Microalbuminuria is a late event in patients with hypertension: Do we need a lower threshold?

Mohamed Abdel Kader Abdel Wahab [a,*], Mohamed Mohamed Saad [a], Khaled Abdel Ghany Baraka [a]

[a] Department of Cardiology, El Minya

[a] Egypt

Background: **Microalbuminuria (MA) is a marker of vascular damage. However, many studies have observed an increased risk at lower levels of albuminuria than are currently used to define MA.**

Aim: **To verify early cardiovascular changes occurring before MA in hypertensive patients.**

Materials and methods: **One hundred and fifty hypertensive patients and 60 normotensive individuals were divided into normotensive individuals with normal left ventricular (LV) geometry (Group I), hypertensive patients with normal LV geometry (Group II), and hypertensive patients with abnormal LV geometry (Group III). The LV mass index, ambulatory arterial stiffness index, flow-mediated dilatation of the brachial artery, and intima-media thickness (IMT) of the common carotid were assessed. Urinary albumin/creatinine ratio was determined using a morning spot-urine sample.**

Results: **Compared with Group I, ambulatory arterial stiffness index and IMT were significantly increased and flow-mediated dilatation was significantly decreased in Group II; however, MA did not differ between both groups. These changes were augmented when Group III was compared with Group II. MA significantly increased in Group III compared with Group II. Receiver operating characteristic analysis revealed that MA, with a cut-off value of 19.25 mg/g, predicted increased IMT, and abnormal LV geometry in a statistically significant manner.**

Conclusion: **Many vascular changes, in the form of increased IMT, reduced vasodilator capacity, and increased arterial stiffness, preceded MA and any change in LV geometry. The results presented here strengthen the usefulness of adopting a lower cut-off to define MA.**

Keywords: Endothelium, Hypertension, Microalbuminuria

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* Corresponding author at: Department of Cardiology, El Minya University Hospital, Minya, Egypt.
E-mail address: makder1999@yahoo.com (M.A.K.A. Wahab).

P.O. Box 2925 Riyadh – 11461KSA
Tel: +966 1 2520088 ext 40151
Fax: +966 1 2520718
Email: sha@sha.org.sa
URL: www.sha.org.sa

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Introduction

Microalbuminuria (MA) is known to be associated with atherosclerosis and is a strong independent predictor of an increased risk of cardiovascular morbidity and mortality independent of traditional risk factors [1,2]. MA is a well-known marker of underlying vascular dysfunction and has been correlated with the structural and functional integrity of the vasculature [3].

The prevalence of MA is low in the absence of cardiovascular risk factors and progressively increases with the number of cardiovascular risk factors. The main correlate of MA is blood pressure, either systolic or diastolic [4]. The relationship between blood pressure and MA is continuous and graded because the prevalence of MA increases with the severity of hypertension [5].

However, some studies have found that increased risk is observed at much lower levels of MA than are currently used to define MA [6].

Methods

This study was carried out in the Cardiology Department at El Minya University Hospital, Minya, Egypt, over a 13-month period between December 2013 and January 2015.

This study included 150 newly discovered untreated hypertensive patients (diagnosed according to European Society of Hypertension/European Society of Cardiology guidelines for management of arterial hypertension) compared with 60 sex and age matched healthy controls [7]. They were classified into the following three groups: Group 1: 60 normotensive apparently healthy individuals with normal (left ventricular) LV geometry; Group 2: 54 hypertensive patients with normal LV geometry; and Group 3: 96 hypertensive patients with abnormal LV geometry.

The following exclusion criteria were used in this study: (1) tobacco smoking; (2) previous myocardial infarction (3) coronary bypass graft; (4) cardiac valve disease; (5) stroke; (6) diabetes; (7) dyslipidaemia; and (8) obesity.

All of the participants (after written consent) were subjected to the following measurements: history taking, clinical examination, office blood pressure measurement (patients with a systolic blood pressure $\geqslant$140 mmHg and diastolic blood pressure $\geqslant$90 mmHg were considered to be hypertensive), body mass index and body surface area measurements, laboratory investigation (fasting and postprandial blood sugar, lipid profile, blood urea nitrogen, serum creatinine, glomerular filtration rate, and quantification of albumin in urine), and ambulatory blood pressure monitoring using AMP50 (China).

Calculation of the ambulatory arterial stiffness index

For each participant, the regression slope of diastolic on systolic blood pressure was calculated from 24-hour recordings. The regression line was not forced through the origin (intercept 0) because blood pressure does not drop to 0 when the flow drops to 0 during diastole [8].

The rationale underlying this is that for any given increase in distending arterial pressure, systolic and diastolic pressures tend to increase in a parallel fashion in a compliant artery. By comparison, in a stiff artery, the increase in systolic pressure is accompanied by a lesser increase, or even by a decrease in diastolic pressure.

The ambulatory arterial stiffness index (AASI) was defined as 1 minus the regression slope. The stiffer the arterial tree, the closer the regression slope and AASI are to 0 and 1, respectively [8].

Assessment of MA

Urinary albumin and creatinine concentrations were determined using a morning spot-urine sample. Urine albumin was determined using the enzyme-linked immunosorbent assay method [disease related group microalbumin enzyme-linked immunosorbent assay (EIA-2361) USA], whereas urinary creatinine was assessed with the kinetic Jaffe' method using the Autoanalyzer Modular (Hitachi–Roche Diagnostics, Manneheim, Germany). The urinary albumin-to-creatinine ratio (ACR) was then calculated. MA was defined as an ACR of 30–300 mg/g in two positive tests from three [9].

Colored duplex ultrasound to measure the intima-media thickness

A carotid ultrasound evaluation was conducted on all of the participants in this study to determine the intima-media thickness (IMT) and to detect carotid plaques using the high-resolution B-mode ultrasound equipment Medison 9900 Multibeam 30 UL (Korea) equipped with a liner probe (7.5 MHz) and a standardized protocol [10].

The individuals were investigated in the supine position, and the IMT of the far wall was evaluated as the distance between the luminal–intimal interfaces and the medial–adventitial interface approximately 1.5 cm proximal to the carotid bifurcation. The IMT measurements were obtained from four contiguous sites at 2-mm intervals, and the

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