

ASSOCIATION OF VENTRICULAR LATE POTENTIALS WITH LEFT VENTRICULAR HYPERTROPHY IN PATIENTS WITH SYSTEMIC ARTERIAL HYPERTENSION

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ABSTRACT

Objective: To determine the association of ventricular late potentials with left ventricular hypertrophy in patients with systemic arterial hypertension.

Study Design: Cohort retrospective study.

Place and Duration of Study: Department of Cardiac Electrophysiology, Armed Forces Institute of Cardiology, Rawalpindi from 11th Nov, 2014 to 10th Nov, 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. CardioScan premium luxury software was used for analysis of ventricular late potentials.

Results: There were 49 (76.6%) males and 15 females (23.4%) with the mean age of 60 ± 11.83 years. Ventricular late potentials were revealed in 10 (31.3%) out of 32 patients with left ventricular hypertrophy whereas in patients without hypertrophy only 1 (3.1%) patient showed it. Ventricular late potentials were strongly associated with left ventricular hypertrophy (p -value=0.03) and the relative risk of developing ventricular late potentials was 10 times higher in patients with left ventricular hypertrophy as compared to those without left ventricular hypertrophy.

Conclusion: Ventricular late potentials were strongly associated with left ventricular hypertrophy.

Keywords: Left ventricular hypertrophy, Systemic arterial hypertension, Ventricular late potentials.

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INTRODUCTION

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, 2}. It is a robust risk factor for left ventricular hypertrophy, a compensatory mechanism in response to increased pressure load on the heart. Systemic arterial hypertension and left ventricular hypertrophy are strong predictors of ventricular arrhythmias which may lead to sudden cardiac death^{2,3}.

Knowledge about arrhythmias developing in patients with hypertension is important

because it can significantly affect the prognosis and management of the disease³. Pathophysiological mechanisms underlying the development of left ventricular hypertrophy involves systolic and diastolic pressure overload along with neurohormonal activation^{4,5}. 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 re-entry which may lead to ventricular arrhythmias⁶.

Ventricular late potentials are noninvasive electrocardiographic parameters which can be used to identify hypertensive patients with increased risk of developing ventricular arrhythmias⁷. They are low amplitude, high frequency signals present in the terminal part of

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QRS complex that may extend up to a variable length in ST segment⁸. Ventricular late potentials are detected by a noninvasive, high resolution signal detecting technique known as signal averaged ECG. They are noninvasive markers of myocardial tissue damage⁹. Increased arterial pressure in hypertension results in myocardial fibrosis, a high resistivity area having delayed conduction velocity and prolonged action potential duration. This affects the electrocardiographic signals between the end of QRS complex and the initial part of ST segment thus generating these low voltage fractionated signals⁶. Detection of ventricular late potentials in hypertensive patients 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 death¹⁰.

The current study was planned to determine the association of ventricular late potentials with left ventricular hypertrophy in hypertensive patients. Results of the study would provide an insight into the probable mechanisms of disturbed electrical activity within ventricular myocardium in these patients. It will prove to be helpful to identify the hypertensive patients who are at high risk of sudden cardiac death due to ventricular arrhythmias. This may also help to devise therapeutic strategies to reduce fatal arrhythmic events in susceptible patients suffering from chronic hypertension, especially those with left ventricular hypertrophy.

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 and 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. Signal averaged ECG data were transferred to the computer and edited with the help of DMS Cardio Scan 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.

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. Association between left ventricular hypertrophy and ventricular late potentials was calculated by using Chi Square test. Relative risk was also calculated to determine the effect of association between left ventricular hypertrophy and ventricular late potentials. Alpha value was kept at 0.05 at confidence level of 95%.

RESULTS

Sixty four patients with systemic arterial hypertension (n=64) were analyzed in the current study. Mean age of the patients was 60 ± 11.83 years ranging from 31 to 96. Forty nine male and

15 female patients were enrolled in the study with male to female ratio of 3.3:1. In group I (with left ventricular hypertrophy) 10 patients (31.3%) had ventricular late potentials whereas 22 (68.7%) were without them. In group II (without left

hypertrophy¹¹. They recruited 107 hypertensive patients with left ventricular hypertrophy. Twenty seven of the patients (25%) showed ventricular late potentials. Results of Palatini et al are comparable to our study. Ventricular late

Table-I: Association of left ventricular hypertrophy with ventricular late potentials (n=64).

