interventricular septal thickness, right ventricular (RV) function, congenital heart disease (CHD), cardiomegaly, and inotrope requirement. Maternal BP was measured using a calibrated sphygmomanometer. GH was defined as BP ≥140/90 mmHg on two readings ≥4 hours apart. Neonatal cardiovascular evaluations were conducted by a Pediatric Cardiologist using Doppler echocardiography following standardized protocols. Resuscitation needs were assessed according to NRP guidelines. To ensure inter-observer reliability, a single cardiologist performed all echocardiographic evaluations. Maternal BP was measured thrice and averaged. Content validity of the proforma was ensured through expert review. Criterion validity was established by cross-checking echocardiographic findings with hospital records. Statistical validity was supported by Shapiro-Wilk and Levene's tests, and the Hosmer-Lemeshow test for logistic regression. Analysis was performed using SPSS version 23.0. A p-value<0.05 was considered statistically significant. Continuous variables were reported as mean ± SD; categorical variables as frequencies and percentages. Shapiro-Wilk test, Q-Q plots, and skewness/kurtosis were used to test normality. One-way ANOVA compared cardiovascular outcomes (HR, BP, LVEF) across GH severity groups. Homogeneity of variance was assessed via Levene's test. Tukey's or Games-Howell post-hoc tests were used as appropriate. Chi-square tests analyzed categorical outcomes (NICU admission, CHD). Bonferroni correction and Cramer's V were applied for post-hoc significance and strength of association. Binary logistic regression was used to assess GH severity as a predictor of NICU admission. Variables included GH severity, gestational age, and birth weight. Adjusted odds ratios (OR) with 95% CI were reported. Model fit was assessed using the Omnibus test, Hosmer-Lemeshow test, classification accuracy, and Nagelkerke R^2.

RESULTS

Most mothers were around 30 years old, and the majority delivered at term. Vaginal delivery was most common. Moderate and mild GH were more frequent than severe GH. Antihypertensive use and proteinuria were reported in nearly half and two-thirds of cases, respectively. Most mothers had booked antenatal care. Among neonates, mean birth weight and Apgar scores were within normal range. However, 34% required NICU admission and 42% were born preterm, indicating adverse neonatal effects despite generally stable maternal parameters (Table 1).

Table 1: Maternal and Neonatal Demographic and Clinical Characteristics (n=150)

Variables		Values
Maternal Characteristics		
Maternal Age (Years)	Range: 16.9–42.3	29.59 ± 4.71
Gravida	G1	45 (30.0%)
	G2	61 (40.7%)
	G3	31 (20.7%)
	G4	13 (8.7%)
Parity	P0	45 (30.0%)
	P1	61 (40.7%)

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	P2	31 (20.7%)
	P3	13 (8.7%)
Gestational Age at Delivery (Weeks)	Range: 32.8–41.4	37.18 ± 1.78
Mode of Delivery	Normal Vaginal	90 (60.0%)
	C-Section	51 (34.0%)
	Assisted	9 (6.0%)
Body Mass Index	Range: 17.2–38.3	28.02 ± 4.18
Pre-existing Conditions	Yes	53 (35.3%)
	No	97 (64.7%)
GH Onset	Early	43 (28.7%)
	Late	107 (71.3%)
GH Severity	Mild	61 (40.7%)
	Moderate	63 (42.0%)
	Severe	26 (17.3%)
Antihypertensive Use	Yes	72 (48.0%)
	No	78 (52.0%)
Proteinuria	Present	94 (62.7%)
	Absent	56 (37.3%)
Antenatal Care	Booked	116 (77.3%)
	Un-booked	34 (22.7%)
Neonatal Characteristics		
Gender	Male	70 (46.7%)
	Female	80 (53.3%)
Birth Weight (Grams)	Range: 1651–3865	2802.39 ± 410.87
Apgar Score	1 min / 5 min	7.01 ± 1.34 / 7.50 ± 1.40
NICU Admission	Yes	51 (34.0%)
	No	99 (66.0%)
Resuscitation Needed	Yes	57 (38.0%)
	No	93 (62.0%)
Gestational Age at Birth	Preterm (<37 Weeks)	63 (42.0%)
	Term (≥37 Weeks)	87 (58.0%)

Neonatal vitals (heart rate, BP, oxygen saturation) were largely within normal limits. LVEF and RV function indicated good cardiac output in most cases. However, PDA was observed in 48%, and 15–18% of neonates had CHD, cardiomegaly, or required inotropes, suggesting subtle cardiovascular stress despite stable averages (Table 2).

