ORIGINAL ARTICLE

Microalbuminuria and left ventricular hypertrophy in patients with essential hypertension

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Abstract

Objectives: To assess the relationship between microalbuminuria and left ventricular hypertrophy in patients with essential hypertension.

Methods: The case-control study was conducted at Al-Yarmouk Teaching Hospital, Baghdad, Iraq, from June 1 to December 31, 2016, and comprised patients with essential hypertension. Based on echocardiography, the patients were divided into 2 equal groups of those with and without left ventricular hypertrophy. Spot urine sample for the patients in the 2 groups was collected to assess microalbuminuria. Blood pressure, smoking status, family history of hypertension, serum creatinine, total cholesterol and blood sugar levels were evaluated. SPSS 22 was used for data analysis.

Results: Of the 100 subjects, 47(47%) were males and 53(53%) were females. The overall mean age was 59±7.2 years. The case and control groups had 50(50%) patients each. The mean albumin-to-creatinine ratio of the patients was 70.5±4.6µg/mg compared to 30.3±16.6µg/mg of the controls ($p<0.05$). The mean systolic blood pressure of patients with microalbuminuria was 163.6±10.5 mmHg, mean diastolic blood pressure was 104.7±7.3mmHg, and mean albumin-to-creatinine ratio was 74±43µg/mg compared to 157.1±0.2mmHg, 96.5±6.8mmHg and 23±13µg/mg, respectively, in patients without microalbuminuria ($p<0.05$ each).

Conclusion: There was found a positive relationship between left ventricular hypertrophy and microalbuminuria in patients with essential hypertension.

Keywords: Microalbuminuria, Left ventricular hypertrophy, Hypertension. (JPMA 69: S-13 (Suppl. 3); 2019)

Introduction

Hypertension (HTN) is a disease that affects about one billion individuals worldwide. It increases the risk for the development of cerebral, cardiac and renal events.[1] Essential hypertension (EH) is one of the most common medical problems in the general population and is one of the most important modifiable cardiovascular (CV) risk factors. Many EH patients may present with overt or sub-clinical target organ damage (TOD) involving the heart, kidneys, central nervous system (CNS) or retina at the time of their initial diagnosis.[2] The cost-effectiveness of blood pressure (BP) reduction using drug therapy is greater in the presence of target organ abnormalities and/or co-morbidities. In this context, assessment of sub-clinical TOD has become the key element in evaluating hypertensive patients. Microalbuminuria (MA), defined as the excretion of >30mg and <300mg a day of albumin in the urine, is one of the earliest indications of kidney injury in patients with diabetes mellitus (DM) and HTN, and is associated with high incidence of cardiovascular morbidity.[3] MA is an early indicator of renal damage and has been demonstrated as one of the principal predictive factors of all-cause CV complications, and CV mortality independent of the traditional risk factors like dyslipidaemia and HTN.[4,5] Left ventricular hypertrophy (LVH) is another manifestation of preclinical disease and has long been known as a powerful independent risk factor for all CV complications of HTN and has also been associated with increased morbidity and mortality.[6] The development of both MA and LVH run parallel due to the impact of not only haemodynamic but also non-haemodynamic factors.[7,8]

The current study was planned to assess the relationship between MA and LVH in patients with EH.

Patients and Methods

The case-control study was conducted at Al-Yarmouk Teaching Hospital, Baghdad, Iraq, from June 1 to December 31, 2016, and comprised newly-diagnosed adult EH patients attending the outpatient clinic, or admitted to the medical ward. Those included were hypertensives with systolic blood pressure (SBP) equal to or >140mmHg, diastolic blood pressure (DBP) equal to or >90mmHg, or both.

Those excluded were patients with secondary HTN, concomitant illnesses, any chronic cardiac, hepatic or renal condition, history of DM, and patients who were febrile, anaemic, disabled or uncooperative, as well as, patients with inflammatory diseases or cancer.

