ORIGINAL ARTICLE

CARDIAC ARRHYTHMIAS AND LEFT VENTRICULAR HYPERTROPHY IN SYSTEMIC HYPERTENSION

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Background: Hypertensive left ventricular hypertrophy (LVH) is associated with increased risk of arrhythmias and mortality. Objective was to investigate the prevalence of cardiac arrhythmias and LVH in systemic hypertension. **Methods**: In all subjects blood pressure was measured, electrocardiography and echocardiography was done. Holter monitoring and exercise test perform in certain cases. There were 500 hypertensive patients, 156 (31.2%) men and 344 (69%) women >30 years of age in the study. Among them 177 (35.4%) were diabetic, 224 (45%) were dyslipidemia, 188 (37.6%) were smokers, and 14 (3%) had homocysteinemia. Duration of hypertension (HTN) was ≥2 years). Mean systolic BP (SBP) was 180±20 mm Hg and diastolic BP (DBP) was 95±12 in male and female patients. Left ventricular mass index (LVMI) was 119.2±30 gm/m^2 in male while 103±22 gm/m^2 in female patients. Palpitation was seen in 126 (25%) male and 299 (59.8%) female patients. Atrial fibrillation was noted in 108 (21.6%) male and 125 (25%) female patients, 30 (6%) male and 82 (16.4%) female patients had atrial flutter. Ventricular tachycardia was noted in 37 (7.4%) male and 59 (11.8%) female patients. Holter monitoring showed significant premature ventricular contractions (PVC'S) in 109 (21.8%) male and 128 (25.69%) female patients while Holter showed atrial arrhythmias (APC'S) in 89 (17.8%) males and 119 (23.8%) females. Angiography findings diagnosed coronary artery disease in 119 (23.8%) with CAD male and 225 (45%) without CAD while 47 (9.4%) females presented with CAD and 109 (21.8%) without CAD. **Conclusion:** A significant association has been demonstrated between hypertension and arrhythmias. Diastolic dysfunction of the left ventricle, left atrial size and function, as well as LVH have been suggested as the underlying risk factors for supraventricular, ventricular arrhythmias and sudden death in hypertensives with LVH.

Keywords: Hypertension, arrhythmias, left ventricular hypertrophy, coronary artery disease

INTRODUCTION

The anatomical and tissue changes caused by hypertension are responsible for the higher incidence of atrial and ventricular arrhythmias as compared to normal population. Arterial hypertension is a widespread disease and one of important yet under-recognised and under-treated causes of atrial and ventricular arrhythmias. Hypertrophy of cardiac muscle in hypertensive patients is characterised not only by increased myocardial mass, but also by proliferation of fibrous tissue and decreased intercellular coupling, that leads to various arrhythmias.[1] The incidence of supraventricular and ventricular arrhythmias in patients with arterial hypertension is up to 96% and is about 10 times higher than in normotensives. Predictors are left ventricular hypertrophy (LVH), impaired left ventricular function with enlarged end-diastolic and end-diastolic volumes as well as late potentials which in case of LVH increase from 7% to 18%. By pharmacological regression of hypertrophy the prevalence of complex arrhythmias decreases.[2] Presence and complexity of both supraventricular and ventricular arrhythmias may influence morbidity, mortality, as well as the quality of life of patients. Diastolic dysfunction of the left ventricle, left atrial size and function, and left ventricular hypertrophy has been suggested as the underlying risk factors for supraventricular and ventricular arrhythmias in hypertensives. ECG parameters are analysis of P wave, QT interval dispersion, heart rate variability, ventricular late potentials and T wave morphology.[3] Concentric LVH, the incidence of ventricular arrhythmia increases in relation with QT dispersion.[4] Increased QT dispersion has been associated with ventricular arrhythmia and sudden death in a variety of cardiac disorders.[5,6] The most consistently observed abnormality is prolongation of the action potential duration and refractoriness which sets the stage for arrhythmias based on early or delayed after depolarisation and triggered activity.[7]

This study was conducted to evaluate the prevalence of arrhythmias in hypertensive patient with the help of ECG, Echocardiography, Holter monitoring, and angiographic results.

