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COMPARATIVE ASSESSMENT OF CARDIAC AUTONOMIC FUNCTION IN PREHYPERTENSIVES WITH AND WITHOUT FAMILIAL HYPERTENSION

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ABSTRACT

Background: Hypertension often has a familial predisposition, and early autonomic dysfunction may serve as a preclinical marker in individuals with a positive family history (FH) of hypertension. Cardiac autonomic function assessment using heart rate variability (HRV) and cardiovascular reflex tests may help identify such subclinical changes in prehypertensive individuals.

Materials and Methods: This cross-sectional study included 174 prehypertensive individuals aged 18–45 years, divided equally into two groups based on FH of hypertension: Group I (with FH, n=87) and Group II (without FH, n=87). Anthropometric and clinical data were collected. Cardiac autonomic function was evaluated using time-domain and frequency-domain HRV parameters and standard cardiovascular autonomic reflex tests (E:I ratio, Valsalva ratio, 30:15 ratio, handgrip test, and orthostatic test). Statistical analysis included independent t-tests and chi-square tests, with $p < 0.05$ considered significant.

Results: Group I had significantly higher waist circumference, waist-hip ratio, resting heart rate, and blood pressure values compared to Group II ($p < 0.05$). Time-domain HRV parameters (SDNN, RMSSD, pNN50, NN50, SDANN) and frequency-domain parameters (total power, HF component) were significantly reduced in the FH group, while LF (nu) and LF/HF ratio were elevated ($p < 0.001$), indicating sympathovagal imbalance. Reflex test parameters, including E:I ratio, Valsalva ratio, 30:15 ratio, and ΔDBP on handgrip, were significantly lower, while ΔHR on standing and sustained HR rise were higher in the FH group ($p < 0.001$). A significantly higher proportion of individuals in the FH group had ≥2 abnormal tests (43.7% vs. 11.5%, $p < 0.001$), sympathetic dysfunction (40.2% vs. 13.8%), and parasympathetic dysfunction (46.0% vs. 17.2%).

Conclusion: Prehypertensive individuals with a positive family history of hypertension show significant cardiac autonomic dysfunction, characterized by sympathetic overactivity and parasympathetic withdrawal. Early identification of autonomic impairment in this high-risk group may facilitate timely lifestyle interventions to prevent progression to hypertension.

Keywords: Cardiac autonomic function, Heart rate variability, Prehypertension, Family history, Hypertension, Sympathovagal balance, Reflex tests.

INTRODUCTION

Hypertension is a major public health problem globally, affecting over 1.28 billion adults aged 30–79 years, with a disproportionately high burden in low- and middle-income countries.[1] In India, the prevalence of hypertension among adults has been estimated to range between 25% to 30% in urban areas and 10% to 15% in rural populations.[2] However, a much larger proportion of the population falls under the category of prehypertension, which is defined by the Joint National Committee (JNC 7) as

a systolic blood pressure of 120–139 mmHg or diastolic blood pressure of 80–89 mmHg.[3] The National Family Health Survey-5 (NFHS-5) data indicates that approximately 43% of Indian adults aged 15 years and above have blood pressure levels in the prehypertensive range.[4] Prehypertension is not a benign condition—it is associated with a 2- to 3-fold increased risk of developing hypertension and a higher incidence of cardiovascular events.[5]

Autonomic imbalance, especially enhanced sympathetic and reduced parasympathetic activity, has been implicated in the pathogenesis of both prehypertension and established hypertension.[6] Cardiac autonomic function can be objectively assessed using standard autonomic function tests such as heart rate variability (HRV), deep breathing test, Valsalva maneuver, isometric handgrip test, and orthostatic stress test.[7] Reduced HRV and abnormal responses in these tests have been demonstrated in prehypertensive individuals, indicating early autonomic dysfunction even in the absence of sustained high blood pressure.[8]

A family history of hypertension is a well-known non-modifiable risk factor that significantly increases the likelihood of developing hypertension. Studies have shown that individuals with a positive family history have up to a 3-fold higher risk of developing hypertension compared to those without such history.[9] Importantly, genetic predisposition may influence autonomic regulation through altered baroreflex sensitivity, sympathetic overactivity, or decreased vagal tone, thereby contributing to early cardiovascular changes.[10]

Despite this, limited data exist on the interaction between family history and autonomic dysfunction in prehypertensive individuals. Understanding whether prehypertensives with a family history exhibit more pronounced autonomic imbalance than those without such history could have significant implications for early risk stratification and preventive cardiology. In the Indian context, early detection of subclinical autonomic changes in genetically predisposed prehypertensive individuals may help delay or prevent the onset of hypertension through timely lifestyle and pharmacological interventions. Therefore, this study aimed to assess and compare cardiac autonomic functions in prehypertensive individuals with and without a family history of hypertension, using standardized autonomic function testing protocols.

