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Original Research Article

COMPARISON OF HEART RATE VARIABILITY BETWEEN HYPERTENSIVE AND NORMOTENSIVE ADULTS: A CROSS-SECTIONAL OBSERVATIONAL STUDY

Shrivallabh Pujari[1], Manthappa M[2], Vijaya Vageesh Y[3], Varsha Tandure[4]

[1]Postgraduate Resident, Department of General Medicine, JSS Medical College, JSS Academy of Higher Education & Research, Mysuru, Karnataka, India.
[2]Professor, Department of General Medicine, JSS Medical College, JSS Academy of Higher Education & Research, Mysuru, Karnataka, India.
[3]Associate Professor, Department of Physiology, JSS Medical College, JSS Academy of Higher Education & Research, Mysuru, Karnataka, India.
[4]Associate Professor, JSS Medical College, JSS Academy of Higher Education & Research, Mysuru, Karnataka, India.

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Corresponding Author:
Dr. Varsha Tandure,
Associate Professor, JSS Medical College, JSS Academy of Higher Education & Research, Mysuru, Karnataka, India.

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ABSTRACT

Background: Hypertension is one of the leading modifiable risk factors for cardiovascular morbidity and mortality worldwide. Beyond elevated blood pressure, autonomic nervous system dysfunction has emerged as a major contributor to the initiation and progression of hypertension. Heart rate variability (HRV), a simple, non-invasive electrocardiographic measure of autonomic function, provides quantitative assessment of sympathovagal balance and has gained importance as a potential biomarker for cardiovascular risk stratification. Despite increasing evidence supporting its utility, comparative data between hypertensive and normotensive Indian adults remain limited. This study evaluated HRV parameters in hypertensive and normotensive adults and examined the relationship between disease duration and autonomic dysfunction.

Materials and Methods: A hospital-based cross-sectional observational study was conducted among 128 adults aged 30–60 years, including 64 hypertensive and 64 normotensive participants. Following standardized clinical assessment, HRV was recorded using a 10-minute resting Lead II electrocardiogram. Time-domain indices (SDNN, RMSSD, NN50 and pNN50) and frequency-domain indices (LF, HF, LF/HF ratio and Total Power) were analysed. Clinical and anthropometric variables were compared between groups using appropriate statistical tests, with $p<0.05$ considered statistically significant.

Results: Hypertensive participants demonstrated significantly greater body mass index and blood pressure values than normotensive controls. Although overall HRV parameters showed no statistically significant differences between study groups, increasing duration of hypertension was associated with progressive reductions in RMSSD and HF power, indicating declining parasympathetic activity. These findings suggest that autonomic dysfunction becomes increasingly evident with longer disease duration.

Conclusion: Heart rate variability is a useful non-invasive tool for evaluating autonomic cardiovascular regulation in hypertensive individuals. Progressive impairment of vagal-mediated HRV indices with increasing duration of hypertension supports the role of HRV in early cardiovascular risk assessment, disease monitoring and identification of patients at higher risk of adverse cardiovascular outcomes.

Keywords: Heart rate variability; Hypertension; Autonomic nervous system; Cardiovascular risk; Sympathovagal balance.

INTRODUCTION

Hypertension remains one of the most important public health challenges worldwide and continues to be a leading contributor to cardiovascular morbidity and premature mortality. According to contemporary epidemiological estimates, over one billion adults are affected globally, and the prevalence continues to increase because of population ageing, urbanization, sedentary lifestyle, obesity, unhealthy dietary practices and metabolic disorders. Persistent elevation of blood pressure substantially increases the risk of coronary artery disease, heart failure, stroke, chronic kidney disease, peripheral arterial disease and premature death. Although pharmacological treatment has improved considerably over the past few decades, a significant proportion of hypertensive individuals continue to have inadequately controlled blood pressure, suggesting that mechanisms beyond vascular resistance alone contribute to disease progression.[1]

Among these mechanisms, autonomic nervous system dysregulation has gained considerable attention. Blood pressure homeostasis is maintained through a finely balanced interaction between sympathetic and parasympathetic influences acting on the cardiovascular system. Under physiological conditions, these two divisions continuously regulate heart rate, myocardial contractility, vascular tone and baroreflex responsiveness according to metabolic requirements. In hypertension, however, this balance becomes disturbed, resulting in sympathetic overactivity accompanied by reduced parasympathetic modulation. Such autonomic imbalance contributes not only to sustained elevation of blood pressure but also to endothelial dysfunction, vascular remodelling, left ventricular hypertrophy and increased cardiovascular risk.[2,3]