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After the participants provided informed consent, they were divided into 2 equal groups comprising those with and without LVH. The presence or absence of LVH was determined by echocardiogram (ECG) findings.

Demographic information collected included age, gender, smoking status and family history of HTN. Blood samples were drawn and evaluated for fasting blood glucose (FBG), serum creatinine, and total cholesterol (TC).

BP was measured with a mercury sphygmomanometer while the patients were in the sitting position, and the arms were positioned at the level of the heart. Two measurements were made at least 5 minutes apart. The average of the two values was taken as the baseline BP.

All ECGs were performed using ultrasonography machines (Vivid EQ and Philips CX50). Left ventricle (LV) mass, size of cardiac chambers, ejection fraction (EF) and shortening fraction were calculated, and the valves were examined. Pulse Doppler was done to estimate the E/A wave ratio of the mitral valve, while colour Doppler and tissue Doppler were also performed. All the measurements and views were obtained according to the recommendations of the American Society of Echocardiography.[9,10]

The LV mass was calculated in grams on the basis of ventricular septal thickness (VST), left ventricular internal dimension (LVID), and posterior wall thickness (PWT) using the formula[11]:

LV mass = 0.80 × 1.04 [(VST × LVID × PWT) 3 - (LVID) 3] + 0.6.

A patient was considered to have LVH if the LV mass was >150g for women, and >200g for men.[12]

A random urine sample from each patient was tested for albumin and creatinine separately. After taking the sample, it was centrifuged for 7-10 minutes after which 0.02 ml of it was taken and was mixed with 1ml of a antiserum reagent and incubated for 5 minutes at 37.0 C~. The absorbance of the sample was measured at 623nm against the reagent blank, using a spectrophotometer. Next, 10 microliter of water and standard solution were used to measure the absorbance of the reagent blank and the standard respectively. The concentration of albumin was calculated by using the equation:

(A sample/a standard)× Concentration of standard= concentration of albumin.[13]

Urinary creatinine was determined by taking 0.2 ml from the centrifuged sample and mixing it with 10ml distilled water and putting in an autoanalyzer (Flexor ELITech) and noting down the result .

Finally, albumin-to-creatinine ratio (ACR) was calculated using the equation: ACR= urine albumin / urine creatinine. MA was defined as ACR ranging 30-300µg/mg.[14]

Data was analysed using SPSS 22 and Microsoft Excel. and was expressed as means ± standard deviation (SD) as well as and frequencies and percentages. Chi-square test of significance of association was used to assess relations between categorical variable, while independent t-test was used to analyse the difference between the means of continuous variables. Level of significance was set at $p<0.05$.t.

Results

Of the 100 subjects, 47(47%) were males and 53(53%)

Table-1: Relationship between demographic, clinical, echocardiography, and laboratory results of the patients with essential hypertension and left ventricular hypertrophy (LVH).

Characteristics of the patients			Left ventricular hypertrophy		P value
			Yes	No	
Age			61.7 7	57.4 6	0.003
Sex	Male	47	26(55.3%)	21(44.7%)	0.897
	Female	53	24(45.3%)	29(54.7%)	
Smoking	Present	49	27(55.1%)	22(44.9%)	0.028
	Absent	51	17(33.3%)	34(66.7%)	
Family history of essential hypertension	Present	47	20(42.6%)	27(57.4%)	0.784
	Absent	53	24(45.3%)	29(54.7%)	
Systolic blood pressure (mmHg)			164.34 10.9	157.1 8.8	<0.001
Diastolic blood pressure (mmHg)			102.4 8.2	98.8 7.8	0.028
Ejection fraction%			55.8 3.8	58.8 4.6	0.001
Serum cholesterol(mg/dl)			195.3 24.7	181.4 17.9	0.002
FBG (mg/dl)			93.7 9	90.2 7.8	0.043
Serum creatinine (mg/dl)			0.9 0.4	0.8 0.3	0.042
ACR (g/mg)			70.5 4.6	30.3 16.6	<0.001
Microalbuminuria			31(64.5%)	17(35.5)	<0.001

FBG: Fasting Blood Glucose ACR: Albumin Creatinine Ratio

Table-2: Relationship between demographic and clinical characteristics of the patients and microalbuminuria.