MATERIALS AND METHODS

Study Design and Setting: This cross-sectional observational study was conducted in the Department of Physiology at a tertiary care academic hospital in North India. The study was carried out over a period of 2 years between June 2022 to June 2024, following approval from the Institutional Ethics Committee. Written informed consent was obtained from all participants prior to their enrollment in the study.

Study Population and Grouping: The study enrolled apparently healthy individuals aged between 18 and 40 years who were classified as prehypertensive according to the JNC 7 criteria, with systolic blood pressure between 120–139 mmHg and/or diastolic blood pressure between 80–89 mmHg. Blood pressure was measured on two separate occasions at least one week apart using a calibrated digital sphygmomanometer (Omron HEM-7120, Japan) in the right arm, with the subject seated comfortably after a 5-minute rest. The average of two readings per session was recorded.

Participants were divided into two groups based on their family history of hypertension. Group I consisted of prehypertensive individuals with a first-degree relative (parent or sibling) diagnosed with hypertension before the age of 60, whereas Group II included prehypertensive individuals without any known family history of hypertension. Family history was obtained through a structured questionnaire and verified with clinical records wherever available.

Inclusion and Exclusion Criteria

Inclusion criteria were: age between 18 and 40 years, blood pressure within the prehypertensive range on two separate visits, and willingness to participate. Exclusion criteria included previously diagnosed hypertension, diabetes mellitus, cardiovascular or neurological disorders, obesity (BMI ≥30 kg/m²), current smoking, alcohol consumption, regular caffeine intake, use of medications affecting autonomic function (e.g., beta-blockers, antidepressants), and engagement in athletic training. Pregnant and lactating women were also excluded.

Sample Size Calculation

The sample size was calculated based on a previous Indian study by Narasimhan et al., comparing HRV parameters (SDNN) in prehypertensive individuals with and without a family history of hypertension.[11] The mean SDNN in the two groups was reported as 39.5 ± 12.1 ms and 47.8 ± 13.4 ms, respectively. With a 95% confidence level, 80% power, and using the formula for comparison of two independent means, the minimum required sample size was calculated to be 78 participants per group. Accounting for a 10% potential attrition rate, the final sample size was fixed at 174 participants, with 87 individuals in each group.

Anthropometric and Clinical Measurements

Anthropometric data including height, weight, and body mass index (BMI) were recorded using standard techniques. Height was measured to the nearest 0.1 cm using a wall-mounted stadiometer, and weight to the nearest 0.1 kg using a digital scale. BMI was calculated as weight in kilograms divided by the square of height in meters (kg/m²). General physical and systemic examinations were carried out to ensure participant eligibility and exclude underlying pathologies.

Autonomic Function Testing Protocol

Cardiac autonomic function tests were performed in a temperature-controlled autonomic function laboratory between 9:00 AM and 11:00 AM to minimize circadian variation. Participants were

instructed to fast for at least 8 hours, avoid caffeine and strenuous activity for 12 hours prior to testing, and were allowed to rest for 15 minutes before the tests commenced. The following non-invasive autonomic function tests were conducted:

Heart Rate Variability (HRV) Analysis was performed using a 5-minute resting ECG recorded in lead II using the RMS Polyrite D system (Recorders & Medicare Systems Pvt. Ltd., India). The RR intervals were analyzed using Kubios HRV software (version [insert version]). Time-domain parameters such as SDNN, RMSSD, and pNN50, and frequency-domain parameters such as LF (low frequency), HF (high frequency), and LF/HF ratio were calculated in accordance with the guidelines of the Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology.

Deep Breathing Test involved instructing the participants to perform six deep breathing cycles (inhaling for 5 seconds and exhaling for 5 seconds). ECG was recorded continuously, and the E:I ratio was computed as the ratio of the longest R-R interval during expiration to the shortest R-R interval during inspiration, with the average of all six cycles considered.

Valsalva Maneuver required the participant to blow into a mouthpiece connected to a mercury manometer and maintain an intrathoracic pressure of 40 mmHg for 15 seconds. ECG was monitored throughout the maneuver. The Valsalva ratio was calculated as the ratio of the longest R-R interval after the maneuver to the shortest R-R interval during the strain phase.