Table 2: Neonatal Cardiovascular Physiology Parameters

Parameter	Unit	(Mean ± SD) / Frequency (%)
Heart Rate	bpm	140.76 ± 10.70
Systolic Blood Pressure	mmHg	65.68 ± 4.61
Diastolic Blood Pressure	mmHg	39.81 ± 5.20
Oxygen Saturation	%	96.27 ± 2.26
PDA	Present	72 (48.0%)
	Absent	78 (52.0%)
Pulmonary Artery Pressure	mmHg	30.32 ± 7.23
LVEF	%	59.96 ± 4.51
RV Function	Normal	133 (88.7%)
	Impaired	17 (11.3%)
Interventricular Septal Thickness	mm	3.96 ± 0.52
CHD	Yes	Yes: 15 (10.0%)
	No	135 (90.0%)
Cardiomegaly	Yes	27 (18.0%)
	No	123 (82.0%)
Inotropes Needed	Yes	22 (14.7%)
	No	128 (85.3%)

One-way ANOVA showed no significant differences in continuous variables across GH groups. LVEF had a borderline p-value (0.059) but was not significant after correction. Chi-square tests revealed a strong association between GH severity and NICU admission (p=0.000, Cramer's V=0.638). Other outcomes like PDA, CHD, and inotrope use showed no significant variation (Table 3).

Table 3: Association of GH Severity with Neonatal Cardiovascular Outcomes

Neonatal Variables	Mild GH	Moderate GH	Severe GH	p-value	Statistical Test	Interpretation
Heart Rate (bpm)	140.16 ± 11.68	140.70 ± 10.16	142.31 ± 9.78	0.695	One-Way ANOVA	No Significant Difference
Systolic BP (mmHg)	65.95 ± 4.65	65.84 ± 4.42	64.65 ± 5.03	0.458	One-Way ANOVA	No Significant Difference
Diastolic BP (mmHg)	39.72 ± 5.14	39.83 ± 4.95	39.96 ± 6.10	0.980	One-Way ANOVA	No Significant Difference
Pulmonary Artery Pressure (mmHg)	30.51 ± 6.86	29.53 ± 7.38	31.78 ± 7.71	0.398	One-Way ANOVA	No significant difference
LVEF (%)	58.91 ± 4.30	60.78 ± 4.89	60.44 ± 3.64	0.059	One-Way ANOVA	Trend (NS After Bonferroni Correction, p≈0.198)
NICU Admission (Yes)	13 (21.3%)	12 (19.0%)	26 (100.0%)	0.000	Chi-square	Significant (Cramer's V=0.638)
PDA (Present)	28 (45.9%)	29 (46.0%)	15 (57.7%)	0.553	Chi-square	No significant difference
CHD (Yes)	9 (14.8%)	4 (6.3%)	2 (7.7%)	0.270	Chi-square	No significant difference
Cardiomegaly (Yes)	9 (14.8%)	15 (23.8%)	3 (11.5%)	0.271	Chi-square	No significant difference
Inotropes Needed (Yes)	10 (16.4%)	11 (17.5%)	1 (3.8%)	0.226	Chi-square	No significant difference

Statistical tests: One-Way ANOVA for continuous variables, Chi-square test for categorical variables.

Findings display the results of a logistic regression model analysing predictors of NICU admission. GH severity emerged as a strong and statistically significant predictor (OR: 0.181, 95% CI: 0.097–0.339, p<0.001). The odds ratio below 1 indicates that higher GH severity was associated with markedly increased odds of NICU admission. The narrow 95% confidence interval confirms the precision and robustness of this effect, directly addressing the journal comment regarding the importance of interpreting CIs for key outcomes. Gestational age at birth showed a borderline association (p=0.070), suggesting that preterm delivery may also contribute to NICU admissions, although not significantly in this model. Birth weight was not a

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significant predictor (p=0.575), and its odds ratio was nearly 1 (1.000), indicating a negligible impact in the presence of other variables. The model's goodness-of-fit was acceptable, with a Nagelkerke R^2 value of 0.348, indicating that roughly 35% of the variability in NICU admission could be explained by the included predictors. These results emphasize GH severity as a clinically relevant and statistically validated independent predictor of adverse neonatal outcomes, particularly NICU requirement (Table 4).

Table 4: Binary Logistic Regression Predicting NICU Admission (n=150)

Predictors	B	SE	Wald	p-value	OR (Exp (B)	95% CI for OR
GH Severity	-1.710	0.321	28.43	<0.001	0.181	0.097-0.339
Gestational Age (Preterm)	0.746	0.411	3.29	0.070	2.108	0.942–4.720
Birth Weight (g)	0.000	0.001	0.315	0.575	1.000	0.999–1.001
Constant	1.954	1.607	1.479	0.224	7.059	–

The graph shows a direct relationship between gestational hypertension (GH) severity and NICU admission. In mild GH, 21.3% of neonates required NICU care, while 19% were admitted in moderate GH cases. However, all neonates (100%) born to mothers with severe GH required NICU admission. This trend highlights the impact of worsening maternal hypertension on neonatal outcomes, emphasizing the need for early intervention (Figure 1).

