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COMPARISON OF SIGNAL AVERAGED ECG PARAMETERS IN PATIENTS WITH AND WITHOUT LEFT VENTRICULAR HYPERTROPHY

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ABSTRACT

Objective: Comparison of signal averaged ECG parameters in patients with and without left ventricular hypertrophy.

Study Design: Cohort retrospective study.

Place and Duration of Study: Department of Cardiac Electrophysiology, Armed Forces Institute of Cardiology, Rawalpindi from 11th November, 2014 to 10th November, 2015.

Material and Methods: Sixty-four patients with systemic arterial hypertension were divided into two equal groups on the basis of left ventricular hypertrophy. Patients with acute or old myocardial infarction, diabetes mellitus, cerebrovascular accident, heart failure, structural heart disease, bundle branch block and cardiomyopathies were excluded from the study. DMS 300 4L Holter monitors were used to obtain 3 channel signal averaged ECG recording. cardio scan premium luxury software was used for analysis of signal averaged ECG.

Results: There were 49 (76.6%) males and 15 females (23.4%) with the mean age of 60 ± 11.83 years. The mean values for filtered QRS complex, low amplitude signals and root mean square voltage in patients with and without left ventricular hypertrophy were 118.1 and 98.9 ms, 35.4 and 22.2 ms, 89.9 and 94.4 μv respectively. The mean values of filtered QRS complex and low amplitude signal were significantly higher in patients with left ventricular hypertrophy as compared to those without the hypertrophy. Whereas, difference between the mean values of root mean square voltage were statistically insignificant.

Conclusion: Signal averaged ECG parameters are significantly deranged in hypertensive patients with left ventricular hypertrophy as compared to those without the hypertrophy.

Keywords: Heart ratventricular late potentials, Left ventricular hypertrophy, Systemic arterial hypertension, , Signal averaged ECG.

INTRODUCTION

The term "signal averaged electrocardiography" incorporates any technique in which multiple electric signals from the heart are averaged to improve the signal to noise ratio in order to reveal ventricular late potentials[1]. Three bipolar orthogonal leads, XYZ are used which represent horizontal, sagittal and coronal planes respectively[2]. The leads are recorded, averaged, filtered and combined into a vector magnitude called the filtered QRS complex. Filtered QRS complex is analyzed for the presence or otherwise of ventricular late potentials[3].

Ventricular late potentials are noninvasive electrocardiographic parameters which can be used to identify hypertensive patients with increased risk of developing ventricular arrhythmias[4]. They are low amplitude, high frequency signals present in the terminal part of QRS complex that may extend up to a variable length in ST segment[5]. Ventricular late potentials, being extremely small signals, are detected by signal averaged ECG. They are noninvasive markers of myocardial tissue damage[6]. Left ventricular hypertrophy results in myocardial fibrosis which through gap junctions and ion channel remodelling

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provokes significant electrophysiological changes which lead to delayed conduction velocity[7]. This affects the electrocardiographic signals between the end of QRS complex and the initial part of ST segment thus generating these low voltage fractionated signals[8]. Detection of ventricular late potentials in patients with left ventricular hypertrophy provides a practical and cost effective method to identify the possible electrophysiological substrate underlying the life threatening ventricular arrhythmias which may result in sudden cardiac death[9].

Hypertension is a major health problem with an increasing prevalence worldwide. It is considered a silent killer because of its symptomless proceedings during pathogenesis[1]. It is a robust risk factor for left ventricular hypertrophy, a compensatory mechanism in response to increased pressure load on the heart[10,11]. Left ventricular hypertrophy is a strong predictor of ventricular arrhythmias which may lead to sudden cardiac death[5].

Knowledge about arrhythmias developing in patients with hypertension and left ventricular hypertrophy is important because it can significantly affect the prognosis and management of the disease[9]. Pathophysiological mechanisms underlying the development of left ventricular hypertrophy involve systolic and diastolic pressure overload along with neurohormonal activation[12]. Left ventricular hypertrophy results in myocardial fibrosis which through gap junctions and ion channel remodelling provokes significant electrophysiological changes which lead to delayed conduction velocity. This provides an ideal substrate for reentry which may lead to ventricular arrhythmias[13].

The current study was planned to compare signal averaged ECG parameters in hypertensive patients with and without left ventricular hypertrophy. The study will provide knowledge whether derangements in signal averaged ECG parameters are associated with left ventricular hypertrophy. Results of the study would provide an insight into the probable mechanisms of disturbed electrical activity within ventricular myocardium in these patients. This may also help to devise therapeutic strategies to reduce fatal arrhythmic events in susceptible patients suffering from chronic hypertension.

