Original Article

Sympathovagal imbalance is enhanced by smoking in Saudi male young adults with prehypertension

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Abstract

Background and Aim: Hypertension and smoking are among the most common universal risk factors for heart attack and stroke, involving the impairment of the autonomic nervous system. Therefore, in this study, we aim to investigate the contribution of smoking to sympathovagal imbalance (SVI) in prehypertension.

Methods: Hundred and twenty-two male subjects aged 19-30 years were classified into nonsmoker normotensive (n = 38), nonsmoker prehypertensive (n = 38), smoker normotensive (n = 30), and smoker prehypertensive (n = 16). SVI was assessed from low-frequency power to high-frequency power (LF-HF ratio) and correlated with number of cigarette smoked/day in all the groups by Pearson's correlation.

Results: LF-HF ratio, LF and LF in normalized units were significantly ($P < 0.001$) increased and total power, HF and HF in normalized units were significantly ($P < 0.001$) decreased in prehypertensive subjects in comparison to normotensive subjects and these changes were more prominent in prehypertensive smokers. LF-HF ratio was positively correlated to basal heart rate, blood pressure (BP) and number of cigarette smoked/day ($P < 0.001$) in prehypertensive smokers. Smoking was found to be an independent contributing factor to SVI ($P < 0.001$) among prehypertensives.

Conclusion: In prehypertensive smokers, SVI was linked to number of cigarette smoking. Stopping smoking would enable to achieve the sympathovagal balance and BP homeostasis in prehypertensives.

Key words: Heart rate variability, prehypertension, smoking, sympathovagal imbalance

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INTRODUCTION

Cardiovascular disease is now documented as the universal leading cause of death and debility.[1] Hypertension, obesity, diabetes, dyslipidemia, poor diet, smoking, alcohol consumption, and physical inactivity are the most common universal risk factors for heart attack and stroke.[2,3]

It is well known that habitual cigarette smoking affects the respiratory and cardiovascular systems, and also also it strongly contributes to many diseases such as coronary artery disease, stroke, sudden death, and chronic obstructive pulmonary disease. The pathogenesis of some of these conditions comprises impairment of the autonomic nervous system (ANS) triggered by tobacco smoke.[4]

Heart rate variability (HRV) is a commonly used technique for noninvasive evaluation of the ANS and it may be relevant in the assessment of early preclinical alterations in the autonomic regulation in smokers. Heavy smokers have decreased HRV parameters.[5]

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Sustained sympathetic overactivity increases vasoconstrictor tone of the systemic vasculature and acts as a major mechanism for the genesis of essential hypertension.[6-8] Several studies have revealed autonomic imbalance in hypertensive patients,[9,10] but there is lack of data on the nature of autonomic imbalance that slowly leads to the progression from normotensive state into the state of prehypertension. However, prehypertension has recently been reported to be related to damage of the coronary vasculature and adverse cardiovascular events.[11-13]

A previous study based on the spectral analysis of HRV revealed that sympathovagal imbalance (SVI) in the form of sympathetic overactivity and vagal withdrawal shares in the development of prehypertension and hypertension in Indian people.[14-16] Previous studies have revealed prehypertension is more predominant among males and vagal withdrawal is more noticeable compared to sympathetic overactivity in male prehypertensives.[17,18] Studies on young prehypertensives with parental history of hypertension have reported that SVI is more strong in offspring of two parents hypertensive compared to the offspring of one parent hypertensive.[19] SVI was observed as vagal withdrawal significantly associated with sympathetic overactivity in prehypertensive subjects.[20] However, there was paucity of data on the contribution of smoking in the genesis of SVI in prehypertension among Saudi young adult population. So, in this study we aimed to investigate the individual contribution of smoking to the genesis of SVI in young adult Saudi prehypertensives.

MATERIALS AND METHODS

Study design and setting

This cross-sectional study was conducted from mid-February to mid-August 2015, involving healthy 122 male students of College of applied medical sciences in Taif University, Taif, Saudi Arabia. All the subjects experienced routine clinical examination after obtaining the approval of Research and Ethics Committee of Taif University to exclude the presence of any acute or chronic illness.