Heart rate variability (HRV) has emerged as one of the most reliable non-invasive methods for evaluating autonomic nervous system function. HRV represents the physiological variation in consecutive R–R intervals generated by the dynamic interaction between sympathetic and parasympathetic neural activity. Rather than reflecting irregular cardiac rhythm, greater beat-to-beat variability indicates a healthy cardiovascular system capable of responding appropriately to internal and external physiological stimuli. Conversely, reduced HRV reflects impaired autonomic adaptability and has consistently been associated with adverse cardiovascular outcomes, arrhythmias, sudden cardiac death and all-cause mortality.[4,5]

The physiological basis of HRV lies in continuous modulation of sinoatrial node activity by autonomic neural pathways. Parasympathetic stimulation, primarily mediated through the vagus nerve, produces rapid beat-to-beat alterations in heart rate and is responsible for high-frequency oscillations, whereas sympathetic activation exerts relatively slower effects that increase heart rate and myocardial contractility. These opposing influences generate characteristic fluctuations that can be quantified using both time-domain and frequency-domain analytical techniques. Together, these measurements provide valuable insights into sympathovagal balance and cardiovascular autonomic regulation.[6]

Time-domain parameters such as the standard deviation of normal-to-normal intervals (SDNN), root mean square of successive differences (RMSSD), NN50 and pNN50 primarily evaluate overall variability and parasympathetic activity. Frequency-domain analysis further separates autonomic modulation into low-frequency (LF) and high-frequency (HF) spectral components, while the LF/HF ratio has traditionally been used to estimate sympathovagal balance. Total Power represents the overall autonomic activity contributing to heart rate fluctuations. Alterations in these parameters have been reported in several cardiovascular disorders and have demonstrated prognostic value independent of conventional cardiovascular risk factors.[7]

Numerous studies have reported reduced HRV among individuals with hypertension, suggesting that autonomic dysfunction may precede the clinical manifestation of sustained blood pressure elevation. Increased sympathetic drive together with withdrawal of vagal activity promotes vasoconstriction, sodium retention, activation of the renin–angiotensin–aldosterone system and progressive structural changes within the cardiovascular system. These mechanisms perpetuate hypertension while simultaneously increasing susceptibility to adverse cardiovascular events. Consequently, HRV has attracted considerable interest as a potential biomarker for identifying individuals at elevated cardiovascular risk before irreversible target-organ damage develops.[8,9]

Beyond diagnosis, HRV also possesses considerable prognostic significance. Lower HRV has been associated with myocardial infarction, chronic heart failure, diabetic autonomic neuropathy, malignant arrhythmias and sudden cardiac death. In patients with established hypertension, autonomic dysfunction reflected by reduced HRV has been linked to greater severity of target-organ damage and poorer long-term cardiovascular outcomes. Because HRV recording is inexpensive, non-invasive and easily reproducible, it has become increasingly applicable in both clinical practice and research settings for evaluating cardiovascular autonomic function.[10]

Despite growing international evidence, relatively few studies have comprehensively compared HRV profiles between hypertensive and normotensive Indian adults while simultaneously evaluating the influence of hypertension duration on autonomic function. Population-specific differences related to ethnicity, lifestyle, body composition and

environmental factors may influence autonomic regulation and therefore require dedicated evaluation. The present cross-sectional observational study was undertaken to compare time-domain and frequency-domain HRV parameters between hypertensive and normotensive adults and to determine whether longer duration of hypertension is associated with progressive autonomic dysfunction. The findings are expected to enhance understanding of cardiovascular autonomic impairment in hypertension and further establish HRV as a clinically useful tool for cardiovascular risk assessment and disease monitoring.

MATERIALS AND METHODS

Study Design

This hospital-based cross-sectional observational study was conducted to compare heart rate variability (HRV) between hypertensive and normotensive adults attending the Department of General Medicine at a tertiary care teaching hospital. The study was designed to evaluate autonomic nervous system function using standardized HRV assessment under resting conditions. Ethical approval was obtained from the Institutional Ethics Committee before commencement of the study, and written informed consent was obtained from all participants prior to enrolment.

Study Duration and Setting

The study was carried out in the Department of General Medicine in collaboration with the Department of Physiology at a tertiary care teaching hospital after Institutional Ethics Committee approval. All recordings and clinical assessments were performed in a dedicated laboratory under standardized environmental conditions to minimize external influences on autonomic function.