Isometric Handgrip Test was carried out using an INCO handgrip dynamometer. Participants were instructed to sustain 30% of their maximum voluntary contraction for 3 minutes with their dominant hand. Diastolic blood pressure was recorded at baseline and at the end of the handgrip period, and the rise in DBP was noted. An increase of ≥16 mmHg was considered a normal sympathetic response.

Orthostatic Test (Lying to Standing Test) evaluated parasympathetic reactivity. Participants moved from a supine to a standing position, and ECG was continuously recorded. The 30:15 ratio was determined by calculating the ratio of the R-R interval at the 30th beat after standing to the 15th beat. A ratio >1.04 was considered normal.

Statistical Analysis

All data were compiled in Microsoft Excel and analyzed using IBM SPSS Statistics version 20.0. Quantitative variables were expressed as mean ± standard deviation (SD), and categorical variables were presented as frequencies and percentages. Normality of distribution was assessed using the Shapiro–Wilk test. Intergroup comparisons of continuous variables were performed using the independent samples t-test or Mann–Whitney U test, as appropriate. The Chi-square test was used for categorical variables. A p-value of less than 0.05 was considered statistically significant.

RESULTS

Participants with a family history of hypertension (Group I) demonstrated a more unfavorable cardiovascular profile compared to those without such history (Group II). Although the mean age and gender distribution were similar across groups, Group I had significantly higher waist circumference (91.4 ± 6.1 cm vs. 88.7 ± 5.9 cm) and waist-hip ratio (0.92 ± 0.04 vs. 0.89 ± 0.05), suggesting greater central obesity—an established risk factor for hypertension progression. Systolic and diastolic blood pressures were also significantly elevated in Group I, alongside a higher resting heart rate and longer prehypertension duration, indicating early autonomic dysregulation. Lifestyle factors such as physical activity, smoking, and alcohol use did not differ significantly, implying that the observed variations are more likely attributable to familial predisposition [Table 1].

Table 1: Comparison of Sociodemographic and Clinical Characteristics between Prehypertensive Individuals with and without a Family History of Hypertension (n=174).

Variable	Group I (With FH) (n=87)	Group II (Without FH) (n=87)	Test Statistic, p-value
	Frequency (%)/mean ± SD		
Age (years)	34.6 ± 6.2	33.9 ± 6.5	t = 0.81, 0.419
Gender			
Male	54 (62.1%)	52 (59.8%)	χ^2 = 0.08, 0.773
Female	33 (37.9%)	35 (40.2%)	
BMI (kg/m²)	26.1 ± 2.8	25.3 ± 2.7	t = 1.91, 0.058
Waist Circumference (cm)	91.4 ± 6.1	88.7 ± 5.9	t = 2.81, 0.006
Waist-Hip Ratio	0.92 ± 0.04	0.89 ± 0.05	t = 3.54 (p < 0.001
Physical Activity			
Low	38 (43.7%)	28 (32.2%)	χ^2 = 2.57, 0.276
Moderate	37 (42.5%)	43 (49.4%)	
High	12 (13.8%)	16 (18.4%)	
Smoking Status	19 (21.8%)	15 (17.2%)	χ^2 = 0.53, 0.468
Alcohol Use	16 (18.4%)	12 (13.8%)	χ^2 = 0.63, 0.428
Systolic BP (mmHg)	134.8 ± 4.2	132.6 ± 4.5	t = 3.11, 0.002
Diastolic BP (mmHg)	86.9 ± 3.7	85.2 ± 3.4	t = 3.08, 0.002
Resting HR (bpm)	80.2 ± 6.3	77.5 ± 6.1	t = 2.76, 0.006
Duration of Prehypertension (months)	11.2 ± 3.8	9.7 ± 4.1	t = 2.40, 0.017

Heart rate variability (HRV) parameters were significantly reduced in prehypertensive individuals with a family history of hypertension (Group I) compared to those without (Group II), indicating impaired autonomic regulation. Time-domain indices such as SDNN (39.8 ± 11.5 vs. 47.2 ± 12.9 ms, $p < 0.001$), RMSSD (27.4 ± 9.6 vs. 32.9 ± 10.2 ms, $p < 0.001$), and pNN50 (10.6 ± 4.2% vs. 14.3 ± 5.1%, $p < 0.001$) were significantly lower in Group I. Similarly, NN50 counts, Mean RR intervals, SDANN, and the HRV triangular index were all significantly reduced in Group I ($p < 0.01$ for all), reflecting decreased parasympathetic activity and overall autonomic imbalance. These findings suggest that even in the prehypertensive stage, familial predisposition may accelerate autonomic dysfunction [Table 2].

