Journal of Indian College of Cardiology 7 (2017) 159–162

Contents lists available at ScienceDirect

Journal of Indian College of Cardiology

journal homepage: www.elsevier.com/locate/jicc

Original Article

The micro-albuminuria is a new predictor of atrial fibrillation in hypertensive patients

Wael Ali Khalil*, Mohammad Gouda, Moataz Adel, Mohammad Wafaie

Zagazig University, Zagazig, Egypt

ARTICLE INFO

Article history:
Received 6 May 2017
Accepted 15 October 2017
Available online 31 October 2017

Keywords:
Micro-albuminuria
Hypertension
Atrial fibrillation

ABSTRACT

Background: Hypertension increases the risk of atrial fibrillation by about twofold. Micro-albuminuria is an independent predictor of cardiovascular morbidity and mortality in patients with atrial fibrillation.
Aim of the work: The aim of this work was to investigate the relationship between micro-albuminuria 1 (MAU) and occurrence of atrial fibrillation in patients with arterial hypertension.
Patients and methods: Our study was conducted in Cardiology Department, Zagazig University. We recruited 300 consecutive patients with newly diagnosed hypertension from outpatient clinics. In this prospective study, we checked for micro-albuminuria (MAU) 1 at presentation. Then a follow up for 6 months to all cases to elicit any documented attack(s) of paroxysmal or persistent atrial fibrillation. Then patients were classified according to development of AF into Group I (patients who did not develop AF during 6 months after diagnosis of hypertension) (230 patients) and Group II (patients who developed AF during 6 months after diagnosis of hypertension) (70 patients).
Results: The micro-albuminuria was the strongest predictor to develop AF in our cases as the upper and lower "confidence interval" CI was (0.138–0.014) with high significant difference ($p < 0.001$), odds ratio = 0.051. Systolic blood pressure, diastolic blood pressure and heart rate were significant ($p < 0.05$) but less powerful than micro-albuminuria to predict the occurrence of AF.
Conclusion: Micro-albuminuria was the strongest predictor of AF in hypertensive patients.
Recommendation and clinical implications: The early detection and treatment of micro-albuminuria is accompanied by an improved cardiovascular prognosis in hypertensive patients and prevent new onset of atrial fibrillation.

1. Introduction

Micro-albuminuria (MAU) is an independent predictor of cardiovascular morbidity and mortality in both men and women with essential hypertension.[2] Micro-albuminuria defined as urinary albumin excretion (UAE) is more than >30 mg/24 h (20 μg/tm) and less than <300 mg/24 h (200 μg/tm) regardless of how the sample was collected.[1] Albumin excretion in healthy individuals ranges from 1.5 to 20 μg/min.[2] Micro-albuminuria (MAU) is an accepted marker of generalized endothelial injury and dysfunction. It is closely related to vascular disease in ischemic heart disease, congestive heart failure or severe arterial hypertension.[3] In addition to its function as a marker of kidney damage, (MAU) represents an indirect window to monitor the status of whole vasculature. The pathophysiological mechanisms involved in trans-capillary leakage of albumin might be related to an increased intra-glomerular pressure in hypertension.[4] High heart rate has been shown to be associated with cardiovascular complications. The association of an elevated UAE to a series of alterations, such as endothelial dysfunction, insulin resistance, altered lipid levels, higher body mass index, increased serum uric acid and salt-sensitivity. This applies to cardiovascular complications in general population, in hypertensive individuals,[5] in ischemic heart disease,[6] in Heart failure and in diabetes.[7] Atrial fibrillation (AF) is the most common sustained cardiac arrhythmia and increases the risk of cardiovascular mortality by approximately two-fold and is linked to the severity of underlying heart disease.[8] The presence of hypertension accompanied by left ventricular hypertrophy and left atrial chamber dilatation is regarded as cardiac risk factor for the development of AF.[9] In heart failure patients, new onset of AF can occur in 5–50% of cases depending on severity of heart failure and is associated with higher rates and worse outcomes.[10] In patients without a history of AF, an association between heart rate and (MAU) has been described to occur independently of beta-blocker use and physical activity.[11] Higher heart rates and AF are associated with (MAU). Atrial

* Corresponding author.
E-mail address: Drwael123456@yahoo.com (W.A. Khalil).

https://doi.org/10.1016/j.jicc.2017.10.002
1561-8811/

fibrillation might be either considered as a cause of end organ damage or as a result from prolonged vascular damage, both indicated by (MAU). (MAU) is also very common in hypertensive patients at high risk indicating vascular end organ damage. This helps for effective primary and secondary preventive measures.[12]

2. Patients and methods

Our study was conducted in Cardiology Department, Zagazig University in the period from November 2014 until December 2016. We recruited 300 consecutive patients with newly diagnosed hypertension from outpatient clinics. In this prospective study, we checked for micro-albuminuria (MAU) at presentation. Then a follow up for 6 months to all cases to elicit any documented attack (s) of paroxysmal or persistent atrial fibrillation through clinical symptoms, serial examination and ECG or 24–48 h Holter examination. Then patients were classified according to development of AF into Group I (patients who did not develop AF during 6 months after diagnosis of hypertension) (230 patients) and Group II (patients who developed AF during 6 months after diagnosis of hypertension) (70 patients).

