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Pulse Pressure and Lipid Parameters: A Cross-Sectional Analysis in Hypertensive and Normotensive Adults.

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ABSTRACT

Background: Hypertension is a major contributor to cardiovascular morbidity and mortality, with arterial stiffness playing a pivotal role. Pulse pressure (PP), a measure of large arterial stiffness, is increasingly recognized as an independent predictor of cardiovascular events. This study explores the association between PP and lipid parameters among normotensives and hypertensive individuals with varying PP levels. **Methods:** A total of 112 adults (58% male, 42% female; aged 35-60 years) were stratified into three groups: normotensives (Group A, n=36), hypertensives with normal PP (Group B, n=43), and hypertensives with wide PP (Group C, n=33). Lipid parameters including total cholesterol, low-density lipoprotein (LDL), high-density lipoprotein (HDL), triglycerides (TG), and total cholesterol/high-density lipoprotein ratio (TC/HDL) were compared across groups using unpaired t-tests via SPSS v25. **Results:** Mean values of lipid parameters were highest in hypertensives with wide PP and lowest in normotensives. All lipid parameters, except HDL, were significantly higher in hypertensives compared to normotensives. HDL was significantly reduced in hypertensives overall, but showed no difference between normal and wide PP subgroups. **Conclusions:** Elevated PP demonstrates a positive correlation with dyslipidemia in hypertensive individuals, reinforcing its utility as a pragmatic marker in cardiovascular risk stratification.

KEYWORDS

Non-communicable diseases; Pulse pressure; Lipid parameters; Hypertension; Arterial Stiffness.

BACKGROUND

Cardiovascular diseases remain the leading global cause of death, accounting for 31% of global mortality and 28.1% of deaths in India as of 2016. CVD related deaths have nearly doubled in comparison to the 1900 estimates [1,2]. The Great Indian Blood pressure survey reports that hypertension affects nearly one-third of Indian adults, which accounts for 17.6% of the global hypertensive population [3]. Despite public

health efforts, the intersection of vascular and metabolic markers remains underutilized in risk stratification protocols.

Pulse pressure (PP), the numerical difference between systolic blood pressure (SBP) and diastolic blood pressure (DBP), and a proven marker of large artery stiffness, has emerged as an independent predictor of myocardial infarction, stroke, and cardiovascular mortality [2-5]. Compared to arterial stiffness index measured by finger photoplethysmography, PP estimation offers greater clinical utility in routine settings, enhancing cardiovascular risk assessment and mortality prediction [5,6].

Concurrently, dyslipidemia, marked by elevated low-density lipoprotein (LDL), triglycerides (TG), and total cholesterol/high-density lipoprotein (TC/HDL) ratio, is an important contributor to atherogenesis and endothelial dysfunction. Lipid derangements underlie hypertension onset and progression, by driving vascular stiffening and endothelial imbalance between vasoconstrictive angiotensin II and vasodilatory nitric oxide [4]. Emerging markers such as the Atherogenic Index of Plasma (AIP) and Lipid Accumulation Product (LAP) have demonstrated strong associations with PP and cardiovascular risk across diverse populations [7-9]. Their associations with elevated PP and LDL suggest a mechanistic link between lipid driven vascular remodeling and hemodynamic burden [10-12]. However, their integration into routine clinical practice is limited, underscoring the need for more accessible screening approaches, particularly in low-resource settings.

While a linear relationship between PP and adverse cardiovascular outcomes among normotensive and hypertensive individuals has been researched, a comparative analyses of conventional lipid parameters across hypertensive subgroups stratified by PP severity remains limited [10]. This study seeks to address that gap by examining the relationship between PP and traditional lipid metrics, including total cholesterol, LDL, HDL, TG, and the TC/HDL ratio among normotensive and hypertensive adults with either normal or widened PP. By identifying early deviations in lipid profiles associated with PP elevation, we seek to contextualize its relevance in complementing conventional cardiovascular risk indicators.

METHODS

A cross-sectional study was conducted following ethical clearance from the Institutional Ethics Committee and written informed consent from all participants. The study included 112 individuals aged 35-60 years, comprising 76 hypertensive patients from the Medicine outpatient department and 36 normotensive individuals from the Master Health Check-up department of PSG Hospitals. Subjects were categorized into three groups: Group A (normotensive; n = 36), Group B (hypertensive with normal pulse pressure of 40-60 mmHg; n = 43), and Group C (hypertensive with wide pulse pressure of >60 mmHg; n = 33).