Study population

To be suitable for this study, subjects were chosen among healthy, physically fit and those aged between 19 to 30 years. Smoker subjects were chosen among those who are light smokers (smoking less than 10 cigarettes a day). Fit participants were interviewed and were clarified about their participation and the nature of investigations to be conducted in the study. In the smokers group, only subjects smoking less than 10 cigarettes a day were included in the study. Before the recordings, informed written consent was obtained from all of them. If subjects encountered any of the following conditions they were excluded from the study: (1) Subjects on antihypertensive therapy or receiving any medication, (2) subjects with acute or chronic ailments, (3) subjects performing regular sports activities because the grade of physical fitness is a topmost determining factor of vagal tone.[21-23] Subjects performing regular athletic activities and body-building trainings were also omitted from the study, (4) known cases of hypertension, myocardial infarction, heart failure, kidney disease, diabetes mellitus, or any endocrinal disorder.

Sample size

A total of 245 students were studying in different levels in this faculty during the study period. Total coverage was approved and all students were asked to participate in the study. The purpose of this study was clarified to the students and verbal and written consents were taken. The response rate was 81.63%, and a total of 200 students comprised the subjects of the study. Participants were allowed to fill the questionnaire and only 122 of them were eligible for the study.

Grouping

Subjects were classified into following four groups based on their level of systolic blood pressure (SBP) and diastolic blood pressure (DBP) as per Joint National Committee-7 classification.[24]

1. Group 1: Nonsmoker normotensive subjects (n = 38): Healthy subjects with an SBP 100–119 mmHg and a DBP 60–79 mmHg
2. Group 2: Nonsmoker prehypertensive subjects (n = 38): Healthy subjects with an SBP 120–139 mmHg or a DBP 80–89 mmHg
3. Group 3: Smoker normotensive subjects (n = 30): Healthy subjects with a SBP 100–119 mmHg and a DBP 60–79 mmHg
4. Group 4: Smoker hypertensive subjects (n = 16): Healthy subjects with a SBP 120–139 mmHg, or a DBP 80–89 mmHg.

Anthropometric measurements and blood pressure recording

After obtaining the informed consent, age, height, body weight, and body mass index (BMI) were recorded. Bllod pressure (BP) of all the subjects was recorded in the laboratory. Fully Automatic Digital Upper Arm BP Monitor (Gain Express Holdings Limited, Hong Kong, China) was used for BP recording. Cuff fits upper arm circumference 22–32 cm which was appropriate for all the subjects in the study. For BP recording, the subject was asked to sit upright with back straight on a chair with one forearm on a table and the other forearm on the side hand rest of the chair. The BP cuff was tied just tight on the arm about 2 cm above the cubital fossa. The BP cuff

was at the level of the heart. The subject rests for 5 min in the same sitting position. The "Start" button was pressed which automatically inflated and deflated the cuff. SBP, DBP, and basal heart rate (BHR) were noted from the display screen of the equipment. Then, SBP, DBP, and BHR were documented in each arm 2 times at an interval of 5 min in-between. The mean of the four recordings were considered.

Heart rate variability recording

Before the assessment of the HRV, all subjects fasted and finished their health check-up. They were instructed to stop smoking at least 2 h before the test. Routine electrocardiogram (ECG) was performed between 9 and 11 AM in the Physiology Laboratory, College of Applied Medical Sciences, Taif, KSA. For recording of HRV, ECG electrodes (AD Instruments) were connected and Lead II ECG was acquired at a rate of 1000 samples/s during supine rest under standardized conditions.[23,25] An artifact-free 5-min segment of the ECG was analyzed offline using LabChart software that permits visual inspection of the raw ECG to obtain the HRV parameters in time-domain and frequency-domain. The recorded ECG signals were transported through analog digital converter FE132 Bio Amp (using Power Lab, 8/35 model PL3508, 8 channel data acquisition system, ADInstruments, Australia) with a sampling rate of 20 Hz. The following parameters were derived from the RR data.

