ORIGINAL ARTICLE

EFFECT OF HYPERTENSION AND SMOKING ON HEART RATE VARIABILITY IN PATIENTS WITH MYOCARDIAL INFARCTION

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Background: Patients afflicted with myocardial infarction and hypertension are at increased risk of developing arrhythmias due to autonomic imbalance. Heart rate variability, a non invasive analytical measure, can be employed for evaluating the autonomic nervous system. Cigarette smoking increases the possibility of acute cardiac events associated with augmented sympathetic drive and endothelial dysfunction. Autonomic dysfunction is a known symptom of impaired cardiac activity and is robustly related with amplified overall mortality risk. This study aimed to identify heart rate variability (HRV) indices in hypertensive and non hypertensive individuals with acute myocardial infarction (AMI) and the effect of smoking on HRV of patients with AMI. **Methods:** Fifty patients with AMI underwent 24 hours holter monitoring. Time and frequency domain measures of HRV were analysed including SDNN, SDNNi, SDANN, rMSSD, pNN50, power, VLF, LF, and HF. **Results:** The time domain indices SDNN, SDANN, SDNNi, RMSSD and pNN50 were decreased in hypertensive patients when evaluated against non hypertensive patients with AMI. Frequency domain indices; The Total power, VLF, LF and HF amongst hypertensive and non hypertensive patients showed decreased HRV in hypertensive patients with AMI. SDNN, SDANN, SDNNi, total power, VLF and LF were decreased in smokers suffering AMI as compared to non smokers with acute myocardial infarction ($p<0.05$). **Conclusions:** Heart rate variability is diminished in hypertensive individuals after AMI. It reflects autonomic imbalance in individuals with co-existent hypertension and AMI. The study also suggested a noteworthy attenuation of time and frequency domain indices in the study population with smoking habits.

Keywords: Autonomic nervous system, myocardial infarction, ambulatory electrocardiography

INTRODUCTION

Enhanced sympathetic activity transpires in hypertension (HTN). Coexistent acute myocardial infarction (AMI) and hypertension are correlated with augmented cardiovascular risk.[1] There is definite cardiovascular autonomic dysfunction in hypertensive patients as compared to the normotensive subjects.[2] Studies have testified diminished HRV among hypertensives. Statistics from the Framingham and Atherosclerosis Risk in Communities (ARIC) cohorts propose that people with reduced HRV are at a higher risk of developing hypertension[3]. Another study estimated the prognostic significance of HRV after AMI when compared with established risk factors like hypertension.[4] Cigarette smoking is responsible for manifesting coronary artery disease. Numerous research studies have verified that smokers have a favourable prognosis in comparison with non-smokers with MI if they abandon smoking.[5]

Research work has been done but there is paucity of data regarding evaluation of all the time and frequency domain indices of hypertensive and normotensive patients with acute myocardial infarction within 24 hours of their symptoms. Unlike most of the studies, in our study the hypertensive and normotensive patients with AMI were holter monitored for 24 hours. Likewise the effect of smoking on both time and frequency domain parameters of heart rate variability in our population has not been studied previously. Since heart rate variability is a measure of the autonomic status therefore the study focused on evaluating the effect of hypertension and smoking in patients with acute myocardial infarction.

The purpose of our study was to identify heart rate variability (HRV) indices in hypertensive and non hypertensive individuals with acute myocardial infarction (AMI). We also identified the effect of smoking on the time and frequency domain measures of HRV in patients with AMI.

MATERIAL AND METHODS

The study was conducted after approval by Medical Ethics Committee of Army Medical College and Institutional Review Board of Armed Forces Institute of Cardiology (AFIC/NIHD), Pakistan. Written informed consent was acquired from the individuals under study. 24 hours holter monitoring was done in a total of 59 patients. Fifty subjects diagnosed with acute myocardial infarction by a cardiologist were studied. Infarction greater than 2 days old, Patients with a previous episode of myocardial infarction, patients indisposed or incapable of giving written informed consent were excluded from the study population.

Artefacts in the ECG recording, decreased duration of holter monitoring and malfunction due to some technical problem resulted in the exclusion of 9 patients in the study group. HRV was analysed in the remaining 50 patients with acute myocardial infarction. Twelve lead standard ECG (Motara ELI 250 EKG Machine; Absolute Medical Equipment, New York) was performed on all the patients included in study population. Recording of Ten cardiac cycles was done to assess the heart rhythm. 'DMS 300-3A Serials Holter Recorder' and 'DMS Serials Holter Software Premier 11' (Diagnostic monitoring software, Kingsbury, UK) were employed for HRV analysis. Visual checks were employed for editing artefacts in the recordings.