Blood pressure classification followed the International Society of Hypertension - Global Practice Guidelines (ISH 2020 guidelines) [13]. Hypertension was defined as SBP ≥140 mmHg and/or DBP ≥90 mmHg for ≥1

year. Normotension was defined as SBP <120 mmHg and DBP <80 mmHg, recorded across three readings within one month, with no prior hypertension history and/or use of antihypertensives.

Exclusion criteria included individuals with co-existing comorbidities such as obesity, longstanding hypercholesterolemia, cardiovascular disease, stroke, malignancy, hematologic disorders, chronic renal or hepatic dysfunction and recent blood transfusions (within 3 months). Newly diagnosed hypertensives (duration <1 year) were also excluded according to ISH 2020 guidelines. While participants with diabetes mellitus, alcohol use, or smoking history were included, these variables were analyzed descriptively to assess their potential influence on lipid parameters and PP.

Sample size calculation was performed using OpenEpi version 3.01. Based on the difference in means formula, and incorporating reference values for PP and TG from Gaudio et al. [12], the estimated minimum sample size per group was 30, achieved in all three cohorts. Power was set at 80% with a 90% confidence interval.

Demographic details including age, sex, and medical history were recorded. Participants underwent general, systemic, and cardiovascular examinations. PP was measured via three consecutive blood pressure readings in accordance with ISH protocols. Laboratory investigations included complete blood count, random blood glucose, lipid profile (Total cholesterol, LDL, HDL, TG), and calculated TC/HDL ratio. Serum creatinine and ECG assessments were used to exclude participants with renal or cardiac pathology.

Group-wise comparisons focused on associations between lipid parameters across normotensives and hypertensives with normal/wide PP. The following analyses were performed: (1) Group A vs Group B, (2) Group A vs Group C, and (3) Group B vs Group C. Data analysis was performed using SPSS version 25. Mean, standard deviation, and percentages were calculated. Unpaired t-tests were applied for group comparisons, and a p-value of <0.05 was considered statistically significant.

OBSERVATIONS AND RESULTS

112 patients were studied (65 males; 47 females) consisting of seventy six hypertensives (normal PP = 43; wide PP = 33) and thirty six normotensives. Group A (normotensives) had 20 males and 16 females, group B (hypertensives with normal PP) had 25 males and 18 females and group C (hypertensives with wide PP) had 20 males and 13 females. The percentage gender distribution in the study and in each group is represented in Figure 1 and 2 respectively.

Figure 1: Gender-based population distribution.

