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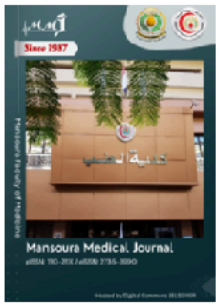

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By

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ABSTRACT

Background : Left ventricular hypertrophy (LVH) is an independent risk factor for cardiovascular morbidity and mortality in hypertensive patients. The identification of risk factors for the initiation of LVH in patients with hypertension (HTN) is important including microalbuminunuria (MAU) and hyperaldosteronism.

Objectives : Evaluation of the relationship of MAU and plasma aldosterone to blood pressure (BP) and LVH in patients with essential HTN.

Subjects and Methods : Thirty male patients with essential HTN and 15 healthy subjects as a control group were subjected to thorough clinical examination, transthoracic echocardiography, lipid profile, serum potassium, serum aldosterone estimation. MAU was evaluated with dipstick Micral-II Test of fasting midstream morning urine on two successive days. Left ventricular mass index (LVMI) was calculated and values >134gm/m^2 were considered as LVH.

Results : Patients with LVMI >134 gm/m^2 had higher serum aldosterone, BMI, Interventricular septal

dence that MAU is an independent predictor of atherosclerosis and premature death in the general population [10].

Numerous clinical studies found an association between MAU and other CV risk factors, TOD and risk of cardiovascular disease (CVD) in clinical contexts different from diabetes mellitus DM and including HTN [11,12].

MAU is a useful, cost-effective marker of increased CV risk although still too often neglected in clinical practice [13].

The identification of risk factors for the initiation of LVH in hypertensive patients is important. The objectives of the present study was to identify the relationship of aldosterone and MAU to LVH in patients with essential HTN.

Although primary aldosteronism is a well recognized cause of secondary HTN, yet it is unknown whether serum aldosterone levels within the physiological range do influence the risk of HTN-TOD [14] and this was one of the goals of the present study. It was also aimed to identify whether MAU in essential HTN, is the victim or villain on LVH and its relation to the studied CV risk factors including aldosterone.

The availability of selective aldosterone antagonists that could improve myocardial dysfunctions and arrest the enhancing extracellular matrix, collagen deposition and fibrosis of hyperaldosteronism [15], and improving myocardial function in hypertensive heart disease [5] was one of the triggers to the present work.

PATIENTS AND METHODS

The present study comprised 30 male patients randomly selected from the outpatient clinic of Specialized Medical Hospital, Mansoura University with mean age 53±6.2 years, and 15 non-hypertensive normal male subjects of matched age and body weight as control group in the period from August 2004 to July 2005.

Complete medical history, physical examination, routine blood and urine analyses, and specific diagnostic procedures to exclude cases with secon-

the sum of IVST and PWT; relative wall thickness (RWT) was computed as (IVST+PWT)/ EDD [19].

Left ventricular mass (LVM) was calculated from M mode echocardiograms according to the formula described by Devereux et al. (1993) [19]: Left ventricular mass M mode (g) = 0.8 (1.04 [EDD +SWth + PWth]^3 - EDD^3) + 0.6g.

Left ventricular mass was indexed to body surface area as left ventricular mass index in g/m^2 body surface area. Left ventricular hypertrophy by M mode criteria was considered when left ventricular mass index > 134 g/ m^2 body surface area in men (19).

BIOCHEMICAL MEASUREMENTS :

Fasting blood samples (3ml each) were withdrawn from patients and control subjects after resting in supine position for 60 minutes and delivered into plain tubes. The separated sera were aliquoted and kept frozen (-70 c) till analysis of serum cholesterol, triglycerides, LDL, and HDL using the respective kits supplied by Aumon, Germany, and serum aldosterone using coat-A-count solid phase radioimmunoassay [20]. The materials were supplied by diagnostic product corporation (DPCs). Serum potassium was assessed using electrolyte analyzer AVL 980, Switzerland.

The fasting midstream morning urine is tested for microalbuminuria using Micral-II test strips for the immunological semiquantitative determination of microalbuminuria. The screening result is positive when the reaction colour corresponding to 20mg/L (threshold value of microalbuminuria) or more on two successive occasions.

STATISTICAL ANALYSIS

All data were analyzed using a SPSS/PC statistical (SPSS Inc. Chicago, IL) package. Inter-group differences between continuous variables were assessed by two-tailed t-tests. Pearson correlation coefficient was used to study correlation between variables. Significance was considered when the P-value less than 0.05 at a confidence interval 95%.

SBP, pulse pressure, BMI, LVMI and serum aldosterone.

Figure (1) shows that serum aldosterone is significantly higher in hypertensive cases than in normotensive control subjects.

Figure (2) shows that serum aldosterone is significantly higher in hypertensive cases with LVH than in those without LVH.

Figure (3) shows that there is a significant positive correlation of microalbuminuria with serum aldosterone in hypertensive patients.