Time and frequency domain indices of HRV were analysed according to the recommendations of Task Force of the European Society of Cardiology (ESC) and the North American Society of Pacing and Electrophysiology (NASPE).[6] SDNN (standard deviation of R-R intervals), SDNNi (SDNN index; a measures of variability due to cycles shorter than 5 minutes), SDANN (standard deviation of average NN intervals), pNN50 (the number of interval differences of successive NN intervals greater than 50 ms (NN50)/total number of NN intervals) and RMSSD (square root of the mean squared differences of successive NN intervals) were analysed. Likewise total power, LF (low frequency), VLF (very low frequency) and HF (high frequency) parameters of frequency domain were analysed. Variations in all the time and frequency domain parameters of HRV were evaluated that depicts autonomic status of the individual.

Data was analysed using SPSS-15 and Microsoft Excel 2007. HRV indices were expressed as Mean±SD. Hypertensive and non-hypertensive patients as well as smokers and non-smokers suffering acute myocardial infarction were compared by using Student's *t*-test and $p<0.05$ was considered significant.

RESULTS

Table-1 shows a significant decrease in time domain indices of HRV in hypertensive patients after acute MI as compared non hypertensive patients with AMI. The value of SDNN in hypertensive patients with AMI was decreased as compared to the value in non hypertensive patients (58±17 ms vs. 92±30 ms; $p<0.05$), SDANN was also attenuated in infarcted patients with hypertension when compared with non-hypertensive patients (49±16 ms vs. 81±29 ms). Likewise SDNNi decreased in hypertensive study group as compared to non hypertensive study group (28±11 ms vs. 41±14 ms; $p<0.05$), RMSSD and pNN50 were also decreased in hypertensive infarcted patients when compared with non-hypertensive infarcted patients but the difference was not significant statistically.

Table-1 also shows the frequency domain indices of HRV in hypertensive patients with AMI and non hypertensive patients with AMI. It shows that total power, VLF, LF and HF has distinctly declined in hypertensive patients with AMI when compared with non hypertensive patients with AMI. The decrease in frequency domain parameters in MI patients with history of hypertension was found statistically significant ($p<0.05$) by student's *t*-test.

Table-1: Time and frequency domain parameters of HRV in hypertensive and non-hypertensive patients with acute myocardial infarction

PARAMETERS	Hypertensive (Mean±SD)	Non-hypertensive (Mean±SD)	*p*
Time Domain Parameters			
SDNN (ms)	58±17	92±30	0.0006
SDANN (ms)	49±16	81±29	0.00006
SDNNi (ms)	28±11	41±14	0.002
RMSSD (ms)	27±16	29±9	0.6
pNN50%	7±15	6±4	0.95
Frequency Domain Parameters			
Total Power (ms^2)	815±73	1756±13	0.006
LF (ms^2)	546±64	1242±10	0.01
VLF (ms^2)	123±10	363±29	0.001
HF (ms^2)	75±9	124±8	0.08

SDNN=Standard deviation of normal to normal intervals, SDANN=Standard deviation of averages of the normal to normal intervals, SDNNi=Mean of standard deviation of 5 minute segment of entire 24 hour recording, RMSSD=Square root of mean of the sum of squares of differences between successive normal to normal intervals, pNN50=Number of NN intervals greater than 50 ms/total number of NN intervals, VLF=Very low frequency, LF=Low frequency, HF=High frequency

Table-2 depicts the time and frequency domain indices of HRV in smokers with acute myocardial infarction and non-smokers with AMI. It shows that all the time and frequency domain indices are attenuated in smokers with AMI when compared with non smokers with AMI. The differences in SDNN, SDANN, SDNNi, total power, LF and VLF was found statistically significant ($p<0.05$) by Student's *t*-test.