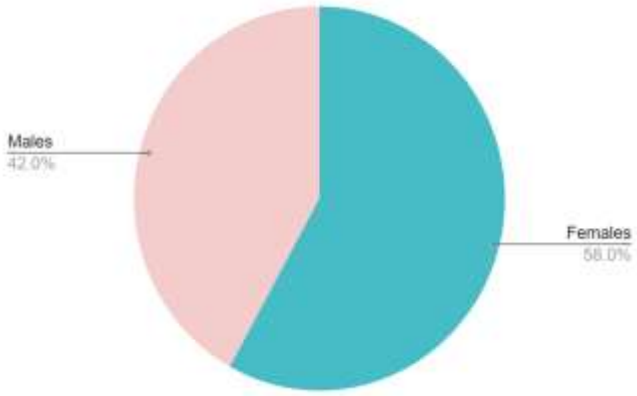
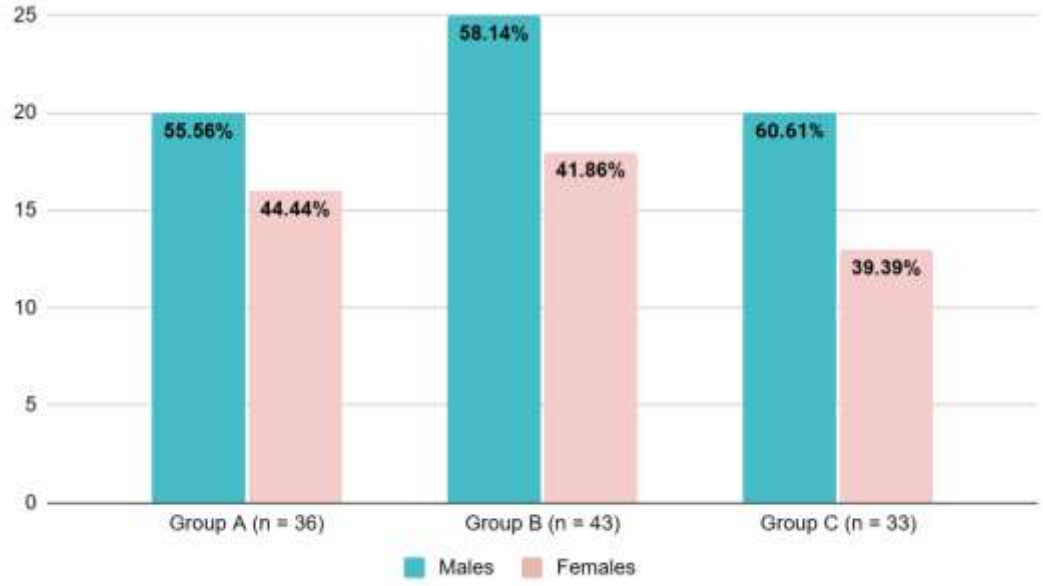
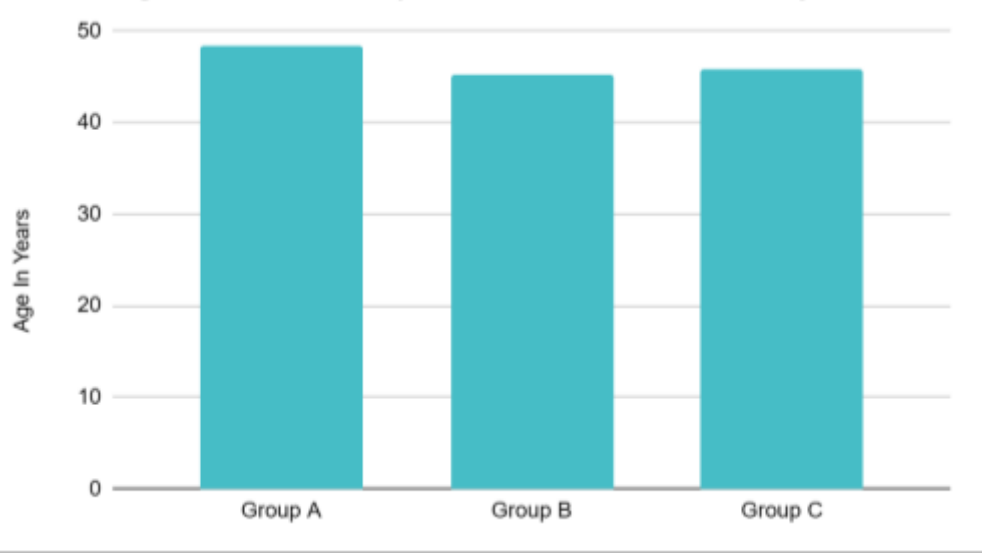


Figure 2: Gender distribution among 3 groups in the study.