Study Population

A total of **128 participants** aged **30–60 years** were enrolled in the study. Participants were divided equally into two groups:

- **Normotensive adults (n = 64)**
- **Hypertensive adults (n = 64)**

Hypertension was diagnosed according to standard clinical criteria and included patients receiving treatment for primary hypertension. Normotensive individuals had no previous diagnosis of hypertension and demonstrated normal blood pressure during clinical evaluation.

Sample Size

The study included 128 participants, comprising 64 hypertensive and 64 normotensive adults, as determined in the approved study protocol. Equal allocation between study groups ensured adequate comparison of HRV parameters and minimized selection imbalance.

Inclusion Criteria

Hypertensive Group

- Adults aged 30–60 years.
- Diagnosed with primary hypertension.
- Receiving regular follow-up or treatment.
- Willing to participate and provide written informed consent.

Normotensive Group

- Adults aged 30–60 years.
- No previous diagnosis of hypertension.
- Normal blood pressure at evaluation.
- Apparently healthy individuals willing to participate.

Exclusion Criteria

Participants were excluded if they had conditions known to influence autonomic nervous system function or interfere with HRV measurements, including secondary hypertension, significant cardiovascular disease, diabetes with autonomic neuropathy, cardiac arrhythmias, thyroid disorders, chronic kidney disease, respiratory illnesses, neurological disorders, acute systemic illness, pregnancy, or use of medications known to significantly affect autonomic activity beyond standard antihypertensive therapy. Individuals with poor-quality ECG recordings unsuitable for HRV analysis were also excluded.

Clinical Assessment

Each participant underwent a detailed clinical evaluation including demographic profile, medical history, duration of hypertension, medication history, and physical examination. Height and body weight were measured using standardized techniques, and body mass index (BMI) was calculated as weight (kg)/height (m^2). Resting systolic blood pressure (SBP), diastolic blood pressure (DBP), heart rate, pulse pressure, mean arterial pressure, and rate-pressure product were recorded after adequate rest using standardized procedures.

Heart Rate Variability Recording

Heart rate variability was recorded under controlled laboratory conditions using a **10-minute resting Lead II electrocardiogram (ECG)**. Participants were instructed to avoid caffeine, nicotine, alcohol, vigorous physical activity, and heavy meals before the recording session. After an adequate period of rest in the supine position, ECG recordings were obtained in a quiet environment with comfortable room temperature to minimize external autonomic influences.

The ECG recordings were subsequently analysed using validated HRV software to derive both time-domain and frequency-domain indices according to established international recommendations.

HRV Parameters Analysed

Time-domain Parameters

The following time-domain indices were evaluated:

- Standard Deviation of Normal-to-Normal intervals (SDNN)
- Root Mean Square of Successive Differences (RMSSD)
- NN50
- pNN50

These parameters primarily reflect overall heart rate variability and parasympathetic (vagal) modulation.

Frequency-domain Parameters

Frequency-domain analysis included:

- Low Frequency (LF)
- High Frequency (HF)
- LF/HF Ratio
- Total Power (TP)

These indices were used to assess sympathetic activity, parasympathetic activity, sympathovagal balance, and overall autonomic modulation.

Outcome Measures

The **primary outcome** was comparison of HRV parameters between hypertensive and normotensive adults.

The **secondary outcome** was assessment of the relationship between duration of hypertension and HRV indices among hypertensive participants to determine whether autonomic dysfunction progressed with increasing disease duration.

Statistical Analysis

Data were entered into Microsoft Excel and analysed using appropriate statistical software. Continuous variables were expressed as **mean ± standard deviation (SD)**, whereas categorical variables were summarized as **frequency and percentage**. The independent Student's *t*-test was used to compare continuous variables between hypertensive and normotensive groups. Categorical variables were analysed using the Chi-square test. HRV parameters according to duration of hypertension were compared using appropriate comparative statistical tests. A **p-value <0.05** was considered statistically significant for all analyses.

RESULTS

A total of 128 participants were included in the study, comprising 64 normotensive and 64 hypertensive individuals. Baseline demographic characteristics, anthropometric measurements, clinical parameters, and heart rate variability (HRV) indices were compared between the two groups. Among hypertensive participants, HRV parameters were further analyzed according to the duration of hypertension.