Table-2: Time and frequency domain parameters of HRV in smokers and non-smokers with acute myocardial infarction

PARAMETERS	Smokers (Mean±SD)	Non Smokers (Mean±SD)	*p*
Time Domain Parameters			
SDNN (ms)	61±19	89±31	0.001
SDANN (ms)	52±21	78±29	0.001
SDNNi (ms)	30±11	40±15	0.017
RMSSD (ms)	27±16	30±9	0.57
pNN50 %	7±15	6±4	0.66
Frequency Domain Parameters			
Total Power (ms^2)	809±73	1763±13	0.005
LF (ms^2)	507±56	1279±10	0.004
VLF (ms^2)	146±15	341±28	0.007
HF (ms^2)	82±10	117±8	0.2

SDNN=Standard deviation of normal to normal intervals, SDANN=Standard deviation of averages of the normal to normal intervals, SDNNi=Mean of standard deviation of 5 minute segment of entire 24 hour recording, RMSSD=Square root of mean of the sum of squares of differences between successive normal to normal intervals, pNN50=Number of NN intervals greater than 50 ms/total number of NN intervals, VLF=Very low frequency, LF=Low frequency, HF=High frequency

DISCUSSION

Research shows that individuals with attenuated HRV at baseline are at a greater risk of developing hypertension when examined in a follow-up study over a span of nine years, thus signifying that hypertension is often preceded by low HRV.[7] Mainly the research work has been done on frequency-domain indices, with very few reporting on rMSSD or SDNN.[8–11] We have come across only two studies that have analysed the relationship between HRV and hypertension. The Framingham study gives an account of a collective incidence of 4-year hypertension supported by 2-hour HRV recording and established a relationship between low frequency power among men only and no correlation with SDNN and HF in either sex.[12] In ARIC study, Liao *et al* testified an increased 3-year prevalence of hypertension associated with decreased HF and SDNN showing no association with LF.[12] The studies on HRV and hypertension suggest that sympathovagal dysregulation proclaim the development of hypertension. This research elucidation is also supported by the research work of Shroeder[7], Reaven *et al*[13], Palatini and Julius[14] as well as Julius and Nesbitt[15]. They have emphasised the increase in the sympathetic activity due to hypertension. Julius *et al* explained both sympathetic hyperactivity and vagal withdrawal in hypertensive patients.

Another study aimed at assessing the effect of hypertension on HRV in individuals with Q-wave myocardial infarction (Q-MI) on fifth post admission day. HRV was found appreciably attenuated in hypertensive patients.[16] Quintana and colleagues performed ambulatory electrocardiography for 24 hours post admission and showed that patients afflicted with non-fatal infarction and cardiac events had a decline in HRV than those without. Multivariate regression analysis depicted that diminished heart rate variability independently predicted mortality or non-fatal infarction.[17]

Therefore in our study the hypertensive and normotensive patients with AMI were holter monitored for 24 hours with 24 hours of admission. Both the time and frequency domain parameters were analysed which showed a significant decrease in HRV in infarcted hypertensive patients depicting the sympathovagal dysregulation.

Research work has been done to analyse the acute effect of smoking on HRV. In one study fifteen subjects were analysed for time and frequency domain measures of HRV for each 5-minute segment: The SDNN, mean R-R interval and RMSSD values significantly decreased. It was revealed that acute cigarette smoking modifies HRV measures, especially within the initial 5 to 10 minutes post smoking.[18] Likewise in another research twenty four chronic heavy smokers (men) and twenty-two non-smoking individuals (hospital employees) were taken as study population. Time domain (RR, SDNN, RMSSD) and frequency domain (HF, LF, LF/HF) indices of HRV were recorded from the study population after 15 minutes resting period, exercise and during controlled respiration over a span of 5-minute. It was found that SDNN and RMSSD values were attenuated in smokers in comparison with non-smokers. R-R interval, HF, and LF did not show a radical variation.[19] Alyan *et al* also showed that SDNN, SDANN, RMSSD and HF values were attenuated in smokers when compared with non-smokers. LF and LF/HF ratio were appreciably greater in smokers as compared to non-smokers.[20]

Another research revealed that LF and LF/HF ratio were remarkably augmented whereas SDNN, RMSSD, SDANN, HF were notably declined in heavy smokers after 24-hour Holter recordings.[21]

Andrikopoulos *et al* established that the values of total power, LF, and HF components are significantly attenuated. All the time domain parameters were appreciably lower during smoking.[22] In our study the smokers and non smokers after AMI underwent holter monitoring for 24 hours within 24 hours of their admission and a significant decrease was identified in both time and frequency domain measures that shows an imbalance in sympathovagal activity. Larger studies may assist in predicting mortality of decreased HRV.

CONCLUSION

Patients with myocardial infarction and hypertension portray increased expression of sympathetic activity and depressed vagal activity according to values of HRV when compared with non hypertensives. Smokers with myocardial infarction also suffer sympathovagal imbalance depicted by decreased HRV.

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