Table 2: Comparison of Time Domain Heart Rate Variability (HRV) Parameters between Groups (n=174).

HRV Parameter	Group I (With FH) (n=87)	Group II (Without FH) (n=87)	Test Statistic, p-value
	mean ± SD		
SDNN (ms)	39.8 ± 11.5	47.2 ± 12.9	t = 4.14, < 0.001
RMSSD (ms)	27.4 ± 9.6	32.9 ± 10.2	t = 3.54, < 0.001
pNN50 (%)	10.6 ± 4.2	14.3 ± 5.1	t = 5.04, < 0.001
NN50 (count)	38.2 ± 18.6	49.5 ± 21.2	t = 3.57, < 0.001
Mean RR (ms)	742.8 ± 92.5	782.4 ± 89.3	t = 2.73, 0.007
HRV Triangular Index	14.2 ± 3.5	16.6 ± 3.8	t = 4.01, < 0.001
SDANN (ms)	28.1 ± 9.2	33.5 ± 9.8	t = 3.48, < 0.001

Frequency-domain analysis further revealed significant autonomic imbalance in prehypertensive individuals with a family history of hypertension. Group I showed markedly lower Total Power (1320.2 ± 425.4 vs. 1655.6 ± 467.1 ms², $p < 0.001$), HF power (370.9 ± 129.2 vs. 491.7 ± 134.4 ms², $p < 0.001$), and VLF and LF components ($p < 0.05$ and $p = 0.003$, respectively), indicating reduced overall and parasympathetic activity. Moreover, normalized LF was significantly higher (62.8 ± 10.6 vs. 56.3 ± 9.9, $p < 0.001$), while HF (nu) was lower in Group I, leading to a significantly elevated LF/HF ratio (1.72 ± 0.62 vs. 1.29 ± 0.57, $p < 0.001$), suggestive of sympathetic predominance. These findings underscore a shift toward sympathovagal imbalance in individuals with a familial predisposition to hypertension, even at the prehypertensive stage [Table 3].

Table 3: Comparison of Frequency Domain HRV Parameters between Groups (n=174).

HRV Parameter	Group I (With FH) (n=87)	Group II (Without FH) (n=87)	Test Statistic, p-value
	mean ± SD		
Total Power (ms²)	1320.2 ± 425.4	1655.6 ± 467.1	t = 4.68, < 0.001
VLF (ms²)	215.7 ± 96.1	248.6 ± 104.2	t = 2.00, 0.047
LF (ms²)	435.1 ± 138.5	502.5 ± 149.8	t = 3.01, 0.003
HF (ms²)	370.9 ± 129.2	491.7 ± 134.4	t = 5.41, < 0.001
LF (nu)	62.8 ± 10.6	56.3 ± 9.9	t = 3.95, < 0.001
HF (nu)	37.2 ± 9.1	43.7 ± 8.5	t = 4.46, < 0.001
LF/HF Ratio	1.72 ± 0.62	1.29 ± 0.57	t = 3.83, < 0.001

Autonomic reactivity tests demonstrated significant differences between groups, with prehypertensive individuals having a family history of hypertension (Group I) showing lower parasympathetic responses. The E:I ratio (1.18 ± 0.06 vs. 1.26 ± 0.07, $p < 0.001$), Valsalva ratio (1.21 ± 0.07 vs. 1.28 ± 0.06, $p < 0.001$), and 30:15 ratio (1.01 ± 0.05 vs. 1.07 ± 0.06, $p < 0.001$) were significantly lower in Group I, indicating reduced cardiovagal modulation. Sympathetic function also showed notable alterations, with Group I exhibiting a blunted rise in diastolic BP during handgrip (13.4 ± 2.7 vs. 17.1 ± 3.2 mmHg, $p < 0.001$) and a significantly higher heart rate increment on standing (14.8 ± 4.9 vs. 11.2 ± 5.1 bpm, $p < 0.001$). Moreover, the proportion of participants with sustained HR rise was higher in Group I (24.1% vs. 10.3%, $p = 0.015$), and the average number of abnormal tests was also greater (2.1 ± 1.2 vs. 1.3 ± 0.9, $p < 0.001$), reinforcing the presence of early autonomic dysfunction in those with a positive family history of hypertension [Table 4].