Table (1): Clinical and laboratory data of the test group:

Parameter	Mean	± SD
Age (year)	53	6.2
Weight (Kg)	83.46	13.28
Height (m)	165.	11.42
BMI (kg/m^2)	30.46	4.11
Systolic blood pressure (mmHg)	162.0	12.4
Diastolic blood pressure (mmHg)	99.66	4.7
Serum creatinine (mg/dL)	0.9	0.1
Serum potassium (meq/L)	3.8	0.1
Serum sodium (meq/L)	140	1.0
Serum cholesterol (mg/dL)	203.73	42.90
Serum triglycerides (mg/dL)	111.80	57.53
HDL-C (mg/dL)	38.06	6.99
LDL-C (mg/dL)	143.73	48.14
Microalbuminuria (mg/L)	38.66	29.21
Serum aldosterone (ng/dl)	17.71	8.58

Table (5): Comparison between hypertensive cases with LVH (i.e. LVMI> 134 gm/m^2) and those with no LVH (i.e. LVMI< 134 gm/m^2) :

Parameter	No LVH: LVMI <134(gm/m^2) (n=18)	LVH: LVMI >134 (gm/m^2) (n=12)	P value
Age (year)	52.22± 6.82	54.50± 5.31	NS
Serum aldosterone (ng/dl)	13.87± 7.25	23.47± 7.24	0.001**
BMI (kg/m^2)	28.44± 3.56	33.50± 2.87	0.0001***
SBP (mmHg)	161.11± 12.07	163.33± 13.37	NS
DBP (mmHg)	100.0± 5.94	99.16± 1.94	NS
IVST (mm)	1.19± 0.19	1.78± 0.18	0.0001***
PWT (mm)	1.08± 0.14	1.61± 0.26	0.0001***
RWT	0.40±0.0004	0.62	0.0001***
LV wall thickness	2.22± 0.33	3.39± 0.41	0.0001***
EDD (mm)	5.35± 0.54	5.41± 0.22	NS
LA (mm)	3.65± 0.59	3.78± 0.40	NS
MFS (%)	30.33± 7.42	31.83± 7.14	NS
Ejection fraction (%)	58.55± 11.53	63.16± 9.35	NS
S. cholesterol (mg/dL)	212.88± 51.74	190.0±19.28	NS
S. triglycerides (mg/dL)	103.33± 54.46	124.5±62.04	NS
HDL-C (mg/dL)	37.88± 8.49	38.33± 4.16	NS
LDL-C (mg/dL)	154.22±56.71	128.0± 26.28	NS

Table (6): Prevalence of microalbuminuria in the hypertensive group:

	N	%
Cases with microalbuminuria	14	46.7
Cases without microalbuminuria	16	53.3

Table (7): Correlation of microalbuminuria to some studied parameters:

	Microalbuminuria	
	r	P
Age	N.S.	0.10
SBP	0.34	**0.02***
DBP	0.13	N.S.
LVMI	0.18	**0.048***
BMI	-0.30	**0.041***
Pulse pressure	0.389	**0.01***
Aldosterone	0.423	**0.003****
Total cholesterol	0.025	N.S.
Serum triglycerides	N.S.	-0.20
HDL-C	-.0126	N.S.
LDL-C	-0.59	N.S.

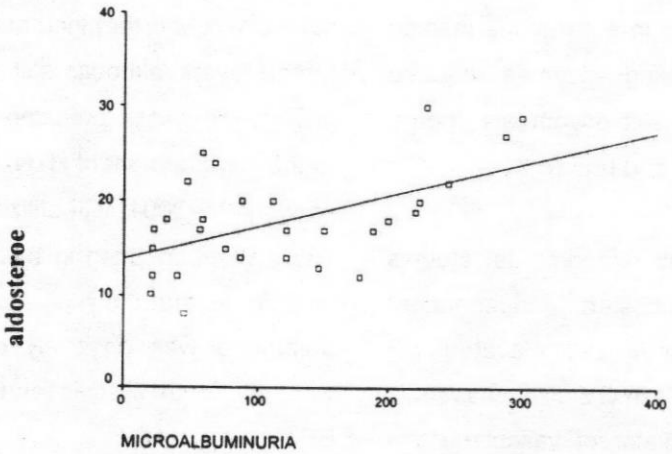

Figure (3): Correlation of microalbuminuria with serum aldosterone in hypertensive patients.

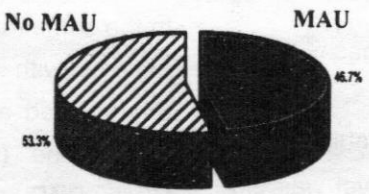

Figure (4): Prevalence of microalbuminuria in hypertensive patients

(2004) [28] had found that the serum aldosterone was strongly related to septal and posterior wall thickness. Ahmet and coworkers [27], Soylu et al. (2004) [28] found the highest plasma aldosterone concentration were in cases of essential HTN with the concentric type of LVH.

Ramarchandran et al. (2004) [23] concluded that increased aldosterone levels even within the physiologic range predispose to development of HTN. However, the present results are not in agreement with Vasan et al. (2004) [29] who found significant positive relation of serum aldosterone to LVWT and RWT only in women. The studied cases were all males (tables 1& 4).

Aldosterone influence LV remodeling independent of its impact on systemic BP [30] and this is achieved via different effects on collagen synthesis and fibroblast proliferation [4], endothelial dysfunction, autonomic dysfunction [31,32].

In the present study, the prevalence of MAU in the hypertensive patient was 46.7% (tables 6 & fig. 4). This is somewhat higher than that reported by previous studies [33,34] where the prevalence ranges between 4 to 46%. This could be due to the huge intra-individual variability in urinary albumin excretion rate, the discrepancies of techniques of measurement and the wide range of MAU (30-300 mg/24 hs). The BMI of the studied group was 30.46±4.11 (table 1) could explain the higher prevalence of MAU in our hypertensive group.

Lieb et al. (2006) [35] found that at general population level, even low grade albuminuria is associated with LVH and the conventional urinary albumin threshold of MAU (30 mg/day) may be too conservative and end organ damage (LVH) is observed with increased frequency at much lower level.

In the present study, MAU showed significant positive correlation with serum aldosterone (P=0.003) (table 7 & fig. 3) and this is in agreement with

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