Original Article
DOI: https://doi.org/10.70749/ijbr.v3i4.2081

INDUS JOURNAL OF BIOSCIENCE RESEARCH
https://ijbr.com.pk
ISSN: 2960-2793/ 2960-2807

Frequency of Pregnancy Induced Hypertension in Primigravida

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ARTICLE INFO

Keywords: Family history, Blood pressure, Pregnancy-induced hypertension, Primigravida, Antenatal screening.

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Declaration
Authors' Contribution: All authors equally contributed to the study and approved the final manuscript.

Conflict of Interest: No conflict of interest.

Funding: No funding received by the authors.

Article History
Received: 21-03-2025 Revised: 02-05-2025
Accepted: 14-05-2025 Published: 31-05-2025

ABSTRACT

Background: Pregnancy-induced hypertension is a common complication of initial pregnancies, being a leading cause of maternal and foetal morbidity. Primigravidas, due to a lack of prior obstetric history and immunologic accommodation, are vulnerable to PIH. Early diagnosis and locality-specific evidence are of prime importance for outcome enhancement, particularly for scenarios of late antenatal presentation. **Objective:** To determine the frequency of pregnancy induced hypertension in primigravida patients presenting to Ayub Teaching Hospital Abbottabad. **Study Design:** Descriptive cross-sectional study. **Duration and Place of Study:** The study was conducted over a six-month period following ethical approval, at the Department of Obstetrics and Gynaecology, Ayub Teaching Hospital, Abbottabad from October 2024 to February 2025. **Methodology:** A total of 119 primigravida women aged 18–35 years with gestational age >20 weeks were enrolled through non-probability consecutive sampling. Patients with pre-existing hypertension or chronic illnesses were excluded. Blood pressure was measured using standard auscultatory techniques. A diagnosis of PIH was made when systolic BP was ≥140 mmHg on two separate readings four hours apart without proteinuria. **Results:** The mean age of participants was 25.59±5.05 years. The frequency of PIH in primigravida patients was 18.5%. A significant association was found between PIH and positive family history of hypertension (p=0.001). A borderline association was noted with maternal age >30 years (p=0.050). **Conclusion:** Pregnancy-induced hypertension is not rare among primigravidas. A positive family history of hypertension is a potent predictor, which underlines the value of selective screening and early management techniques among high-risk patients.

INTRODUCTION

Primigravida refers to a woman in her first pregnancy. These expectant individuals will normally require stringent obstetric follow-up because their bodies will be experiencing physiological changes of pregnancy for the first time.[1] In comparison with multigravidas, primigravidas will have prolonged labors as well as increased risk of interventions due to lack of past obstetric history.[2] Due to lack of past history, risk assessment as well as care must be founded purely on this current pregnancy, hence vigorous prenatal care is required.[2]

Pregnancy-induced hypertension (PIH), or gestational hypertension, is one condition defined as new hypertension (≥140/90 mmHg) arising at or after 20 weeks of gestation in a woman who was normotensive.[3] PIH can occur with or without signs of organ dysfunction and can transform into severe forms of hypertensive disorders of pregnancy like preeclampsia or eclampsia unless it is identified early and treated in a timely manner.[4] PIH is associated with significant maternal as well as fetal morbidity in the form of placental abruption, fetal growth restriction, as well as prematurity, thus requiring early vigilance with timely intervention.[5]

Several studies have corroborated that primigravidas are at a higher risk of PIH as opposed to multigravidas.[6] It is a multifactorial pathway of PIH consisting of immunological maladaptation, abnormal placentation as well as endothelial dysregulation, being exaggerated in initial pregnancies due to lack of preceding maternal immunological experience with paternal antigens.[7] It is a testament of need for particular antenatal screening as well as counseling of primigravidas in reducing occurrence as well as unfavorable perinatal outcome of PIH.[8]

Pregnancy-induced hypertension in primigravidas is managed through early diagnosis, control of blood pressure, as well as continuous fetal and maternal surveillance.[9] Treatment with an antihypertensive agent, such as labetalol, methyldopa, or nifedipine, is considered when blood pressure is above safe limits, usually ≥160/110 mmHg.[10] Ongoing antenatal care is necessary in evaluating maternal symptomatology as well as in detecting signs of disease advancement, such as proteinuria, increased liver enzymes, as well as fetal intrauterine growth retardation.[11] Fetal status is

appraised using repeat ultrasounds, non-stress test, as well as Doppler studies. Delivery planning is in relation to gestation age, severity of hypertension, as well as maternal-fetal status, with expectant management being potential in stable individuals before 37 weeks.[12] In those situations, wherein maternal or fetal condition declines, early delivery is considered with a view of averting disease complications.[13]