Time-domain analysis

This comprises of comparing two different signals and the data were analyzed using descriptive statistical methods. Fluctuations of the HR were measured by various variables including:[23,25]

a. Standard deviation of all normal to normal RR intervals sensitive to all sources of variation (SDNN)
b. Standard deviation of the averages of NN intervals in all 5 min segments of the entire recording (SDANN)
c. Root mean square successive difference of RR intervals (RMSSD).

Frequency-domain analysis

Fast Fourier technique (FFT), is a nonparametric technique that was performed for frequency-domain parameters. The different components of FFT and their specific frequency ranges[23,25] were:

a. Total power (TP) (0–0.4 Hz) which indicates sympathetic and parasympathetic tone
b. High-frequency (HF) (0.15–0.4 Hz) which is revealing parasympathetic tone and respiration
c. Low-frequency (LF) (0.04–0.15 Hz) which specifies sympathetic as well as parasympathetic tone
d. Very LF (VLF) (0.003–0.04 Hz) which points thermoregulation, and can be used to compute LF normalized unit (LFnu) and HF normalized unit (HFnu) that symbolizes the relative value of each component in proportion to the TP minus the VLF component
e. LF/HF reveals the sympathovagal balance and sympathetic modulation.

To reduce the impact of diurnal and environmental disparities, the HRV measurements were caught in the subjects in a sitting position after 20 min rest. The recordings were taken in the morning at the same room by one qualified research assistant in accordance with a standardized technique. The HRV recordings were taken twice for each subject with a short-term interval in-between. Premature beats (i.e. >20% shortening) were omitted manually and substituted with interpolated values and represented <1% of each participants' collection. The same duration (5 min) of data were analyzed as proven by the task force system. Measurements with nonsinus beats that were >1% of the total number of beats were also omitted. Premature beats and artifacts were carefully eradicated automatically and manually by visual checkup of all RR intervals.[25]

Statistical analysis of data

SPSS version 22 (SPSS Software Inc., Chicago, IL, USA) was used for statistical analysis of data. All the data were expressed as mean ± standard deviation. Kolmogorov–Smirnov test was used to determine normality of data. Analysis of variance (ANOVA) of data within the four groups was done by one-way ANOVA. The association between LF-HF ratio and studied parameters was assessed by Pearson correlation analysis. Multiple regression analysis was applied to weigh the independent contribution of various factors such as age, BMI, SBP and DBP to SVI (LF-HF ratio). The statistical significance was considered with $P < 0.05$.

RESULTS

The general characteristics of the study population are depicted in Table 1. There were no significant ($P > 0.05$) differences between the four groups regarding age, weight, and BMI. Moreover, SBP, DBP, and BHR were significantly ($P < 0.05$) higher in Group 4 compared to that in Group 1 and 3, also, BHR in Group 4 was higher than in Group 2.

Table 2 describes the frequency and time domain indices of the HRV of participants in various groups recorded in the supine position. Regarding time domain indices of HRV, all indices were significantly ($P < 0.05$) decreased in Group 2, 3, and 4 as compared to Group 1, also they were decreased in Group 3 and 4 when compared to Group 2, but there were no significant ($P > 0.05$) differences between Group 3 and 4 except for mean NN. SDNN, SDDNN, and RMSSD were significantly ($P < 0.05$) decreased in Group 2, 3, and 4

as compared to Group 1. By comparing the decrease in these time domain indices in Group 4 with Group 3, there was no significant ($P > 0.05$) difference. Moreover, the decrease in Group 4 was significantly ($P < 0.05$) more than in Group 2.