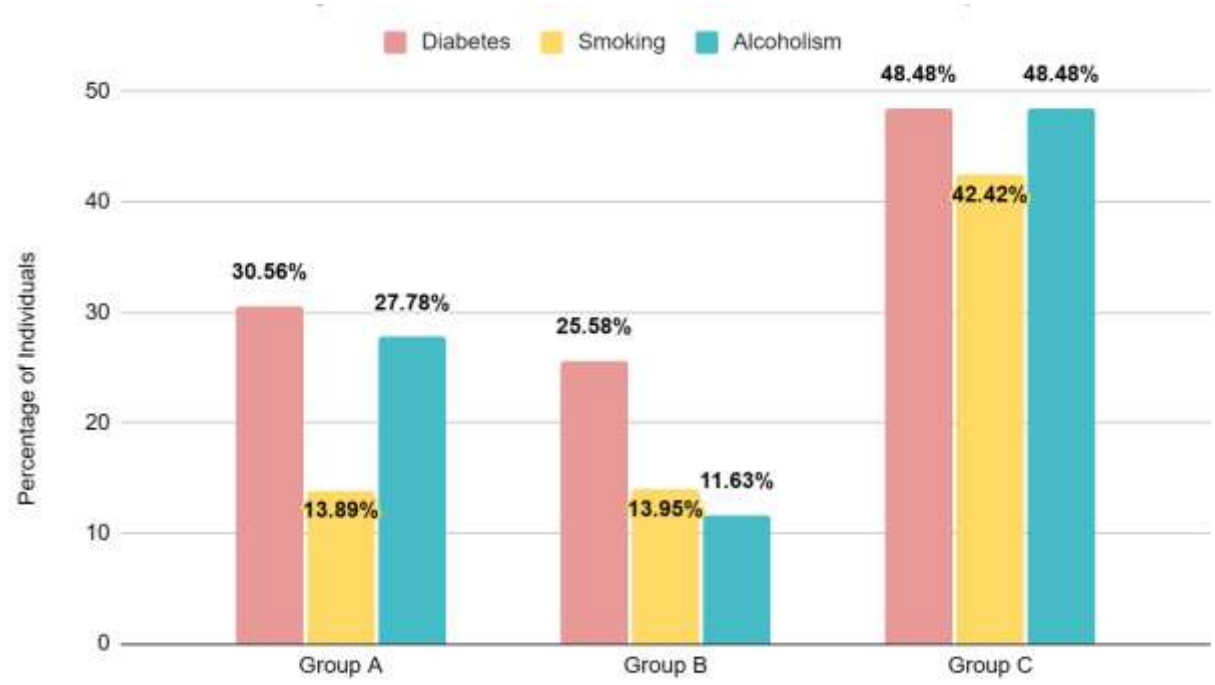
The mean and SD of age in each group are as follows: 48.33±8.117 in group A, 45.30±9.563 in group B and 45.75±10.304 in group C, as shown in Figure 3.

Figure 3: Mean age of 3 groups in the study.



The patients were assessed for history of smoking, alcohol abuse and comorbidities (diabetes mellitus). Figure 4 shows that compared to Group A and B, Group C has a larger percentage of individuals with diabetes mellitus, history of smoking and alcohol abuse.

Figure 4: Percentage of individuals with diabetes, smoking and alcohol intake in 3 groups.



Average PP, Pulse Rate and Random Blood Sugars were estimated and the average values are reported in Table 1.

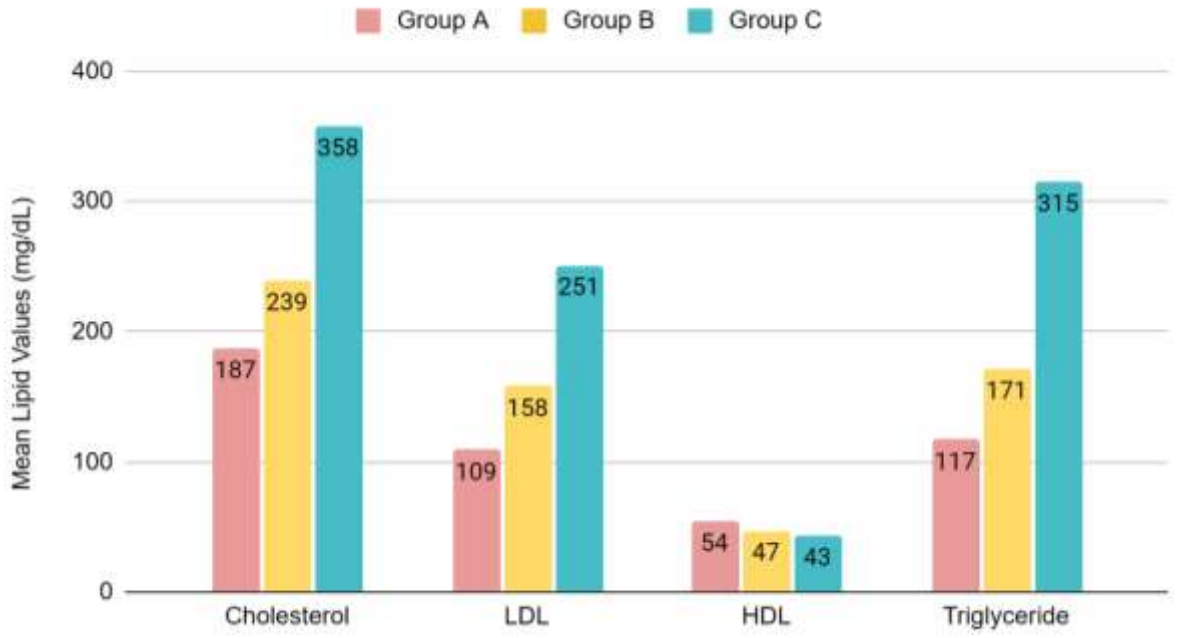
Table 1: Mean values of Pulse Pressure, Pulse Rate and Random blood sugars in 3 groups.