MATERIAL AND METHODS

This cohort retrospective study was conducted at the department of Cardiac Electrophysiology, Armed Forces Institute of Cardiology in collaboration with Army Medical College, Rawalpindi. An official approval was obtained prior to commencement of the study from medical ethics committee of Army Medical College. Written informed consent was taken from all the patients included in the study. Sixty four patients with systemic arterial hypertension were recruited through non-probability purposive sampling. Patients with acute or old myocardial infarction, diabetes mellitus, cerebrovascular accident, heart failure, structural heart disease, bundle branch block and cardiomyopathies were excluded from the study.

The patients were divided into two groups on the basis of presence or absence of left ventricular hypertrophy. Group I comprised of 32 hypertensive patients with left ventricular hypertrophy whereas group II included 32 hypertensive patients without left ventricular hypertrophy. Selected patients were requested to visit Electrophysiology department of AFIC for Holter monitoring in order to perform signal averaged ECG according to the standard protocol. Signal averaged ECG data were transferred to the computer and edited with the help of DMS CardioScan software. Time domain analysis was used to analyze the cardiac signal. Ventricular late potentials were detected through analysis of filtered QRS complex which characteristically included duration of the filtered QRS complex (fQRS) greater than 114 ms, low amplitude signals (LAS) under 40 μv in the terminal QRS complex greater than 38 ms and root mean square (RMS) voltage in the terminal 40 ms less than 20 μv. Ventricular late potentials were considered positive if any two out of three criteria were fulfilled[14].

Data were analyzed using computer software IBM SPSS version 22. Qualitative variables were presented as frequency and percentages whereas quantitative variables as mean and standard deviation. Comparison of different parameters of signal averaged ECG in patients with and without left ventricular hypertrophy was done by using independent samples t test. Biserial correlation coefficient was used to calculate the correlation between left ventricular hypertrophy and each parameter of signal averaged ECG. Alpha value was kept at 0.05 at confidence level of 95%.

RESULTS

There were 49 male and 15 female patients (N=64) with mean age of 60 ± 1.83 years ranging from 31 to 96. Mean values of signal averaged ECG parameters along with mean value of noise are shown in table-1. Table-2, shows comparison of mean values of signal averaged ECG parameters in patients with and without left ventricular hypertrophy. The table shows that mean values of filtered QRS complex and low amplitude signal are significantly higher in patients with left ventricular hypertrophy as compared to those without the hypertrophy. Whereas, difference between the mean values of root mean square voltage are statistically insignificant. The table also illustrates that difference between the noise levels at which signal averaged ECG was carried out is also insignificant between the two groups.

Table-1. Descriptive statistics of signal averaged ECG parameters and noise level (N=64).

SAECG parameter	Mean ± SD
Filtered QRS complex (fQRS)	108.52 ± 23.63
Low amplitude signal (LAS)	28.81 ± 20.78
Root mean square voltage (RMS)	92.17 ± 51.02
Noise	0.29 ± 0.25

Table-2: Comparison of signal averaged ECG parameters in patients with and without left ventricular hypertrophy (N=64).

SAECG parameter	Mean ± SD		*p*-value
	With LVH	Without LVH	
Filtered QRS complex (FQRS)	118.09 ± 28.49	98.94 ± 11.54	0.001*
Low amplitude signal (LAS)	35.44 ± 25.07	22.19 ± 12.58	0.01*
Root mean square voltage (RMS)	89.94 ± .37	94.41 ± 41.90	0.72
Noise	0.35 ± 28	0.23 ± .19	0.06

*p-value significant (< 0.05)

DISCUSSION

Our study showed that among the three parameters of signal averaged ECG, filtered QRS (fQRS) complex and low amplitude signals (LAS) were significantly higher in patients with left ventricular hypertrophy. However, the difference between root mean square (RMS) voltages of the two groups was statistically insignificant. This implies that perhaps filtered QRS complex and low amplitude signals have higher contributions than root mean square voltage towards the pathogenesis of ventricular arrhythmias in hypertensive patients with left ventricular hypertrophy. Filtered QRS complex and low amplitude signals are related to duration of the cardiac signal whereas root mean square voltage is related to voltage of the signal. This suggests that electrophysiological modifications pertaining to duration of the cardiac signal are probably involved to greater extent in derangements of signal averaged ECG parameters as compared to the voltage of the signal.