Regarding frequency domain indices of HRV, they were significantly ($P < 0.05$) different between the groups except for the VLF. Moreover, TP, HF, and HF nu were significantly ($P < 0.05$) decreased in Group 2, 3, and 4 when compared to Group 1, also they were lower in Group 3 and 4 when compared to Group 2 and obviously decreased in Group 4 when compared to Group 3. Moreover, LF, and LF/HF ratio and LFnu showed higher levels in Group 2, 3, and 4 when compared to Group 1, also they were significantly ($P < 0.05$) higher in Group 3 and 4 when compared to Group 2 and markedly increased in Group 4 when compared to Group 3. Table 3 depicts Pearson correlation of LF-HF ratio with age, BHR, BMI, BP and number of cigarette smoking/day of subjects of various groups. LF-HF ratio was not correlated to any of the studied variables in Group 1. Moreover, in Group 2, it was positively correlated to SBP, in Group 3, it was positively correlated to BHR, whereas in Group 4, it was positively correlated with BHR, BP and number of cigarette smoking/day. Table 4 depicts multiple regression analysis of LF-HF ratio (as dependable variable) and various other associated factors (as independent variables) in the entire normotensive group ($n = 68$). Number of cigarette smoking/day contributes to LF-HF ratio (as dependable variable) by 83.6% ($R^2 = 0.836$, $F = 51.705$, $P = 0.000$).

Table 5 demonstrates multiple regression analysis of LF-HF ratio (as dependable variable) and various other associated factors (as independent variables) in the whole prehypertensive group ($n = 54$). Number of cigarette smoking/day and HR contribute to LF-HF ratio (as dependable variable) by 94.8% ($R^2 = 0.948$, $F = 143.695$, $P = 0.000$).

Table 1: General characteristics of the study groups

	Group 1 (*n*=38)	Group 2 (*n*=38)	Group 3 (*n*=30)	Group 4 (*n*=16)	*P*
Age	22.9 (2.9)	23.8 (3.8)	24.1 (3.4)	24.5 (3.6)	0.203
Weight	72.9 (14.4)	76.4 (15.3)	77.1 (13.3)	74.1 (12.6)	0.588
BMI	24.1 (4.1)	25.6 (4.3)	26.1 (4. 7)	24.9 (3.4)	0.226
SBP	107.5 (9.7)	129.3 (3.4)[a]	107.5 (7.7)[b]	131.3 (4.5)[a,c]	0.000
DBP	65.85.7)	83.8 (1.8)[a]	67.5 (5.9)[b]	84.5 (2.4)[a,c]	0.000
BHR	73.2 (3.9)	81.9 (2.1)[a]	89.5 (1.9)[a,b]	102.1 (7.9)[a,b,c]	0.000

Data presented are mean (SD), Group 1: Nonsmoker normotensive subjects, Group 2: Nonsmoker prehypertensive subjects, Group 3: Smoker normotensive subjects, Group 4: Smoker hypertensive subjects. [a]Comparison with Group 1, [b]Comparison with Group 2, [c]Comparison with Group 3. *P* is significant at the 0.05 level. SBP: Systolic blood pressure, DBP: Diastolic blood pressure, BHR: Basal heart rate, BMI: Body mass index, SD: Standard deviation. *P*>0.05 was considered significant

DISCUSSION

In this study, we aimed to examine the contribution of smoking in the genesis of SVI in young adult Saudi prehypertensives. TP, in general, reflects the vagal potency of cardiac modulation.[23,25] In this study, a significant decrease in TP of HRV spectrum in prehypertensive population (Groups 2 and 4) compared to that of the normotensive population (Group 1 and 3) represents a significant decrease in HRV, which specifies decreased power of vagal drive in these persons. This was supported by a decrease in absolute HF power and HFnu in prehypertensive participants, since HF and HFnu are the indices of parasympathetic drive to the heart.[23,25] Furthermore, TP, HF, and HFnu were significantly low in

Table 2: Frequency and time domain indices of heart rate variability recorded in supine position of subjects in various groups