Inclusion criteria: all patients with newly diagnosed hypertension from outpatient clinics.[3]

Exclusion criteria: patients were presented with:

(1) Fever.
(2) Concomitant urinary tract infection or renal disease (elevated serum creatinine, on dialysis or Calculated GFR < 90 mL/min/1.73 m^2).[13]
(3) Strenuous physical exercise in the preceding 24 h.
(4) Diabetes mellitus.

3. Methods

Patients fulfilling eligibility criteria were subjected to the following:

(1) Full history taking with special emphasis on the risk factors such as smoking, diabetes mellitus, hypertension and renal impairment.
(2) General examination specially blood pressure and heart rate must be measured by treating physician twice in a supine position after 5 min of rest using standardized mercury sphygmomanometers and proper cuff size was used.[4] The diagnosis of the presence of essential hypertension was considered when systolic blood pressure ≥ 140 mmHg and diastolic blood pressure ≥ 90 mmHg on at least three visits. According to the consensus statement of the European Society of Hypertension and the American College of Cardiology.[14]
(3) Local examination of the heart.
(4) ECG; high quality 12 leads ECG.
(5) Echocardiography was done using Vivid S5, to estimate EF% and LVMI (The ejection fraction is calculated from the difference between end-diastolic and end-systolic LV volume, divided by the end-diastolic LV volume. Preserved left ventricular EF was defined as $\geq$50%. LV mass was calculated with the formula: LV mass = $0.8 \times \{1.04[([\text{LV internal dimension} + \text{septal wall thickness} + \text{posterior wall thickness}]3 - \text{LV internal dimension}3)] + 0.6\}$. Left ventricular mass was indexed to Body Surface Area and normal range define as (43–95) for female and (49–115) for male g/m^2 for both sexes).[15]

In our study, we estimated the presence of urinary albumin excretion by an immunoturbidimetric test.[5,6] The concentrations of albumin in urine samples were measured with a Hitachi 7600 using a turbidimetric immunoassay. The urine samples were collected in the first urine void in the morning. The turbidimetric immunoassay (quantitative method) urine sample causes agglutination of the latex particles coated with anti-human albumin. The agglutination of the particles is proportional to the albumin concentration. Urine should be centrifuged before analysis. Normal value for adults' is up to 20 mg/l.[16]

Follow up of urinary albumin excretion post AF and 6 months later.

Serum creatinine level was estimated to exclude patients with renal impairment.

3.1. Ethical consideration

This study was approved by Ethics Committee of Faculty of Medicine, Zagazig University, Egypt. A written consent from every patient to participate in the study was obtained and Consistence with ethical standards.

3.2. Statistic analyses

All statistical analyses were performed with SPSS version 17.0 (SPSS Inc., Chicago, IL, USA). Continuous variables were expressed as mean $\pm$ standard deviation and categorical data were presented as frequencies. For comparisons between groups, we used Student's t-test to evaluate difference in means and Univariable comparisons were analyzed by the χ^2 test. To test the independent relationship between MAU and variables, potential factors was selected by univariate regression analysis, and selected variables were assessed in a multiple logistic regression analysis mode. A correlation co-efficient and regression analysis were done. p value <0.05 was considered to be statistically significant.

4. Results

The age of our patients ranged 26–83 years with mean of 56.5 ± 13.58 years old. This study included 162 (54%) males and 138 (46%) females. BMI ranged 24–36 kg/m^2 with mean of 30.54 ± 3.08 kg/m^2. We had 132 smokers (44%). The heart rate at diagnosis of HTN was ranged 55–110 bpm with mean of 79.07 ± 14.18 bpm. Systolic blood pressure ranged 140–200 mmHg with mean of 167.33 ± 15.72 mmHg. Diastolic blood pressure ranged 90–130 mmHg with mean of 101.26 ± 9.56 mmHg. Ejection fraction ranged 42–69% with mean of 56.26 ± 9.28%. Left ventricular mass index ranged 45–130 g/m^2 with mean of 89.5 ± 32.11 g/m^2.[6] Micro-albuminuria ranged 2–139 mg/l with mean $\pm$ SD = 30.2 $\pm$ 34.57 mg/l in (group I). The MUA ranged 4–237 mg/l with mean $\pm$ SD = 43.94 ± 51.77 mg/l at 6 months in (group II). Serum creatinine ranged 0.5–1.4 mg/dl with mean of 0.9 ± 0.51 mg/dl. As regarding heart rate, it ranged between 55–110 and 70–200 bpm with mean of 79.07 ± 14.18 and 140.43 ± 39.95 bpm respectively in group I and group II, there was highly significant difference between the two studied groups regarding HR ($t = 91.86$, $p < 0.001$; Table 1). Systolic blood pressure ranged between 140–200 and 100–180 mmHg with mean of 167.33 ± 15.72 and 137.47 ± 18.25 mmHg in-group I and group II respectively. There was high statistical significant difference between the two studied groups ($t = 62.31$, $p < 0.001$; Table 1). Diastolic blood pressure ranged between 90–130 mmHg and 60–120 mmHg with mean of 101.26 ± 9.56 and 87.57 ± 12.09 mmHg for group I and group II respectively. There was a high significant difference between the two groups ($t = 54.52$, $p < 0.001$; Table 1).[7] LVMI ranged between 45–130 and 45–145 g/m^2 with mean of 89.50 ± 32.11 and 99.71 ± 32.58 g/m^2 for group I and group II respectively, a high statistical significant difference between the two studied groups ($t = 2.32$, $p < 0.001$; Table 1). Micro-albuminuria appears in 59