Table 1: Baseline demographic characteristics of the study population

Variable	Normotensive (n=64)	Hypertensive (n=64)	Test statistic	p value
Age category			χ^2=5.927	0.052
30–39 years	15 (23.4)	28 (43.8)		
40–49 years	25 (39.1)	18 (28.1)		
50–60 years	24 (37.5)	18 (28.1)		
Gender			χ^2=0.130	0.719
Male	39 (60.9)	37 (57.8)		
Female	25 (39.1)	27 (42.2)		

Narrative

The age distribution was comparable between the two study groups (χ^2=5.927, p=0.052). Participants aged 30–39 years constituted a relatively larger proportion of the hypertensive group, whereas individuals aged 40–49 and 50–60 years were proportionately more common among normotensive participants. Gender distribution was also similar, with males accounting for 60.9% of the normotensive group and 57.8% of the hypertensive group (p=0.719), indicating appropriate baseline comparability between groups.

Table 2: Comparison of body mass index and clinical parameters between the study groups

Variable	Normotensive (n=64)	Hypertensive (n=64)	Test statistic	p value
BMI (kg/m²)	26.15 (23.05–28.70)	28.45 (24.95–31.40)	U=1317.500	<0.001
Age (years)	47.0 (40.5–52.0)	40.5 (36.5–50.0)	U=1652.500	0.059
Heart rate (bpm)	81.0 (75.5–90.0)	84.0 (75.0–91.0)	U=1892.000	0.457
Systolic BP (mmHg)	118.5 (113.0–124.0)	144.0 (140.0–151.5)	U=0.000	<0.001
Diastolic BP (mmHg)	77.0 (70.0–80.5)	94.0 (90.0–98.5)	U=64.000	<0.001

Values are median (IQR).

Narrative

Hypertensive participants demonstrated a significantly higher body mass index than normotensive participants (median 28.45 vs. 26.15 kg/m²; p<0.001). Median age and heart rate were comparable between the two groups (p=0.059 and p=0.457, respectively). As expected, both systolic and diastolic blood pressures were markedly elevated among hypertensive participants (both p<0.001), confirming appropriate clinical separation of the study groups.

Table 3: Comparison of time-domain heart rate variability parameters

Variable	Normotensive (n=64)	Hypertensive (n=64)	Test statistic	p value
SDRR (ms)	36.18 (19.07–55.62)	27.02 (16.82–45.10)	U=1690.500	0.088
RMSSD (ms)	27.27 (14.61–44.95)	19.55 (9.45–35.24)	U=1678.500	0.078

Narrative

Median SDRR and RMSSD values were consistently lower in hypertensive individuals than

in normotensive controls, suggesting reduced cardiac autonomic modulation. However, neither SDRR (p=0.088) nor RMSSD (p=0.078) demonstrated statistically significant differences between the groups, although both parameters showed a trend toward reduced parasympathetic activity among hypertensive participants.

Table 4: Comparison of frequency-domain heart rate variability parameters

Variable	Normotensive (n=64)	Hypertensive (n=64)	Test statistic	p value
Total Power (ms²)	1357.00 (336.70–3029.00)	661.75 (260.95–2080.50)	1694.000	0.092
VLF (ms²)	380.30 (100.09–975.50)	251.95 (114.60–571.70)	1718.500	0.116
LF (ms²)	299.75 (75.26–1198.50)	171.10 (66.37–623.98)	1774.500	0.192
HF (ms²)	333.65 (83.34–917.90)	168.60 (34.85–576.20)	1673.500	0.074
LF/HF ratio	1.02 (0.66–1.39)	1.63 (0.60–2.66)	1654.500	0.061

Narrative

All frequency-domain HRV parameters tended to be lower among hypertensive participants, whereas the LF/HF ratio was comparatively higher. Despite these trends, differences in total power, VLF, LF, HF, and LF/HF ratio did not reach statistical significance (all p>0.05). Nevertheless, the observed pattern suggested reduced overall autonomic variability with relative sympathetic predominance among individuals with hypertension.

Table 5: Heart rate variability according to duration of hypertension (n=64)

HRV Parameter	H value	p value
SDRR	6.82	0.078
RMSSD	7.94	**0.047**
Total Power	7.12	0.068
VLF	4.96	0.175
LF	5.38	0.146
HF	**8.21**	**0.042**
LF/HF ratio	6.44	0.092

Narrative

Among hypertensive participants, HRV progressively declined with increasing duration of hypertension. RMSSD (p=0.047) and HF power (p=0.042) demonstrated statistically significant reductions across duration categories, indicating progressive impairment of parasympathetic modulation with longstanding hypertension. Although SDRR, total power, VLF, LF, and LF/HF ratio also showed declining trends, these changes did not achieve statistical significance.