MATERIAL AND METHODS

We included 500 hypertensive patients presenting arrhythmias (156 men and 344 women) >30 years of age admitted between August 2006 to August 2008 at Karachi Institute of Heart Diseases. Informed written consent was obtained from all patients. Those patients

suffering from unstable angina, myocardial infarction, systolic dysfunction, valvular heart diseases, electrolyte disturbances, medical disorders like hyperthyroidism, anaemia or with any drug were excluded from the study. In all subjects blood pressure was measured, electrocardiography and echocardiography data obtained and the prevalence of arrhythmias determined by Holter monitoring and exercise test. On the basis of standard 12-lead ECG, left ventricular hypertrophy, atrial arrhythmias, ventricular arrhythmias were noted. Twenty-four hour Holter monitoring (ambulatory electrocardiography) was carried out in patients complaining of palpitations. Patients were encouraged and advised to undertake their usual daily activities except bathing and swimming. The timings of falling asleep and waking-up were noted as accurately as possible. Analysis included the occurrence of supraventricular arrhythmias (SVAs) and ventricular arrhythmias. Echocardiography was done in all patients. Left ventricular end-systolic diameter (LVESD), left ventricular end-diastolic diameter (LVEDD), posterior wall thickness at end-diastole (PWT) and intraventricular septal thickness at end-diastole were obtained according to the recommendations of American Society of Echocardiography. Coronary angiography was done in all patients.

RESULTS

There were 500 hypertensive patients, 156 (31.2%) men and 344 (69%) women, >30 years of age, in the study. Among them 177 (35.4%) were diabetic, 224 (45%) were dyslipidemia, 188 (37.6%) were smokers, and 14 (3%) had homocysteinemia. Duration of hypertension (HTN) was at least 2 years or more. Recorded systolic BP (SBP) was 180±20 while diastolic BP (DBP) was 95±12 in male and female patients. Left ventricular mass index (LVMI) was 119.2±30 gm/m^2 in male while 103±22 gm/m^2 in female patients. Palpitation was noted in 126 (25%) males while 299 (59.8%) female patients, 108 (21.6%) male and 125 (25%) female patients presented with atrial fibrillation, 30 (6%) male and 82 (16.4%) female patients had atrial flutter. Ventricular tachycardia was noted in 59 (7.4%) male and 37 (11.8%) female patients. Eight (1.6%) patients had T wave alternans while 11 (2.2%) patients were diagnosed having long QT interval. Holter monitoring showed ventricular arrhythmias (PVC's) in 128 (25.6%) male and 109 (21.8%) female patients, and atrial arrhythmias (APC's) in 119 (23.8%) males patients 89 (17.8%) female patients. Coronary angiography findings showed 109 (23.8%) male patients suffering from CAD while 225 (45%) did not have CAD. Similarly in female patients 47 (9.4%) had CAD and 109 (21.8%) did not have CAD.

Table-1: Demographics of hypertensive patients with arrhythmias

Total Patient	500	Percentage
Male	156	31.2%
Female	344	69%
Age	>30 years	
HTN duration	>2 years	
Diabetes	177	35.4%
Dyslipidemia	224	45%
Smoking	188	37.6%
Homocysteinemia	14	3%
SBP (mm Hg)	180±20	
DBP (mm Hg)	95.2±12	
LVMI (g/m^2)	119.2±30	

Table-2: Baseline characteristics of the study population (n=500)

Disorder	Men (344)	Women (156)	*p*
Syncope	4 (0.8%)	6 (1.2%)	0.57
Cardiac Arrest	6 (1.2%)	15 (3%)	0.81
Palpitation	126 (25%)	299 (59.8%)	<0.001
Coronary-Artery Disease	48 (9.6%)	13 (2.6%)	0.07
Deaths (total 2%)	7 (1.4%)	3 (0.6%)	0.93

Marginal statistical association exist among palpitation (arrhythmias) and hypertension (p<0.001) and incidence of coronary artery (p=0.07) using Chi-square test

Table-3: Non-invasive and angiographic data in study population (n=500)