Table 4: Comparison of Standard Autonomic Function Test Results between Groups (n=174).

Test	Group I (With FH) (n=87)	Group II (Without FH) (n=87)	Test Statistic, p-value
	Frequency (%)/mean ± SD		
E:I Ratio	1.18 ± 0.06	1.26 ± 0.07	t = 7.48, < 0.001
Valsalva Ratio	1.21 ± 0.07	1.28 ± 0.06	t = 6.89, < 0.001
30:15 Ratio	1.01 ± 0.05	1.07 ± 0.06	t = 7.01, < 0.001
Δ DBP on Handgrip (mmHg)	13.4 ± 2.7	17.1 ± 3.2	t = 7.35, < 0.001
Δ HR on Standing (bpm)	14.8 ± 4.9	11.2 ± 5.1	t = 4.64, < 0.001
Sustained HR Rise (yes %)	21 (24.1%)	9 (10.3%)	$\chi^2 = 5.96$, 0.015
No. of Abnormal Tests (mean ± SD)	2.1 ± 1.2	1.3 ± 0.9	t = 4.84, < 0.001

A significantly higher proportion of participants in Group I (with family history of hypertension) exhibited abnormal autonomic parameters compared to Group II. Specifically, abnormal E:I ratio was observed in 47.1% of Group I versus 19.5% in Group II ($p < 0.001$), and Valsalva ratio abnormalities were present in 37.9% vs. 12.6% ($p < 0.001$). Similarly, abnormal 30:15 ratio and impaired diastolic BP response to handgrip were more frequent in Group I (50.6% and 56.3%, respectively) than in Group II (20.7% and 18.4%, $p < 0.001$ for both). Overall, 43.7% of individuals in Group I had two or more abnormal test results compared to only 11.5% in Group II. Sympathetic and parasympathetic dysfunctions were also significantly more prevalent in Group I (40.2% and 46.0%, respectively) than Group II (13.8% and 17.2%, $p < 0.001$), highlighting an early subclinical autonomic imbalance in individuals with a familial predisposition to hypertension [Table 5].

Table 5: Frequency of Abnormal Autonomic Parameters and Dysfunction Patterns in Both Groups (n=174).

Abnormal Test/Parameter	Group I (With FH) (n=87)	Group II (Without FH) (n=87)	Test Statistic, p-value
	Frequency (%)		
E:I Ratio (<1.21)	41 (47.1%)	17 (19.5%)	$\chi^2 = 15.88$, < 0.001
Valsalva Ratio (<1.21)	33 (37.9%)	11 (12.6%)	$\chi^2 = 14.56$, < 0.001
30:15 Ratio (<1.04)	44 (50.6%)	18 (20.7%)	$\chi^2 = 17.54$, < 0.001
Δ DBP on Handgrip (<16 mmHg)	49 (56.3%)	16 (18.4%)	$\chi^2 = 28.87$, < 0.001
≥ 2 Abnormal Tests	38 (43.7%)	10 (11.5%)	$\chi^2 = 22.16$, < 0.001
Sympathetic Dysfunction	35 (40.2%)	12 (13.8%)	$\chi^2 = 16.91$, < 0.001
Parasympathetic Dysfunction	40 (46.0%)	15 (17.2%)	$\chi^2 = 18.67$, < 0.001

DISCUSSION

In the present study, we observed that prehypertensive individuals with a positive family history (FH) of hypertension exhibited significantly altered cardiac autonomic function when compared to their counterparts without such a family history. This difference was evident across multiple domains—anthropometric, hemodynamic, heart rate variability (HRV), and cardiovascular reflex tests—underscoring the compounded risk that familial predisposition adds even before the clinical onset of hypertension. Individuals in the FH group had notably higher waist circumference and waist-hip ratios, both of which are established markers of central obesity and are strongly correlated with increased sympathetic activity and diminished parasympathetic modulation. Elevated systolic and diastolic blood pressure, along with higher resting heart rates in the FH group, reflect an early autonomic imbalance. These anthropometric and hemodynamic trends mirror findings from previous studies, by Amaral et al. and Goh et al., which emphasize the role of central adiposity and familial predisposition in the pathogenesis of early cardiovascular risk.[11,12]