DISCUSSION

The present cross-sectional study compared heart rate variability (HRV) parameters between hypertensive and normotensive adults to evaluate cardiovascular autonomic function. Although hypertensive participants had significantly higher body mass index (BMI) and blood pressure than normotensive controls, baseline time-domain and frequency-domain HRV parameters were not significantly different between the two groups. However, among hypertensive individuals, longer disease duration was associated with a significant decline in RMSSD and high-frequency (HF) power, indicating progressive impairment of parasympathetic activity. These findings suggest that autonomic dysfunction may become more evident as hypertension progresses rather than during the early stages of the disease.

One of the notable findings of this study was the significantly higher BMI observed among hypertensive participants. Obesity is a well-established independent risk factor for hypertension and has been consistently associated with increased sympathetic nervous system activation, impaired baroreflex sensitivity, insulin resistance, and chronic low-grade inflammation. Excess adiposity contributes to autonomic imbalance through activation of the renin–angiotensin–aldosterone system, leptin-mediated sympathetic stimulation, and endothelial dysfunction. Consequently, hypertensive individuals with increased BMI may experience greater cardiovascular stress even before measurable alterations in conventional HRV parameters become apparent. These observations reinforce the importance of weight management as an integral component of hypertension prevention and treatment.[11]

Despite marked differences in blood pressure between study groups, baseline HRV indices did not differ significantly. Although median values of SDRR, RMSSD, total power, HF, and LF were consistently lower among hypertensive participants, statistical significance was not achieved. Similar findings have been reported in studies involving patients with recently diagnosed or well-controlled hypertension, where compensatory autonomic mechanisms and antihypertensive therapy may preserve resting autonomic function. The absence of significant differences in the present study should therefore not be interpreted as evidence of normal autonomic regulation but rather as an indication that detectable impairment may develop progressively over time.[12]

Time-domain HRV parameters provide valuable information regarding overall cardiac autonomic

modulation. SDNN (or SDRR) reflects total variability generated by sympathetic and parasympathetic influences, whereas RMSSD primarily represents vagally mediated beat-to-beat variability. Both parameters were numerically lower in hypertensive individuals in the present study, indicating reduced autonomic flexibility and diminished parasympathetic modulation. Although these reductions were not statistically significant between groups, the consistent downward trend agrees with the current understanding that hypertension is accompanied by gradual vagal withdrawal and relative sympathetic predominance.[13]

The frequency-domain analysis demonstrated a similar pattern. Hypertensive participants exhibited lower total power, low-frequency (LF), very-low-frequency (VLF), and high-frequency (HF) components, while the LF/HF ratio showed a tendency toward higher values. Total power reflects overall autonomic activity, whereas HF predominantly represents parasympathetic modulation. Reduction in HF power therefore suggests attenuation of vagal influence over sinoatrial node function. An increased LF/HF ratio, although not statistically significant in the present study, is generally considered indicative of sympathovagal imbalance. Collectively, these findings support the hypothesis that hypertension is associated with reduced autonomic adaptability, even when statistical differences are modest.[14]

The most clinically relevant observation of the present study was the significant reduction in RMSSD and HF power with increasing duration of hypertension. RMSSD is regarded as one of the most reliable markers of short-term vagal activity, while HF power reflects respiratory-mediated parasympathetic modulation. Progressive decline in both parameters strongly suggests that chronic hypertension leads to gradual deterioration of parasympathetic cardiovascular control. Persistent exposure to elevated arterial pressure may impair baroreceptor sensitivity, promote vascular remodeling, increase oxidative stress, and enhance sympathetic activation, ultimately reducing vagal responsiveness. These pathophysiological mechanisms explain why autonomic dysfunction becomes increasingly evident with longer disease duration rather than at the initial diagnosis.[15]

The relationship between disease duration and HRV observed in this study has important prognostic implications. Previous investigations have demonstrated that reduced HRV is associated with higher risks of ventricular arrhythmias, myocardial infarction, heart failure, stroke, and sudden cardiac death. Reduced vagal activity also predicts poorer cardiovascular outcomes independent of conventional risk factors such as age, blood pressure, and lipid profile. Therefore, the progressive decline in RMSSD and HF power identified among patients with longstanding hypertension may indicate increasing cardiovascular vulnerability and emphasize the need for early intervention before irreversible autonomic impairment develops.[16]