	Group A (Normotensives)	**Group B** (Hypertensive with Normal Pulse Pressure)	**Group C** (Hypertensives with Wide Pulse Pressure)
Pulse Pressure (mmHg)	45.55 ± 8.09	50 ± 8.16	75.45 ± 6.66
Pulse Rate (Per minute)	79.97 ± 8.61	78.65 ± 9.16	92.33 ± 11.06
Random blood sugar (mg/dl)	135.83 ± 39.07	129.49 ± 41.58	175.12 ± 76.18

Lipid profile of 3 groups:

Total cholesterol, LDL, HDL and TG of hypertensives with normal PP (Group B) were higher than normotensives (Group A) and lipid parameters of hypertensives with wide PP (Group C) was higher than normotensives (Group A) and hypertensives with normal PP (Group B). Figure 5 demonstrates the mean increase in TC, LDL and TG levels in hypertensives and decrease in HDL in hypertensives.

Figure 5: Mean values of lipid parameters in Group A, B and C.



Comparison of lipid parameters between the 3 groups:

On comparison of lipid parameters between 3 groups by unpaired t-test, statistically significant differences were observed between lipid parameters of normotensives (Group A) and hypertensives with normal PP (Group B) as shown in Table 2 as well as between lipid parameters of normotensives (Group A) and hypertensives with wide PP (Group C) as shown in Table 3. On comparison of the two groups of hypertensives, that is, Group B and Group C, as described by Table 4, a statistically significant difference was observed in all lipid parameters except HDL as represented by p values less than 0.05.

Table 2: Comparison of lipid profile between Group A and Group B.

Lipid Parameter (mg/dL)	**Group A** (N=36)	**Group B** (N=43)	**P value**
Total Cholesterol	187 ± 17.44	239.77 ± 74.16	0.0355*
LDL	108.75 ± 20.42	158.16 ± 74.11	0.00022*
HDL	54.83 ± 10.04	47.35 ± 14.97	0.0125*
TG	117.08 ± 20.47	171.42 ± 87.66	0.0005*
TC / HDL Ratio	3.55 ± 0.89	6.2 ± 4.65	0.00125*

Unpaired t test used, values are represented as mean±SD, $p<0.05$ is considered significant.

*represents statistically significant values.

Table 3: Comparison of lipid profile between Group A and Group C.

Lipid Parameter (mg/dl)	**Group A** (N=36)	**Group C** (N=33)	**P value**
Total Cholesterol	187 ± 17.44	358.15 ± 87.18	0.0024*
LDL	108.75 ± 20.42	251.88 ± 84.52	0.00022*
HDL	54.83 ± 10.04	43.21 ± 15.5	0.0004*
TG	117.08 ± 20.47	315.3 ± 119.25	0.0001*
TC/HDL Ratio	3.55 ± 0.89	9.8 ± 4.86	0.00023*

Unpaired t test used, values are represented as mean±SD, $p<0.05$ is considered significant.

*represents statistically significant values.

Table 4: Comparison of lipid profile between Group B and Group C.

Lipid Parameters (mg/dl)	Group B (N=43)	Group C (N=33)	P value
Total Cholesterol	239.77 ± 74.16	358.15 ± 87.18	0.001*
LDL	158.16 ± 74.11	251.88 ± 84.52	0.0046*
HDL	47.35 ± 14.97	43.21 ± 15.5	0.2433
TG	171.42 ± 87.66	315.3 ± 119.25	0.0023*
TC/HDL Ratio	6.2 ± 4.65	9.8 ± 4.86	0.0015*

Unpaired t-test used, values are represented as mean±SD, $p<0.05$ is considered significant.
*represents statistically significant values.