A study by Mathew R et al. reported a pregnancy-induced hypertension frequency of 18.6% among primigravida patients.[14]

There was an urgent need to study pregnancy-induced hypertension in primigravidas in the context of Abbottabad due to this region's particular population as well as health facility accessibility problems. There was a high percentage of late presentations of woman in this region with consequent delay in diagnosis as well as treatment of disorders of hypertension. Moreover, lack of region-specific data further added towards difficulties in preparing region-specific clinical guidelines. There was a requirement of this research study with a goal of implementing immediate locale-specific evidence in order to facilitate early diagnosis as well as effective PIH care in order to improve maternal as well as neonatal outcome in this vulnerable population.

METHODOLOGY

This descriptive cross-sectional study was carried out over a six-month period following approval from the institutional review board at the Department of Obstetrics and Gynaecology, Ayub Teaching Hospital, Abbottabad from October 2024 to February 2025. A total of 119 women were enrolled, all experiencing their first pregnancy. The sample size was calculated using WHO software, with a confidence level of 95%, a 7% margin of error, and an anticipated prevalence of gestational hypertension of 18.6% in this population.

Participants were recruited through non-probability consecutive sampling. Women aged 18 to 35 years, carrying a single fetus on ultrasound, and having a gestational age of more than 20 weeks based on the last menstrual period were considered eligible. Women were classified as primigravida if they had no prior history of pregnancy beyond the age of viability. Those with known hypertension prior to conception, chronic conditions such as renal disease or diabetes, or any pre-existing cardiovascular disorders were excluded. After informed written consent was obtained, demographic and clinical information including maternal age, gestational age, educational background, occupational status, area of residence, socioeconomic status, and family history of hypertension was collected using a structured data collection tool.

Blood pressure was measured using a standardized protocol to ensure consistency. All participants rested for at least five minutes in a seated position with the arm supported at heart level. A properly sized cuff was applied snugly to the upper arm, and blood pressure was assessed using the auscultatory method. The systolic value was noted at the first Korotkoff sound and the diastolic value at its disappearance. A diagnosis of gestational hypertension was made in women who recorded a systolic pressure of 140 mmHg or higher on two separate occasions spaced four hours apart after 20 weeks of gestation, in the absence of proteinuria confirmed by laboratory testing.

All data were analyzed using IBM SPSS version 26. Continuous variables such as age and gestational age were summarized as means with standard deviations. Categorical variables, including gestational hypertension, education level, employment status, residence type, socioeconomic class, and family history of hypertension, were presented as frequencies and percentages. Stratification was done for potential effect modifiers, and associations were assessed using the chi-square test. A p-value of ≤0.05 was considered statistically significant.

RESULTS

The study included 119 primigravida patients with a mean age of 25.59±5.05 years, gestational age of 30.27±6.34 weeks, and systolic blood pressure of 123.64±15.11 mmHg (as shown in Table-I). Among the participants, 24 (20.2%) had a family history of hypertension while 95 (79.8%) did not. The residential distribution was nearly equal with 60 (50.4%) from rural areas and 59 (49.6%) from urban areas. Regarding socioeconomic status, 37 (31.1%) were classified as poor, 67 (56.3%) as middle class, and 15 (12.6%) as rich. Educational attainment showed that 19 (16.0%) were uneducated, 51 (42.9%) had primary education, 37 (31.1%) had secondary education, and 12 (10.1%) had tertiary education (as shown in Table 1).

Table 1

Patient Demographics

Demographics		Mean ± SD
Age (years)		25.59±5.05
Gestational Age (weeks)		30.27±6.34
Systolic Blood Pressure (mmHg)		123.64±15.11
Family History of Hypertension	Yes	24 (20.2%)
	No	95 (79.8%)
Residential Status	Rural	60 (50.4%)
	Urban	59 (49.6%)
Socioeconomic Status	Poor	37 (31.1%)
	Middle	67 (56.3%)
	Rich	15 (12.6%)
Education Level	Uneducated	19 (16.0%)
	Primary	51 (42.9%)
	Secondary	37 (31.1%)
	Tertiary	12 (10.1%)

The overall frequency of pregnancy induced hypertension in primigravida was 22 (18.5%), while 97 (81.5%) remained normotensive throughout pregnancy (as shown in Table 2).