	Group 1 (*n*=38)	Group 2 (*n*=38)	Group 3 (*n*=30)	Group 4 (*n*=16)	*P*
Time domain analysis					
Mean NN	816.6 (57.0)	736.5 (17.8)[a]	672.4 (15.4)[a,b]	591.1 (45.6)[a,b,c]	0.000
SDNN	86.8 (38.5)	52.7 (3.4)[a]	40.3 (4.4)[a,b]	27.3 (6.0)[a,b]	0.000
SDDNN	86.7 (48.9)	40.1 (12.6)[a]	25.5 (10.6)[a,b]	14.8 (4.7)[a,b]	0.000
RMSSD	91.1 (44.7)	38.9 (6.5)[a]	22.4 (4.0)[a,b]	12.6 (2.9)[a,b]	0.000
Frequency domain analysis					
TP	8515.9 (8743.2)	2511.5 (553.5)[a]	1317.8 (230.1)[a,b]	545.3 (308.7)[a,b,c]	0.000
LF	373.1 (158.1)	786.1 (139.4)[a]	1303.8 (281.4)[a,b]	3859.3 (2181.7)[a,b,c]	0.000
HF	2452.9 (2691.5)	394.8 (141.3)[a]	142.0 (41.3)[a,b]	34.6 (15.7)[a,b,c]	0.000
LF/HF ratio	1.04 (0.3)	2.2 (0.36)[a]	4.2 (0.8)[a,b]	6.4 (0.7)[a,b,c]	0.000
VLF	1014.3 (1046.8)	963.2 (766.3)	1131.9 (2192.2)	701.7 (514.0)	0.768
LF nu	36.7 (10.8)	58.7 (5.5)[a]	77.9 (4.6)[a,b]	89.3 (3.8)[a,b,c]	0.000
HF nu	39.1 (6.8)	27.3 (2.7)[a]	17.0 (2.6)[a,b]	8.6 (3.4)[a,b,c]	0.000

Data presented are mean (SD), *P* is significant at the 0.05 level. Group 1: Nonsmoker normotensive subjects, Group 2: Nonsmoker prehypertensive subjects, Group 3: Smoker normotensive subjects, Group 4: Smoker hypertensive subjects.[a]Comparison with Group 1, [b]Comparison with Group 2, [c]Comparison with Group 3. *P* are probabilities for the difference between the subgroups in ANOVA. *P*<0.05 is considered statistically significant. Mean NN: Mean NN interval, SDNN: Standard deviation of normal to normal interval, SDANN: Standard deviation of the averages of NN intervals in all 5 min segments of the entire recording, RMSSD: Root mean square successive difference of RR intervals, LF: Low frequency, HF: High frequency, LF-HF ratio: Low frequency power to high frequency power, HR: Heart rate, TP: Total power of HRV, VLF: Very low frequency power, LFnu: Low frequency power normalized, HFnu: High frequency power normalized, HRV: Heart rate variability, SD: Standard deviation

Table 3: Pearson correlation of low-frequency power to high-frequency power ratio with age, basal heart rate, body mass index, blood pressure, and number of cigarette smoking/day of subjects of various groups

	Group 1 (*n*=38)		Group 2 (*n*=38)		Group 3 (*n*=30)		Group 4 (*n*=16)	
	r	*P*	*r*	*P*	*r*	*P*	*r*	*P*
Age	0.050	0.768	−0.013	0.938	0.113	0.551	0.080	0.768
HR	0.086	0.607	0.257	0.230	0.418	0.048*	0.796**	0.000
BMI	0.179	0.282	0.016	0.926	−0.012	0.949	0.343	0.193
SBP	0.135	0.418	0.374*	0.021	0.145	0.445	0.556*	0.025
DBP	0.110	0.511	0.055	0.743	0.262	0.162	0.500*	0.049
Cigarette/day	-	-	-	-	0.069	0.716	0.905**	0.000