DISCUSSION

Hypertension continues to be a predominant cause of cardiovascular morbidity and mortality globally and in India [14,15]. PP, defined as the difference between SBP and DBP, can also be described as a ratio of stroke volume to compliance [16]. Hence, in individuals with essential hypertension, the increased peripheral vascular resistance, normal or decreased stroke volume, and age-related arterial stiffening may theoretically contribute to a widened PP [17,18]. However, in early hypertensives, particularly among younger individuals, the elevated blood pressure often reflects increased cardiac index and heart rate rather than vascular resistance, leading to minimal or inconsistent PP variations [17].

In our study, the mean PP in normotensives (45.55 ± 8.09 mmHg) closely aligned with population norms described by Chou et al., who found that age-linked PP elevation began at 40 in females and 50 in males [6]. Hypertensives with normal PP (Group B) showed only a modest 5 mmHg increase compared to normotensives, suggesting preserved vascular compliance in early-stage hypertension. Conversely, hypertensives with wide PP (Group C) showed a pronounced elevation, exceeding 30 mmHg above normotensives. Since age distributions across all groups were comparable, these findings support the notion that PP elevation may not be age-dependent alone.

Blacher et al. reported elevated PP as an independent predictor of cardiovascular morbidity, with a 20% increased risk of cardiac pathologies for every 10 mmHg rise in PP [19]. In a similar study, García-Palmieri et al. demonstrated PP ≥57 mmHg as a stand-alone marker of adverse cardiovascular events and mortality [20]. Our wide PP group (Group C), with a mean PP of 75.45 ± 6.66 mmHg, exceeded established thresholds for concern. Moreover, significant elevations in lipid parameters in this group reinforce the interplay between vascular mechanics and metabolic stress.

Diabetes mellitus was commonly encountered, particularly among wide PP hypertensives. Group C exhibited higher random blood glucose levels (175.12 ± 76.18 mg/dL) and greater diabetes prevalence. This corroborates with the findings by Mancusi et al., who noted that diabetics with wide PP were more likely to experience adverse cardiovascular events and structural complications [21]. Elevated systolic

pressures and PP in diabetics may reflect impaired endothelial regulation and increased arterial stiffness, both of which are hallmarks of long-standing metabolic vascular damage.

History of smoking and alcohol use followed a similar trend, with higher prevalence among wide PP hypertensives. Nearly 42% of Group C were smokers, triple the rate seen in normotensives. This corroborates with scientific evidence of reduced HDL and elevated TG among smokers, exacerbating dyslipidemia [22]. Prevalence of alcohol consumption tended towards Group C participants, with 48.48% reporting weekly intake, a 20-point increase over normotensives (Group A, 27.78%), while Group B reported substantially lower rates of use (11.11%). Silvia di Federico et al. documented a linear association between alcohol use and elevated SBP, even at minimal intake levels, which may partially explain the wide PP observed in Group C [23]. Conversely, Group B's preserved PP despite hypertension may reflect relatively maintained vascular compliance and lower metabolic stress. Literature on lipid metabolism also supports a J-shaped curve, whereby low to moderate alcohol may confer cardioprotective effects and increase HDL, while heavy use increases total cholesterol and cardiovascular risk [24]. The significantly deranged lipid profiles in Group C align with these findings. While the absence of stratified consumption data limits causal inference, the observed association between alcohol use, widened PP, and dyslipidemia warrants consideration.

Gender differences in PP and lipid dynamics are not widely studied. An article by Chou et al. found positive PP and lipid correlations in females and negative correlations in males [6]. However, our cohort which was predominantly male, especially in Groups B and C, showed a consistent rise in LDL, TG and TC/HDL ratios with PP. Although gender-specific analysis was not performed, our study highlights a possible area for future investigation.

A study in the Chinese population, showed a significant correlation between dyslipidemia (HDL, TC/HDL ratio, and LDL/HDL ratio) and carotid plaque formation [25]. This reinforces the interconnectedness of lipid imbalances, vascular remodeling, and elevated pulse pressure. Our results, showing significant intergroup differences in LDL, TG, total cholesterol, and TC/HDL ratio, support this association. Although HDL was reduced in hypertensives overall, subgroup differences were nonsignificant. Benetos et al. showed that the relative risk (95% confidence limits) for cardiovascular mortality after adjustment for age, mean blood pressure, and other risk factors was 1.20 (1.01 to 1.44) in normotensives and 1.09 (1.03 to 1.14) in hypertensives for every 10 mmHg increase in pulse pressure. Notably, normotensive men with PP>50 mmHg exhibited similar mortality rates to hypertensive men with PP<45 mmHg, suggesting that arterial stiffness, due to dyslipidemia induced vascular changes, may elevate cardiovascular risk irrespective of blood pressure category [10].

