

Original Research Article

Profile of microalbuminuria in non-diabetic myocardial infarction

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ABSTRACT

Background: Urinary albumin excretion between 20 to 200 mg per litre is defined as microalbuminuria (MAU). MAU acts as a marker for endothelial cell destruction and is associated with atherosclerosis in both diabetics and non-diabetics. This study aimed to assess the MAU prevalence in nondiabetic patients with myocardial infarction.

Methods: A cross-sectional study was conducted at tertiary care hospital. Among non-diabetic patients with myocardial infarction. The study was conducted from October 2019 to March 2020. All patients were subjected to complete physical examination, electrocardiography (ECG) and echocardiography. Quantitative determination of MAU was done in a urine sample. Diagnostic coronary angiogram was performed for all patients. Appropriated statistical tests were applied.

Results: Among 80 study participants, 73.75% were men, and 26.25% were women. Smoking habit was present among 60%, and 56.25% were hypertensive. The prevalence of microalbuminuria was 27.5%. A statistically significant difference was seen between TIMI scoring and presence of MAU ($p < 0.001$). The difference in vessels type between the microalbuminuria status was found to be significant ($p = 0.005$).

Conclusions: Evidence from this study shows that the presence of MAU had a strong association between myocardial infarction and its application as a risk factor of cardiovascular diseases in general non-diabetic population proves practical.

Keywords: Microalbuminuria, Non-diabetic, Myocardial infarction

INTRODUCTION

Worldwide, in cardiovascular disease, patient microalbuminuria (MAU) occurs commonly. MAU is urinary excretion of albumin at a rate of 20 to 200 mg/l or 30 to 300 mg/day. It is also conventionally defined as a rate which lows to is detected by the routine dipstick examination.¹ In routine practice, MAU is used as a proxy marker for the renal damage in diabetes and hypertensive patients. MAU is largely associated with age, diabetes status of the individual, smoking habit, hypertension, dyslipidaemia and poor physical activity.²⁻⁴ Urinary excretion of albumin less than 30 mg/day is considered normal in a healthy individual. The rate of excretion is known to increase in conditions like physical activity and

exercise, high oral intake of protein, urinary tract infections and pregnancy. Twenty-five per cent higher excretion occurs in the day compared to night, and there can be 40% day to day variation. Albumin excretion more than 300 mg/day indicates nephropathy.

MAU is associated with high risk for renal and cardiovascular impairments in diabetes patients.⁵⁻⁹ hypertensive individuals and elderly population.¹⁰⁻¹⁴ With this literature, there is a lacuna in the research regarding the significance of MAU in nondiabetic and non-hypertensive individuals. This lacuna has to be filled in order to consider the potential significance of MAU as a risk factor in the general population. Research observations over the past years have shown that MAU is

not only the predictor of diabetic complications but also acts as an independent predictor of cardiovascular disease status, which cause atherosclerosis.¹⁵ The prevalence of MAU among non-diabetic individuals varies from 5 to 40%. A wide range of factors are responsible for this high variability: quality of blood pressure control and associated lipid abnormalities, patient selection and inclusion criteria biases such as age, race, coexisting renal disease, techniques used for detection of MAU, sampling size, and day to day variability of albumin excretion.¹⁶

The aim of this present study was to find the prevalence of MAU among non-diabetes individuals who has a myocardial infarction.

METHODS

A cross-sectional study was conducted among 80 myocardial infarction patients admitted at tertiary care hospital from October 2019 to March 2020. Informed written consent was obtained from the study participants, and confidentiality was maintained. Diabetics, patients with urinary tract infections, chronic stable angina and positive MAU through urine dipstick were excluded from the study. A complete in-depth medical history was collected from the patients using a standardized proforma, and thorough clinical examination was conducted with a special interest in the cardiovascular system. Laboratory investigations, electrocardiography (ECG) and echocardiography, were performed for all patients. For the assessment of MAU morning urine sample was obtained from each patient. Centrifugation of the sample was done, and the precipitate was discarded. The supernatant fluid was stored at -20 degree celsius. Immunometric enzyme immunoassay was done for quantitative assessment of MAU. Values above 30 mg/day were considered as pathological. All patients were subjected to coronary angiography, and lesions, more than 70% stenoses were considered pathological.