Group 1: Nonsmoker normotensive subjects, Group 2: Nonsmoker prehypertensive subjects, Group 3: Smoker normotensive subjects, Group 4: Smoker hypertensive subjects. *Correlation is significant at the 0.05 level (two-tailed), **Correlation is significant at the 0.01 level (two-tailed). HR: Heart rate, BMI: Body mass index, SBP: Systolic blood pressure, DBP: Diastolic blood pressure

Table 4: Multiple regression analysis of low-frequency power to high-frequency power ratio (as dependable variable) with various other associated factors (as independent variables) in the entire normotensive group (*n*=68)

Model	Unstandardized coefficients		Standardized coefficients	*t*	Significant
	B	SE	*β*		
Constant	−2.966	3.049		−0.973	0.335
Age	0.004	0.030	0.007	0.124	0.901
HR	0.034	0.030	0.177	1.159	0.251
BMI	0.012	0.021	0.032	0.571	0.570
SBP	0.002	0.010	0.011	0.203	0.840
DBP	0.015	0.017	0.051	0.882	0.381
Cigarette/day	0.107	0.023	0.729	4.671	0.000

Dependent variable: LF/HF ratio, R^2=0.836, F=51.705, P=0.000. HR: Heart rate, BMI: Body mass index, SBP: Systolic blood pressure, DBP: Diastolic blood pressure, SE: Standard error, LF-HF ratio: Low-frequency power to high-frequency power. P>0.05 was considered significant

Table 5: Multiple regression analysis of low-frequency power to high-frequency power ratio (as dependable variable) with various other associated factors (as independent variables) in the entire prehypertensive group (*n*=54)

Model	Unstandardized coefficients		Standardized coefficients	*t*	Significant
	B	SE	*β*		
Constant	0.071	3.586		0.020	0.984
Age	0.008	0.020	0.015	0.408	0.685
HR	0.080	0.024	0.417	3.260	0.002
BMI	0.002	0.018	0.005	0.132	0.896
SBP	−0.003	0.019	−0.006	−0.167	0.868
DBP	−0.049	0.035	−0.051	−1.425	0.161
Cigarette/day	0.077	0.016	0.583	4.743	0.000

Dependent variable: LF-HF ratio, R^2=0.948, F=143.695, P=0.000. HR: Heart rate, BMI: Body mass index, SBP: Systolic blood pressure, DBP: Diastolic blood pressure, SE: Standard error, LF-HF ratio: Low-frequency power to high-frequency power. P>0.05 was considered significant

smoker prehypertensive subjects (Group 4) compared to the nonsmoker prehypertensive subjects (Group 2) [Table 2] indicating the more decrease in vagal motivation in smoker prehypertensive subjects. In this study, we have observed an increased absolute LF power and LFnu in prehypertensives in Group (2, 4) compared to their normotensive colleagues in Groups (1, 3) [Table 2]. Thus, these findings confirm the increased state of sympathetic drive in prehypertensive subjects since LF signifies sympathetic modulation. LF-HF ratio is a sensitive index of sympathovagal balance. Increase in this ratio indicates increased sympathetic activity.[23,25] LF-HF ratio was significantly increased (P < 0.001) in prehypertensive subjects (Groups 2 and 4) as compared to their normotensive colleagues (Group 1 and 3), which indicates an intensified sympathetic discharge in prehypertensives. In this study, we found a big difference in LF-HF ratio between normotensive and prehypertensive groups, which was not detected in some studies.[26-28] The results from our study show that at rest, smokers have altered HRV in the form of increased LF/HF ratio and LFnu where the means of both of them were significantly higher among smokers than nonsmokers which is inaccordance with the results of Taralov *et al*.[29] Results from our research are supported by other studies who have established significant decrease in HRV with increased LF/HF ratio in heavy smokers with >15 pack-years.[30,31] Despite the fact that we have measured HRV in younger patients with less pack-years, the same pattern of autonomic function alteration is present. Evidence for sympathetic predominance assessed by LF/HF ratio could be explained by the weakened baroreflex in smokers which normally decreases the sympathetic motivation.[32,33] Therefore, measuring HRV could be a very useful screening test for distinguishing ANS alterations in smokers long before the clinical signs appear. The present study confirms the presence of decreased parameters of the three measures of time-domain analysis (SDNN, SDANN, and RMSSD) in Group 4 as compared with Group 2 and in Group 3 as compared to Group 1. These findings are consistent with the results of the recent study of Taralov *et al*.[29] who revealed a decrease in overall variability, vagal withdrawal, and sympathetic predominance in smokers. Manzano *et al*.[34] have found that smoking cigarette leads to acute modifications of the autonomic control associated