Several physiological mechanisms may explain autonomic dysfunction in hypertension. Chronic activation of the sympathetic nervous system increases circulating catecholamines, enhances peripheral vascular resistance, stimulates sodium retention, and activates the renin–angiotensin–aldosterone system. Simultaneously, reduced parasympathetic activity diminishes baroreflex-mediated inhibition of sympathetic outflow, thereby perpetuating elevated blood pressure. Structural vascular remodeling, endothelial dysfunction, oxidative stress, and chronic inflammation further impair autonomic regulation. Together, these mechanisms establish a vicious cycle in which autonomic imbalance contributes to hypertension progression while hypertension itself aggravates autonomic dysfunction.[17]

The findings of the present study are consistent with several published reports evaluating autonomic dysfunction in hypertension. Kharibam et al. demonstrated significantly lower HF power and higher LF/HF ratios among hypertensive adults, together with increased oxidative stress and poorer sleep quality, indicating pronounced sympathetic predominance and vagal withdrawal. Similarly, Shah et al. reported reduced HRV among hypertensive Indian adults and emphasized the role of autonomic dysfunction in disease progression. Although the magnitude of HRV reduction differed from that observed in the present study, both investigations support the concept that parasympathetic impairment represents an important feature of hypertension and becomes increasingly evident with chronic disease progression.

From a clinical perspective, HRV assessment offers several advantages. It is inexpensive, non-invasive, reproducible, and can be completed within a short period using routine electrocardiographic equipment. Unlike conventional blood pressure measurement, HRV provides insight into underlying autonomic regulation and may identify patients at increased cardiovascular risk before overt target-organ damage develops. Incorporating HRV into the evaluation of hypertensive patients may therefore improve risk stratification and facilitate individualized therapeutic interventions, including lifestyle modification, exercise training, weight reduction, stress management, and optimization of antihypertensive therapy.[18]

The present study possesses several strengths. Equal numbers of hypertensive and normotensive participants improved comparability between groups. Standardized ECG recording under controlled conditions minimized measurement variability, while inclusion of both time-domain and frequency-domain HRV analyses provided a comprehensive assessment of autonomic function. Furthermore, evaluation of HRV according to duration of hypertension offered additional insight

into the progression of autonomic dysfunction that has been inadequately addressed in many previous studies.[19]

Nevertheless, certain limitations should be acknowledged. The cross-sectional design precludes establishment of causal relationships between hypertension and autonomic dysfunction. The study was conducted at a single tertiary care center, which may limit generalizability to the wider population. Potential influences of antihypertensive medications, physical activity, dietary habits, psychological stress, and sleep quality on HRV could not be completely eliminated. Additionally, HRV measurements were obtained during a single resting recording rather than by 24-hour ambulatory monitoring, which may provide a more comprehensive assessment of autonomic variability. Future multicenter prospective studies with larger sample sizes and longitudinal follow-up are warranted to determine whether HRV-guided risk assessment improves long-term cardiovascular outcomes and treatment decisions.[20]

Overall, the findings of this study demonstrate that although baseline HRV parameters may not differ significantly between hypertensive and normotensive adults, progressive reduction in vagally mediated indices such as RMSSD and HF power accompanies increasing duration of hypertension. These observations reinforce the concept that autonomic dysfunction evolves gradually during the course of hypertension and highlight HRV as a valuable non-invasive tool for monitoring disease progression, identifying patients at elevated cardiovascular risk, and supporting comprehensive cardiovascular risk assessment in routine clinical practice.

CONCLUSION

The present study demonstrated that hypertensive adults had significantly higher body mass index and blood pressure than normotensive controls. Although baseline time-domain and frequency-domain heart rate variability (HRV) parameters did not differ significantly between the two groups, hypertensive participants consistently exhibited lower HRV values, suggesting reduced autonomic flexibility and relative sympathetic predominance. More importantly, RMSSD and high-frequency (HF) power declined significantly with increasing duration of hypertension, indicating progressive deterioration of parasympathetic modulation as the disease advances. These findings suggest that autonomic dysfunction becomes increasingly prominent with longstanding hypertension rather than during its early stages. HRV therefore represents a simple, non-invasive, reproducible, and clinically useful tool for evaluating cardiovascular autonomic function, identifying patients at higher cardiovascular risk, and monitoring disease progression. Incorporation of HRV assessment into routine evaluation of hypertensive patients may facilitate earlier risk stratification and support timely lifestyle and therapeutic interventions aimed at reducing long-term cardiovascular complications.

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