Left ventricular hypertrophy	Ventricular late potentials		p-value (χ^2 test)	Phi
	Yes	No		
Yes	10 (31.3%)	22 (68.8%)	0.003*	0.4
No	1 (3.1%)	31 (96.9%)		

*p-value significant (<0.05)

Table-II: Relative risk for development of ventricular late potentials in patients with left ventricular hypertrophy (n=64).

Left ventricular hypertrophy	Ventricular late potentials		Relative risk	95% confidence interval
	Yes	No		
Yes	10	22	10*	1.4 – 73.6
No	1	31		

*Association significant

ventricular hypertrophy), only one patient (3.1%) showed ventricular late potentials whereas 31 patients (96.9%) were without the late potentials. By using Chi Square test, the p-value obtained was 0.003 which showed strong association of ventricular late potentials with left ventricular hypertrophy. Effect size was determined by calculating Phi coefficient which was 0.4 (p-value =0.03) indicating moderate effect of association as shown in table-I.

Relative risk was also calculated to determine the association between left ventricular hypertrophy and ventricular late potentials as shown in table-II. The value of relative risk was 10 with 95% confidence interval of 1.4 to 73.6. This suggested that the risk of developing ventricular late potentials is 10 times greater in patients with left ventricular hypertrophy as compared to those without the hypertrophy and the association was significant as evident from the 95% confidence interval.

DISCUSSION

A strong association between ventricular late potentials and left ventricular hypertrophy was revealed in our study. Palatini et al determined the association of ventricular late potentials in hypertensive patients with left ventricular

potentials were diagnosed in both studies by using the same signal averaged ECG criteria. In our study, we found slightly increased frequency of ventricular late potentials (31.3%) in patients with left ventricular hypertrophy as compared to 25% found by Palatini and his colleagues. This might be due to the two channel Holters used by Palatini et al for recording ventricular late potentials whereas in our study we used the latest three channel Holters which being more sensitive might have led to increased frequency of ventricular late potentials in our study.

Palmiero et al studied ventricular late potentials in 60 hypertensive patients with left ventricular hypertrophy¹². Seventeen (20%) out of these 60 patients exhibited ventricular late potentials. They found significant association between left ventricular hypertrophy and ventricular late potentials (p-value <0.001) which was comparable to the results of our study. However, we found an increased frequency of ventricular late potentials in patients with left ventricular hypertrophy. This seemed to be due to the difference in mean blood pressure. The mean blood pressure of our study population was 114 mmHg whereas that of Palmiero et al was 101 mmHg. Degree of hypertension is associated directly with increase in left

ventricular mass and thus left ventricular hypertrophy. Most patients enrolled in our study had chronic hypertension with an increased systolic stress. Thus, the chances of ventricular remodeling and ventricular late potentials were also increased.

Akdeniz et al conducted a study on 99 hypertensive patients to evaluate the association of left ventricular hypertrophy with ventricular late potentials¹³. They divided the patients into two groups on the basis of presence or absence of left ventricular hypertrophy. Forty three patients were enrolled in hypertrophic group while non-hypertrophic group consisted of 56 patients. Ventricular late potentials were present in 25.7% hypertensive patients with left ventricular hypertrophy as compared to 4.9% in non-hypertrophic group (p -value <0.01). We found a slightly increased frequency of ventricular late potentials in patients with left ventricular hypertrophy as compared to the frequency documented by Akdeniz and his colleagues. This might be attributed to the fact that there was an unequal distribution of patients enrolled in both the groups in their study while we ensured an equal distribution of patients in both the groups. Secondly, Akdeniz and his colleagues registered hypertensive patients with mild to moderate left ventricular hypertrophy while in our study patients with severe left ventricular hypertrophy were also included. In cases with severe left ventricular hypertrophy, the increased left ventricular mass resulted in prolonged QRS complex duration and thus the chances of ventricular late potentials were also increased.

CONCLUSION

Ventricular late potentials are strongly associated with left ventricular hypertrophy.

Hence, probability of ventricular late potentials, which are reflective of arrhythmogenic substrate, increases significantly if systemic arterial hypertension is complicated with left

ventricular hypertrophy. These are the patients who are at risk of ventricular arrhythmogenesis which may lead to potentially fatal outcomes. This subset of patients must be put on appropriate prophylactic antiarrhythmic treatment or under medical surveillance.

CONFLICT OF INTEREST

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

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