TIMI risk scoring was used for all participants. This score identifies seven independent risk factors for myocardial infarction: age older than 65 years; equal or more than 3 Coronary artery disease (CAD) risk factors; documented CAD at catheterization (more than 50% stenosis); ST-segment deviation more than 0.5 mm from pre-stenting ECG; equal or more than two anginal episodes in prior 24 hours; aspirin within prior week; and elevated cardiac biomarkers. Each factor was given one point, and score ranges from 0 to 7.¹⁷

Statistical methods

Demographic variables like age, gender, smoking status, ischemic ECG, ischemic ECHO finding and TMI score were considered as outcome variables. MAU was considered as Primary explanatory variable. Descriptive analysis was carried out by mean and standard deviation for quantitative variables, frequency and proportion for categorical variables.

All quantitative variables were checked for normal distribution within each category of an explanatory variable by using visual inspection of histograms and normality Q-Q plots. Shapiro wilk test was also conducted to assess normal distribution. Shapiro wilk test $p > 0.05$ was considered as a normal distribution.

For normally distributed quantitative parameters, the mean values were compared between study groups using Independent sample t-test (2 groups).

Categorical outcomes were compared between study groups using Chi square test /Fisher's Exact test (if the overall sample size was < 20 or if the expected number in any one of the cells is < 5 , Fisher's exact test was used).

$P < 0.05$ was considered statistically significant. IBM Statistical package for social sciences (SPSS) version 22 was used for statistical analysis.

RESULTS

A total of 80 subjects were included in the final analysis.

Table 1: Demographical characteristics of study population (n=80).

Variables	Number (%)
Age (mean \pm SD)	56.51 \pm 12.85
Men	59 (73.75)
Women	21 (26.25)
Smoker	48 (60)
Hypertension	45 (56.25)
Ischemic ECG changes	67 (83.75)
Ischemic Echo finding	59 (73.75)
Microalbuminuria	
Negative	58 (72.5)
Positive	22 (27.5)

The mean age was 56.51 \pm 12.85 years in the study population. Among the study population, 59 (73.75%) were men, and the remaining 21 (26.25%) were women. Among the study population, 48 (60%) were smokers, 45 (56.25%) had hypertension, 67 (83.75%) participants had ischemic ECG changes, and 59 (73.75%) participants had ischemic ECHO finding. Among the study population, 22 (27.5%) participants had MAU positive. (Table 1)

The mean age (in years) of people with microalbuminuria was 56.51 \pm 11.05, and it was 58.02 \pm 11.56 in people without MAU. The mean difference between two groups was statistically not significant ($p = 0.509$). The difference in gender between the MAU status is found to be insignificant with a $p = 0.659$. The difference in smoking status between the MAU status is found to be insignificant with a $p = 0.683$. The difference in hypertensive between the MAU status is found to be insignificant with a $p = 0.231$. The difference in ischemic ECG between the MAU status is found to be insignificant with a $p = 1.000$.

Table 2: Comparison of demographic parameters between microalbuminuria status (n=80).