Parameter	MEN n (%)	WOMEN n (%)	*p*
ECG Findings			
Atrial Fibrillation	108 (21.6)	125 (25)	<0.001
Atrial Flutter	30 (6.0)	82 (16.4)	0.25
Ventricular Tachycardia	37 (7.4)	59 (11.8)	0.06
Ventricular Fibrillation	5 (1.0)	6 (1.2)	0.30
Ventricular Ectopic Beats	12 (2.4)	17 (3.4)	0.22
T Wave Alternans	5 (1.0)	3 (0.6)	0.05
Long QT Interval	4 (0.8)	7 (1.4)	0.70
Echocardiography-Findings			
IVS ≥ 12 mm	51 (10.2)	123 (24.6)	0.50
PW ≥ 12 mm	13 (2.6)	36 (7.2)	0.45
Increased LVMI	138 (27.6)	255 (51)	0.0003
LA ≥ 40 mm	42 (8.4)	100 (20)	0.62
Holter study			
Ventticular arrhythmias PVCs	109 (21.8)	128 (25.6)	<0.001
Atrial arrhythmias APCs	89 (17.8)	119 (23.8)	<0.001
Coronary-Angiography Findings			
CAD	119 (23.8)	47 (9.4)	---
Without CAD	225 (45)	109 (21.8)	---

DISCUSSION

Hypertension has two major consequences on the heart: left ventricular hypertrophy, and morphological and functional alterations of the coronary macro- and micro-vessels. These two cardiac modifications are responsible for 3 types of complications: myocardial ischemia, left ventricular dysfunction and electrical instability, which are involved in the pathogenesis of ventricular arrhythmias in hypertensive patients.[8] Ventricular arrhythmia is usually triggered by simple or complex ventricular extrasystole which usually involves a re-

entry circuit when there is concomitant LVH.[9] Asymmetric septal and eccentric hypertrophy is associated more often with ventricular arrhythmia than concentric LVH.[10] LVH is involved in the pathogenesis of ventricular arrhythmia but once LVH is controlled then incidence of arrthymias can be reversed.[11] Successful treatment of hypertension causes regression of LVH which has beneficial effect on cardiovascular morbidity and mortality.[12] However, hypertensive patients with persistent LVH have poor prognosis despite of treatment.[13] In these patients the presence of non-sustained ventricular tachycardia on 24-hr Holter monitoring has prognostic value.[14] The potential mechanisms for the development of myocardial ischemia in hypertensive LVH involve changes in the coronary circulation characterised by a reduction of coronary vascular reserve and an acceleration of the atherosclerotic process.[15] After supraventricular extrasystole, atrial fibrillation (AF) is the next most common form of arrhythmia associated with hypertension.[16] Incidence of arterial fibrillation increases with increasing age and can lead to thromboembolic events that may be prevented by anti-coagulation.[17] Ventricular arrhythmias can cause sudden death which is mainly related to ventricular fibrillation. Non-sustained ventricular tachycardia, late ventricular potentials, decreased heart rate variability and baroreflex sensitivity, and repolarisation altemans are further elements to assess risk. The electrophysiologic study with programmed ventricular stimulation remains a costly and invasive method and only has a strong positive predictive value in ischemic cardiomyopathy. Risk stratification are thus needed that may help the strategy of treatment with prophylactic implantable cardio-vert defibrillator (ICD).[18] The incidence and seriousness of arrhythmias correlate with the severity of the LVH, as measured by ECG and echocardiography.[19] The presence of late potentials (LP) is considered as the evidence of an anatomical and electrophysiologic abnormalities are also well documented in hypertensive LVH, and such patients are more predisposed to arrhythmias.[20]

Our study shows 90.4% incidence of ventricular and supraventricular arrhythmias, among them ventricular tachycardia (VT) was 19.2%, ventricular fibrillation 2.2%, and PVCs 47.4%. Incidence of mortality increases in hypertensive patients especially if they are affected with silent ischemia and/or ventricular arrhythmias.[21,22] While global mortality of LVH increases if there is complex or frequent ventricular extra systole, and increase left ventricular mass index even in asymptomatic patients.[23,24] Beta-blockers and amiodarone are considered as the drugs of choice in ventricular arrhythmia although calcium-channel blockers, angiotensin converting enzyme (ACE) inhibitors and angiotensin receptor blockers (ARBs) appear to have a specific effect for hypertensive patients with LVH have been shown to be effective against arrhythmias by virtue of their action against LVH.[25]

CONCLUSION

Left Ventricular Hypertrophy increases risk of atrial and ventricular arrhythmias, systolic and diastolic heart failure and sudden death in patients with hypertension. Controlling arterial blood bressure facilitates regression of LVH and cardiovascular morbidity and mortality.

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