Table 2

Frequency of Pregnancy Induced Hypertension in Primigravida

Pregnancy Induced Hypertension	n (%)
Yes	22 (18.5%)
No	97 (81.5%)
Total	119 (100.0%)

Stratified analysis revealed that age >30 years showed a borderline significant association with pregnancy induced hypertension, with 8 (32.0%) of older mothers developing the condition compared to 14 (14.9%) of younger mothers

(p=0.050). Gestational age showed no significant association, with 12 (19.0%) of those ≤30 weeks and 10 (17.9%) of those >30 weeks developing pregnancy induced hypertension (p=0.867). Family history of hypertension demonstrated a highly significant association, with 10 (41.7%) of those with positive family history developing the condition versus only 12 (12.6%) of those without family history (p=0.001). Residential status showed no significant difference, with 13 (21.7%) of rural and 9 (15.3%) of urban residents developing pregnancy induced hypertension (p=0.368). Socioeconomic status revealed no significant association, with 6 (16.2%) of poor, 13 (19.4%) of middle class, and 3 (20.0%) of rich participants developing the condition (p=0.946). Educational level also showed no significant association, with 2 (10.5%) of uneducated, 10 (19.6%) of primary educated, 7 (18.9%) of secondary educated, and 3 (25.0%) of tertiary educated participants developing pregnancy induced hypertension (p=0.771) (as shown in Table 3).

Table 3

Association of Pregnancy Induced Hypertension with Demographic Factors

Demographic Factors		Pregnancy Induced Hypertension		p-value
		Yes n(%)	No n(%)	
Age (years)	≤30	14 (14.9%)	80 (85.1%)	0.050
	>30	8 (32.0%)	17 (68.0%)	
Gestational Age (weeks)	≤30	12 (19.0%)	51 (81.0%)	0.867
	>30	10 (17.9%)	46 (82.1%)	
Family History of Hypertension	Yes	10 (41.7%)	14 (58.3%)	0.001*
	No	12 (12.6%)	83 (87.4%)	
Residential Status	Rural	13 (21.7%)	47 (78.3%)	0.368
	Urban	9 (15.3%)	50 (84.7%)	
Socioeconomic Status	Poor	6 (16.2%)	31 (83.8%)	0.946*
	Middle	13 (19.4%)	54 (80.6%)	
	Rich	3 (20.0%)	12 (80.0%)	
Education Level	Uneducated	2 (10.5%)	17 (89.5%)	0.771*
	Primary	10 (19.6%)	41 (80.4%)	
	Secondary	7 (18.9%)	30 (81.1%)	
	Tertiary	3 (25.0%)	9 (75.0%)	

*Fischer Exact Test

DISCUSSION

This study investigated the prevalence of pregnancy induced hypertension among primigravida and association of several socio-demographic factors with the condition, reporting a combined prevalence of 18.5% of the study population. This finding is similar to the world health disease burden of hypertensive disorders of pregnancy, which is a leading obstetric complication that predominantly affects first-time pregnant women. Elevated risk of primigravida for pregnancy induced hypertension can be attributed to the immunological hypothesis, through which initial exposure of the expectant woman to paternal antigens during first pregnancy triggers exaggerated maternal immunological response, leading to impaired embedding of the placenta with consequent endothelium dysfunction.

By a significantly higher frequency of pregnancy induced hypertension among positive compared with negative (41.7% vs 12.6%) family histories, the strong genetic predisposition for the condition is well understood. This association is a mirror of the heritable predisposition including multifactorial genetic polymorphism of renin-angiotensin system, endothelium, and inflammatory systems involved in the maintenance of vascular tone as well as blood pressure balance during pregnancy. Borderline significant association of maternal age of >30 years (32.0% vs 14.9%) can be accounted for age-related vessels changes, for examples, endothelial compliance, arterial stiffness, and adaptive responses to hemodynamic changes of pregnancy being impaired. Aging maternal status is accompanied with increased oxidative stress, reduced availability of nitric oxide, resulting impaired perfusion of the placenta with complicating hypertension.

Our study results demonstrated a frequency of pregnancy induced hypertension of 18.5% among primigravida patients, which aligns closely with several international studies while showing notable variations from others. This prevalence is remarkably similar to Saifullah et al. [14] who reported an incidence of 20.6% in their cross-sectional analysis at Sandeman Provincial Hospital, Quetta, Pakistan. The consistency between our findings and this Pakistani study suggests similar population characteristics and diagnostic criteria may contribute to comparable prevalence rates. However, our results contrast significantly with other studies from the region, particularly Hema et al. [15] who found a much lower prevalence of 8.3% for any hypertensive disorder in pregnancy at Cure Well Hospital, Warangal, India. This difference may be attributed to variations in study populations, diagnostic criteria, or the fact that our study focused exclusively on primigravida patients, while Hema et al. [15] included all pregnant women regardless of parity.