A deeper analysis of time-domain HRV parameters revealed significantly lower SDNN, RMSSD, pNN50, NN50, and SDANN values in prehypertensive individuals with FH. For instance, SDNN, an indicator of overall HRV reflecting both sympathetic and parasympathetic contributions, was reduced from 47.2 ± 12.9 ms in Group II to 39.8 ± 11.5 ms in Group I ($p < 0.001$). Similarly, RMSSD and pNN50, which predominantly reflect parasympathetic activity, were also markedly lower in the FH group. These reductions signal a withdrawal of vagal tone and reduced adaptability of the autonomic nervous system to physiologic stressors. Shah et al., and Wadoo et al., reported comparable reductions in time-domain HRV indices among normotensive offspring of hypertensive parents, emphasizing that such changes precede overt clinical hypertension.[13,14] Bansal et al., and Chandrasekaran et al., have similarly documented diminished parasympathetic indices among young adults with hypertensive parents, suggesting that autonomic dysfunction is both an inherited and an early detectable marker of future cardiovascular compromise.[15,16]

Frequency-domain HRV analysis further substantiated these findings. Total power and HF components, which reflect parasympathetic (vagal) modulation, were significantly lower in Group I, indicating parasympathetic withdrawal. Concurrently, the elevated LF (nu) and LF/HF ratio in the FH group signify increased sympathetic activity and a shift toward sympathovagal imbalance. The mean LF/HF ratio in Group I (1.72 ± 0.62) was substantially higher than in Group II (1.29 ± 0.57), with statistical significance ($p < 0.001$). This elevated ratio is considered a hallmark of autonomic imbalance and has been associated with a higher risk of developing hypertension and cardiovascular events. Billman et al., were among the early proponents of using LF/HF ratio as a marker of autonomic regulation, while more recent work by Dhamayanthi et al., and Kumar et al., in South India also identified a significantly higher LF/HF ratio in normotensive offspring of hypertensives, echoing our findings.[17-19]

In addition to HRV measures, standard cardiovascular autonomic reflex tests (CARTs) offered compelling insights into functional autonomic impairments. E:I ratio, Valsalva ratio, 30:15 ratio, and ΔDBP on isometric handgrip—all of which assess vagal and sympathetic responses—were significantly lower in individuals with FH. Conversely, ΔHR on standing and the presence of sustained heart rate rise, indicative of orthostatic sympathetic hyperactivity, were more prominent in the FH group. These findings suggest an impaired

baroreflex and altered central autonomic regulation. Notably, the proportion of participants with two or more abnormal test results was 43.7% in Group I compared to only 11.5% in Group II ($p < 0.001$), highlighting the burden of subclinical autonomic dysfunction in the genetically predisposed population. These observations are in line with the results reported by Karmacharya et al., and Matthews et al., who found reduced reflex responsiveness in normotensive first-degree relatives of hypertensives, thereby validating the use of CARTs as sensitive indicators of early autonomic derangement.[20,21]

Furthermore, 40.2% of individuals in the FH group demonstrated sympathetic dysfunction and 46.0% exhibited parasympathetic dysfunction, while only 13.8% and 17.2%, respectively, were affected in the non-FH group. These proportions reaffirm the hypothesis that familial predisposition contributes to early autonomic dysregulation. The mechanism behind this may be multifactorial, including altered central autonomic processing, increased basal sympathetic tone, and reduced vagal afferent signaling. Genetic studies have identified polymorphisms in genes regulating the renin-angiotensin-aldosterone system and sympathetic receptors (e.g., ADRB2) in individuals with a family history of hypertension, which may partly explain the autonomic differences observed.[22,23] Moreover, lifestyle factors such as reduced physical activity and higher psychological stress, often shared within families, may further aggravate autonomic imbalance.

The clinical implications of these findings are significant. Early identification of autonomic dysregulation in individuals with prehypertension and positive FH can aid in stratifying cardiovascular risk more precisely. Non-pharmacological interventions such as physical activity, stress reduction, and dietary modifications have been shown to improve HRV and restore autonomic balance, potentially delaying the progression to overt hypertension. Studies by Chiang et al., and George et al., have highlighted the plasticity of the autonomic nervous system and the potential for reversing early dysfunction with lifestyle changes.[24,25] Thus, incorporating simple, non-invasive autonomic assessments like HRV and CARTs into clinical screening for at-risk populations may serve as a cost-effective strategy in resource-constrained settings such as India.

CONCLUSION

Thus, our study highlights the importance of evaluating cardiac autonomic parameters in at-risk individuals even before hypertension develops. The findings underscore the utility of HRV and reflex tests as non-invasive, feasible tools in predicting early autonomic derangements. Further longitudinal studies are warranted to evaluate the predictive value of these markers for future cardiovascular events in the Indian population.

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