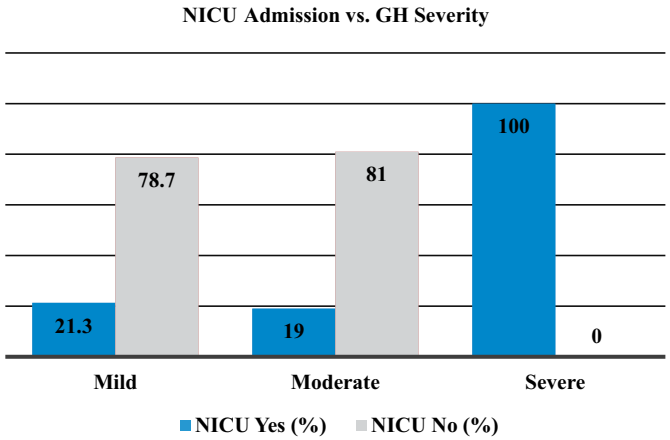

Figure 1: NICU Admission vs GH Severity

DISCUSSION

The findings of this study underscore a significant association between the severity of gestational hypertension (GH) and adverse neonatal cardiovascular outcomes. Notably, a marked increase in NICU admissions was observed among neonates born to mothers with severe GH, with all such cases requiring intensive care. This aligns with existing literature indicating that hypertensive disorders during pregnancy significantly elevate neonatal complications, including preterm birth and NICU admissions [4, 8]. Similar findings have been reported by Bromfield *et al.*, who demonstrated increased neonatal intensive care needs associated with severe hypertensive disorders in pregnancy [9]. Additionally, Li *et al.*, found comparable results, reinforcing the strong predictive relationship between hypertensive severity and neonatal outcomes [10]. The logistic regression analysis in this study identified GH severity as an independent predictor of NICU admission, even after adjusting for gestational age and birth weight. Similar findings were documented by Lin *et al.*, and Rocha de Moura *et al.*, who reported severe GH as a critical determinant of adverse neonatal outcomes [11, 12]. Bond *et al.*, emphasized the necessity for stringent prenatal monitoring in severe GH cases to minimize neonatal morbidity [13]. Furthermore, the precision of our findings is reflected by the narrow confidence interval for GH severity (OR: 0.181, 95% CI: 0.097–0.339), reinforcing the robustness and reliability of this association. Interestingly, while severe GH was significantly associated with higher NICU admissions, other neonatal cardiovascular parameters, including heart rate, blood pressure, pulmonary artery pressure, and left ventricular ejection fraction (LVEF), did not differ significantly across GH severity categories. This finding aligns with recent studies by Miranda *et al.*, and Täufer *et al.*, who observed that maternal hypertensive disorders did not consistently impact specific cardiovascular parameters despite influencing overall neonatal morbidity [14, 15]. A potential explanation for this could be that neonatal compensatory mechanisms, such as improved cardiac resilience or rapid postpartum adaptation, might mitigate detectable differences across severity groups [16]. Although birth weight is widely recognized as a predictor of neonatal outcomes, it was non-significant in our logistic regression model (p=0.575). One possible explanation is multi-collinearity, as birth weight and gestational age may overlap in predicting neonatal outcomes. This is supported by findings from Phoswa *et al.*, and Baschat *et al.*, who identified gestational age as a more robust predictor of neonatal morbidity than birth weight alone, particularly in hypertensive pregnancies [17, 18]. It is also plausible that, in severe GH cases, preterm delivery rather than birth weight per se directly contributes to neonatal risk, a theory echoed by Kulkarni *et al.*, [19]. Future models should consider multi-collinearity assessments or interaction analyses to more accurately elucidate the independent effects of these variables. Our study also indicated a borderline significance in gestational age (p=0.070), suggesting a possible confounding role. Similar borderline effects have been previously reported by Tcheugui *et al.*, and Zbelo *et al.*, who stressed the importance of careful

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interpretation of gestational age effects, especially in the context of maternal hypertension [20, 21]. In summary, this study emphasizes the critical need for early detection and effective management of GH to optimize neonatal outcomes. The demonstrated significant association between GH severity and NICU admission supports targeted prenatal care strategies. Further research into underlying biological mechanisms and intervention strategies could substantially benefit clinical practice, enhancing neonatal health outcomes in pregnancies complicated by hypertension.

CONCLUSIONS

It was concluded that this study highlights a significant association between GH severity and neonatal cardiovascular outcomes, notably emphasizing GH severity as a critical predictor of NICU admission. Although other neonatal cardiovascular parameters were not significantly affected, the strong predictive value of GH severity underscores the need for early detection, close prenatal monitoring, and targeted clinical interventions. Implementing tailored management strategies for hypertensive pregnancies is crucial for enhancing neonatal health outcomes and reducing associated complications.

Authors Contribution

Conceptualization: SA

Methodology: AM, SB, HG, KA

Formal analysis: SA, IK, AM

Writing review and editing: SA, IK, AM, KA

All authors have read and agreed to the published version of the manuscript

Conflicts of Interest

All the authors declare no conflict of interest.

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