Demographic and clinical characteristics	Microalbuminuria		P value
	Yes	No	
Age (year)	62.3 6	56.5 7	<0.001
Systolic blood pressure (mmHg)	163.6 10.5	157.1 .2	0.002
Diastolic blood pressure (mmHg)	104.7 7.3	96.5 6.8	<0.001
Serum cholesterol	199.0 22.5	176.9 15.7	<0.001
FBG	95.5 7.8	88.4 7.6	<0.001
Serum creatinine(mg/dl)	1.1 0.2	0.8 0.1	<0.001
ACR µg/mg	74 43	23 13	<0.001

FBG: Fasting Blood Glucose ACR: Albumin Creatinine Ratio

Table-3: Relationship between stages of hypertension and development of left ventricular hypertrophy (LVH) and microalbuminuria.

Stages of hypertension	Left ventricular hypertrophy		Microalbuminuria	
	Yes	No	Yes	No
I	10(27%)	27(73%)	10(27%)	27(73%)
II	23(51.1%)	22(48.9%)	24(53.3%)	21(46.7%)
III	11(61.1%)	7(38.9%)	14(77.8%)	4(22.2%)
P value	0.025		0.001	

were females. The overall mean age was 59±7.2 years. The case and control groups had 50(50%) patients each. Details of smoking status and family history of HTN as well as serum TC, creatinine and FBS values were noted for both groups (Table-1). Age was positively associated with LVH (p=0.003), while there was no relation between gender or family history of HTN with LVH (p>0.05). Mean SBP, DBP, FBG, TC, creatinine and ACR were higher in patients than in the controls (p<0.05).

Patients with MA had a mean age of 62.3±6 years, which was higher than of those without MA. Likewise, mean SBP, DBP, TC, FBG, creatinine and ACR values were higher in AM patients than those without MA (Table-2).

The relationship between HTN stage and MA was statistically significant (p=0.001) (Table-3).

Discussion

The study found that patients with EH and LVH had higher MA levels than patients without LVH. The other important finding is that MA prevalence higher in patients with HTN and LVH. This observational finding confirms the importance of MA as a risk factor for CV diseases, especially coronary artery disease (CAD).

Regarding the mean age of patients, it was 59±7.2 years which is compatible with other studies.[15-18] With reference to the gender distribution, it was 47% male and 53% female which was also compatible with other studies,[19,20] while it was incompatible with some others.[15,17,18] The current study had 49 (49%) smokers which was consistent with some studies[19,21] and inconsistent with others.[17,18] The current study had 49 (49%) patients with a positive family history of HTN. Again, this was in line with some studies[19] and in contrast with others.[18,21]

Indeed, the same was the case with all the other parameters, including mean SBP, DBPTC, FBG, Creatinine ACR, MA prevalence and and the relationship between MA and LVH.[15-19,21,22]

The limitation of the current study is its small sample size which cannot reflect the whole history of EH patients. Also, the study used an ordinary BP machine instead of opting for 24-hBP monitoring which is more accurate.

Conclusion

There was a high prevalence of MA and LVH in patients with EH. Early detection of LVH can be treated by anti-hypertensive drugs in addition to lifestyle modification that can avoid or reduce HTN-related morbidity and mortality. Besides, MA showed comparable strength of association with LVH in patients with EH, showing a positive correlation between the two conditions. Predicting LVH via an easily obtainable laboratory test, such as MA, can be clinically useful.

Disclaimer: Nothing to declare

Conflict of Interest: The authors have no conflict of interests.

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