The maternal age distribution in our study, with a mean age of 25.59±5.05 years, is consistent with findings from several comparative studies. Saifullah et al. [14] reported a mean age of 25.2±4.2 years, while Nawaz et al. [16] found a mean age of 22.4±4.6 years among primigravid hypertensive women. Our finding of a borderline significant association between age >30 years and pregnancy induced hypertension (32.0% vs 14.9%, p=0.050) supports the conclusions of Ashraf et al.[17] who demonstrated that women ≤35 years had 2.07-fold higher odds of developing PIH. However, this appears contradictory to the general understanding that advanced maternal age increases hypertensive risk. The explanation may lie in the fact that most studies, including ours, have predominantly younger populations, making it difficult to establish clear age-related trends. Rana & Rana [18] similarly found that 43.1% of their PIH cases were aged 16-25 years, reinforcing the observation that PIH commonly affects younger women, particularly primigravidae.

Family history of hypertension emerged as the strongest predictor in our study, with 41.7% of those with positive family history developing PIH compared to only 12.6% of those without (p=0.001). This finding is strongly supported by multiple studies in the literature. Saifullah et al.[14] identified positive family history of PIH as a significant predictor with an adjusted odds ratio of 3.33, while Gudeta & Regassa [19] found an even stronger association with an AOR of 5.25 (95% CI 1.39-19.86). The consistency of this finding across different populations and healthcare

settings underscores the important genetic component in the development of pregnancy induced hypertension and highlights the need for enhanced monitoring of patients with positive family history.

The gestational age at presentation in our study (mean 30.27±6.34 weeks) is comparable to Hema et al.[15] who reported that 40% of their patients presented at 25-30 weeks' gestation. Interestingly, our study found no significant association between gestational age and PIH development (19.0% at ≤30 weeks vs 17.9% at >30 weeks, p=0.867), which contrasts with findings from Saifullah et al. [14] and Gudeta & Regassa [19] both of whom identified gestational age ≥37 weeks as a significant predictor (AOR = 0.096). This discrepancy may be explained by the different gestational age cutoffs used and the timing of patient recruitment in various studies.

Primiparity, which was universal in our study by design, is consistently identified as a major risk factor across multiple studies. Ashraf et al. [17] found that primigravida had 2.50-fold higher odds of PIH, while Rana & Rana [18] reported that 43.3% of their PIH cases were primigravidae. This consistency validates our decision to focus specifically on primigravid patients, as they represent a particularly vulnerable population for pregnancy induced hypertension development.

Our findings regarding socioeconomic status, education, and residential location showed no significant associations with PIH development, which differs from some international studies. Peter & Okafor [20] found that married women had 2.7-fold higher odds of PIH awareness, and educational status influenced knowledge levels, though these studies focused on awareness rather than actual disease prevalence. The lack of association in our study may reflect the relatively homogeneous socioeconomic distribution of our study population or suggest that biological factors may be more important than socioeconomic determinants in our specific population.

The blood pressure readings in our study (mean 123.64±15.11 mmHg) are within the normal range for the overall population, which is expected given that 81.5% remained normotensive. This contrasts with studies focusing on diagnosed hypertensive patients, such as Babore et al.[21] who reported mean systolic BP of 157.3±18.9 mmHg and diastolic of 104.1±9.2 mmHg among cases, and Saifullah et al.[14] who found mean systolic BP of 144.3±5.2 mmHg among their PIH cases.

Clinical correlations of our study are applicable to antenatal care of our population. 18.5% prevalence of PIH for primigravidae, along with the strong association with positive family history, suggests that certain screening and enhanced monitoring protocols must be implemented for high-risk patients. Borderline significance of maternal age of more than 30 years needs studies of a bigger scale but suggests that age risk stratification might be valuable.

This study is also subject to some limitations that need to be considered. It was a center-specific study with a relatively modest subject group of 119 primigravid patients, so the generalizability of findings to more broad-based populations could be limited. This was a cross-sectional study that constrains our ability to assess causality, and without follow-up data, we are unable to assess long-term outcomes for either neonates or for mothers. Lastly, the study was only conducted from a single healthcare center, which may not be representative of the diversity of healthcare settings and patient populations of the region.

CONCLUSION

It was concluded from our analysis that pregnancy induced hypertension is a rather common condition among primigravid women. These findings reaffirmed the importance of genetic predisposition for the development of PIH alongside adopting selective screening procedures for high-risk primigravid women, foremost of which are positive family histories for hypertension.

Acknowledgments

Gratitude is extended to the dedicated staff of the department, whose consistent efforts in accurate documentation and systematic handling of patient information greatly contributed to the success of this work.

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