Comparative studies have demonstrated PP (hazard ratio = 1.29, 95% confidence interval 1.00– 1.78, p = 0.058) as a more reliable prognostic marker than SBP (hazard ratio = 1.14, 95% confidence interval 0.81–

1.60, p = 0.498) alone [26]. Moreover, ambulatory 24-hour PP monitoring had greater predictive outcomes than single-point measurements, suggesting that routine PP assessment could augment risk stratification protocols [9].

Lipid profiles and PP are both independently strong predictors of cardiovascular mortality. Prior studies have shown that PP, TG, and lipid ratios (LDL/HDL, TG/HDL, and LAP) were significantly elevated in pre-hypertensive and hypertensive individuals compared to normotensives, with values highest among hypertensives. Similarly, HDL was significantly lower in hypertensives, while LDL was significantly higher in both pre-hypertensives and hypertensives relative to normotensives [11]. Among these markers, LAP showed the strongest associations with both blood pressure and pulse pressure, though significant variations were also observed in HDL, LDL, and TG. Our findings corroborate to this study, where significant variations ($p<0.05$) were seen in HDL, LDL, total cholesterol, TG, and the TC/HDL ratio between normotensives (Group A) and hypertensives (Groups B and C), as well as between the hypertensive subgroups. However, no significant variations in HDL levels were seen among hypertensives with normal and wide pulse pressure.

Limitations

Our study's cross-sectional design limits causal inference between lipid parameters, PP, and hypertension. The observed relationships represent associations rather than directional effects, and longitudinal follow-up would be required to establish temporality. Gender-specific analysis was not performed despite unequal group distributions, as the sample size was insufficient to allow statistically meaningful stratification. Moreover, while PP and lipid parameters were statistically analyzed, multivariate adjustment for lifestyle confounders such as smoking and diabetes was not undertaken. This reflects the predefined analytic scope of the ICMR protocol, wherein these variables were not central to the study's primary objectives and were therefore not prioritized for modeling.

Public Health Implications

The association of PP with lipid parameters in hypertensive and normotensive adults reinforces its role as a complementary marker alongside traditional parameters. Moreover, it's easily measurable in outpatient settings, which may be useful in resource limited regions for early stratification of individuals with increased cardiovascular risk for prompt health education, targeted lifestyle interventions and lipid management.

CONCLUSIONS

This study demonstrates a positive association between pulse pressure and adverse lipid profiles. Significant elevations in total cholesterol, LDL, TG, and TC/HDL ratio were observed among hypertensives compared to normotensives, with the highest values seen in adults with wide pulse pressure. HDL levels were significantly reduced in hypertensives overall, though no statistical difference was noted between hypertensive subgroups. These findings suggest that pulse pressure may serve as a pragmatic marker for

lipid dysregulation and may complement traditional cardiovascular risk markers in both normotensive and hypertensive adults.

LIST OF ABBREVIATIONS

PP - Pulse Pressure
LDL - Low density lipoprotein
HDL - High density lipoprotein
TG - Triglycerides
TC/HDL ratio - Total cholesterol / high-density lipoprotein ratio
AIP - Atherogenic Index of Plasma
LAP - Lipid Accumulation product
SBP - Systolic Blood Pressure
DBP - Diastolic Blood Pressure
ISH 2020 Guidelines - International Society of Hypertension - Global Hypertension Practice Guidelines.
ECG - Electrocardiogram
SD - Standard Deviation

DISCLOSURES:

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2. **Source of Funding -** This study was conducted under the Indian Council of Medical Research - Short Term Studentship (ICMR STS) program for medical students.
3. **Conflict of Interest -** The authors declare no conflict of interest related to this study.
4. **Informed Consent -** Written informed consent was obtained from all participants for inclusion in the study and for publication of clinical details and results.
5. **Institutional Review Board (IRB) Approval -** Ethical approval was obtained from the Institutional Review Board of PSG Institute of Medical Sciences and Research (Ref No: 23/438). The study complies with the 'Institutional Health Insurance Portability and Accountability Act (HIPAA)' of PSG Institute of Medical Sciences and Research, thus ensuring that Patient Protected health information (PHI) was not collected nor documented.

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