Palatini and his colleagues assessed the prevalence of ventricular late potentials through signal averaged ECG in hypertensive patients with left ventricular hypertrophy[15]. One hundred and seven hypertensive patients with left ventricular hypertrophy were enrolled in their study. By the use of 40 Hz filter, they found that filtered QRS complex had a mean value of 129 ms. In our study, we found the mean value of 118 ms for filtered QRS complex

in patients with left ventricular hypertrophy. The reason for a higher mean value of filtered QRS complex in their study seemed to be the difference in sample size. We recruited 32 hypertensive patients with left ventricular hypertrophy whereas Palatini et al included 107 patients. Higher sample size in the study by Palatine et al might have led to rise in mean value of filtered QRS complex. Palatini et al reported that the duration of filtered QRS complex was greater than 114 ms in 20 out of total 27 patients in which signal averaged ECG was found positive for detection of ventricular late potentials (*p*-value < 0.001). Our study also showed that the duration of filtered QRS complex was greater than 114 ms in higher number of patients with left ventricular hypertrophy in which signal averaged ECG was found positive as compared to those without it(*p*-value = 0.001). Palatini et al documented that in hypertensive patients with left ventricular hypertrophy low amplitude signals had a mean value of 34 ms. In our study, we found that low amplitude signals had a mean value of 35 ms in patients with left ventricular hypertrophy. The results of Palatini et al are similar to our study possibly because the number of cycles averaged were same (400 to 700 cycles) and this had a gross effect on low amplitude signals and filtered QRS complex. Also, signal averaged ECG was diagnosed on the basis of similar criteria i.e. filtered QRS complex greater than 114 ms and low amplitude signals under 40 μv greater than 38 ms. The mean value of root mean square voltage reported by Palatini et al was 26 μv whereas in our study we found the mean value of 89 μv. The reason behind this discrepancy might have been that the mean blood pressure of patients enrolled in Palatini's study was 127.6 mmHg compared to 114 mmHg in our study. It is logical to assume that higher systemic arterial blood pressure by increasing left ventricular mass reduces root mean square voltage of filtered QRS complex. This might have further scrutinized their results by having an increased number of patients with root mean square voltage less than 20 μv as compared to our study.

Akdeniz et al studied signal averaged ECG in 99 hypertensive patients, 43 with left ventricular hypertrophy and 56 without it[16]. They reported that the duration of filtered QRS complex was significantly higher in patients with left ventricular hypertrophy (121.3 ms) as compared to those without the hypertrophy (94.2 ms) and the difference was statistically significant (*p*-value < 0.001). We also found the same results, the duration of filtered QRS complex was significantly higher in patients with left ventricular hypertrophy (118.1 ms) as compared to those without the hypertrophy (98.9 ms) and the difference was significant (p-value = 0.001). Regarding the low amplitude signals, they observed that in patients with left ventricular hypertrophy, the mean value of low amplitude signals (29.1 ms) was significantly higher as compared to the mean value in patients without the hypertrophy (*p*-value = 0.03). This was comparable to our findings where the mean value of low amplitude signals (35.4 ms) was significantly greater in patients with left ventricular hypertrophy as compared to those without it (*p*-value = 0.01). Akdeniz and his colleagues also reported that the mean value of root mean square voltage (64.9 μv) was less in patients with left ventricular hypertrophy as compared to those without the hypertrophy, however the difference was statistically insignificant (*p*-value = 0.78). These findings were similar to our results in which we found the mean value of root mean square voltage to be less in hypertrophic (89.9 μv) as compared to non-hypertrophic group (94.4 μv) but the difference was not significant (p-value = 0.73). Similar inclusion/exclusion criteria and cut off values for signal averaged ECG parameters seemed to be the bases for comparable results of the two studies. It appears that left ventricular hypertrophy leads to some functional or structural modifications which significantly affect duration of the cardiac signal whereas these modifications do not affect voltage of the signal to the same extent.

CONCLUSION

Signal averaged ECG parameters are significantly deranged in hypertensive patients

with left ventricular hypertrophy as compared to those without the hypertrophy. This reflects that hypertensive patients with left ventricular hypertrophy may have the substrate for development of ventricular arrhythmias and must be kept under surveillance.

CONFLICT OF INTEREST

This study has no conflict of interest to declare by any author.

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