with sympathetic activation and vagal withdrawal. These changes are present for 30 min after smoking, so, we tried to eradicate the acute effect of smoking by educating the subjects to cease smoking at least 2 h before the test. HRV has been used as a noninvasive means to quantitatively appraise the cardiac autonomic activity, and it is proved to be of prognostic significance in hypertension.[26-28] HRV has been reported to be decreased in hypertension, and the magnitude of the decrease in HRV predicts the severity of hypertension.[26] HRV also has emerged as a cardiovascular risk marker.[35] Thus, decreased HRV in prehypertensives in this study reinforces the fact that HRV could be used as a predictive means for the future occurrence of hypertension in these people. In smoker prehypertensive, there was a significant decrease in the mean NN in Group 4 subjects compared to that of both Group 1, 2, and 3 subjects. This indicates a decrease in vagal tone in smoker prehypertensive subjects, since the mean NN, in general, reflect vagal modulation of cardiac activities.[23,25] Furthermore, the BHR was significantly more in smoker prehypertensive subjects (Group 4) compared to subjects of (Groups 1, 2, and 3). This indicates a significantly lower vagal tone in smoker prehypertensive because higher BHR is an index of poor vagal tone.[36] Thus, these findings suggest that vagal withdrawal plays an important role in the alteration of sympathovagal balance in prehypertensive smoker subjects. It was assumed that vagal withdrawal could also be important in the causation of prehypertension in adults. From this study, smoking could be a potential factor for the causativeness of SVI, as it was highly correlated with LF-HF ratio in prehypertensive subjects [Table 5]. In addition, smoking has emerged as an important contributor to SVI in the prehypertensives (Group 2 and 4) because it was found to have an independent correlation with LF-HF ratio determined by multiple regression analysis.

In this study, LF-HF ratio in smoker normotensive was positively correlated to BHR, while in smoker prehypertensive, it was positively correlated to BHR, SBP, DBP, and number of cigarette smoking/day. DBP is the reflection of peripheral vascular tone and resistance.[36] In this study, the degree of correlation of LF-HF ratio was maximum with DBP in Groups 4 [Table 3]. Hence, alteration in vascular tone could be directly linked to the degree and nature of SVI, and it appears that smoking is an independent contributor to the genesis of SVI in these subjects. In the entire normotensive group [Table 4], multiple regression analysis shows that number of cigarette smoking/day contributes to the change in LF-HF ratio by 83.6%. Moreover, in the entire prehypertensive group, multiple regression analysis demonstrates that the number of cigarette smoking/day and BHR contribute to LF-HF ratio by 94.8%.

This study emphasizes the necessity to improve vagal tone in subjects with prehypertension so as to restore the sympathovagal balance in these subjects and prevent their progression to the stage of clinical hypertension.

Limitations of the study

Moderate sample size achieved in this study for examining a highly variable data such as HRV could limit the power of statistical results. Furthermore, conventional function tests such as HR and BP response to standing, deep breathing, etc., could have added information on the nature of SVI in these subjects.

CONCLUSION

In this study, SVI was observed in smoker prehypertensive subjects. Smoking was found to be significantly associated with sympathetic overactivity in smoker subjects. The magnitude of SVI was correlated with BHR and smoking, and they were considered as independent contributing factors for LF-HF ratio. Hence, it was advised that lifestyle adjustments should be assumed by smokers for attaining their effective autonomic balance.

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Conflicts of interest

There are no conflicts of interest.

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