Demographic parameters	MAU positive (n=22) N (%)	MAU negative (n=58) N (%)	P value
Age (mean±SD)	56.51±11.05	58.02±11.56	0.599
Gender			
Men	17 (77.27)	42 (72.41)	0.659
Women	5 (22.73)	16 (27.59)	
Smoking status			
Smoker	14 (63.64)	34 (58.62)	0.683
Non- Smoker	8 (36.36)	24 (41.38)	
Hypertensive status			
Hypertensive	10 (45.45)	35 (60.34)	0.231
Non-hypertensive	12 (54.55)	23 (39.66)	
Ischemic ECG			
Ischemic changes	19 (86.36)	48 (82.76)	1.000 *
Non- Ischemic	3 (13.64)	10 (17.24)	
Ischemic Echo finding			
Ischemic findings	20 (90.91)	39 (67.24)	0.032
Non-ischemic	2 (9.09)	19 (32.76)	
TIMI score			
≤2	1 (4.55)	17 (29.31)	<0.001
3	2 (9.09)	28 (48.28)	
4	10 (45.45)	9 (15.52)	
5	8 (36.36)	2 (3.45)	
≥6	1 (4.55)	2 (3.45)	
Vessels			
Single vessels	3 (13.64)	29 (50)	0.005
2 vessels	9 (40.91)	19 (32.76)	
3 vessels	10 (45.45)	10 (17.24)	

*Fisher exact p value.

Table 3: Relation between MAU level and the number of diseased coronary vessels at cutoff point=29 µg/ml.

Number of diseased coronary vessels at cutoff point=29 µg/ml	Below N (%)	Above N (%)
1 VD	2 (100)	0 (0)
2 VD	5 (71.43)	2 (28.57)
3 VD	1 (7)	13 (93)

* No statistical test was applied-due to 0 subjects in the cell

The difference in ischemic ECHO finding between the MAU status is found to be significant with a p=0.032. The difference in TIMI score between the MAU status is found to be significant with a p<0.001. The difference in vessels type between the MAU status is found to be significant with a p=0.005 (Table 2).

Among the single vessel, double vessel diseases, the majority reported below the cutoff point of 29 µg/ml. 100% in single and 71.43% in the double vessel. In triple vessel diseases, majority 93% reported above the cutoff point of 29 µg/ml (Table 3).

DISCUSSION

Many studies show that MAU as a risk factor for cardiovascular diseases in diabetic patients.¹⁸⁻²⁰ Cardiovascular disease leads to the acute coronary syndrome as unstable angina (UA) and non-ST-segment elevation MI (NSTEMI). In this study, MAU was detected in 22 out of 80 non-diabetic patients. Similar findings were seen in a study conducted by Klausen et al, whose conclusion was that MAU acts as a strong determinant of coronary artery disease.²¹ The association determined by Klausen et al was found to be independent of age, sex, diabetes status, hypertension status, renal function and lipid profile. In this present study, we found no significant correlation of MAU with age, sex, smoking status, hypertensive status, ischemic ECG and ECHO findings. Among these, smoking is a recognised cardiovascular risk factor. In our study the MAU was found to be more common in non-smokers than smokers. This is in contrary to the results obtained by Guizer.²² Many studies in the past have been able to establish an association between hypertension status and MAU, however, we were not able to observe a similar association in our study. A similar result was found out by Klausen that MAU predicted cardiovascular diseases irrespective of the hypertension status of the patient.²¹

Several studies done among non-diabetic participants revealed that MAU was an independent predictor of cardiovascular morbidity and mortality. In this present study, MAU was observed more among the patients with ischaemic ECG and echocardiographic changes. This finding was similar to the study Diercks et al., echo described increased mortality in the subgroup of subjects with ischaemic changes.²³ It has been observed that the presence of MAU indicates an augmentation in vascular endothelial permeability which is not restricted to renal vessels. This might advance foam cell formation and atherogenesis by increased leakage of lipoprotein particles into the vessel wall, and increased transcapillary albumin excretion rate, an increased plasma level of Von Willebrand factor (VWF), and an attenuated endothelium-dependent response to vasodilator stimuli in subjects with MAU.²⁴ Hence, patients with ischemic changes adding up to MAU could have a greater than before the risk of augmented development of atherosclerosis and rise in mortality. The efficiency of MAU in predicting cardiovascular risk is not only restricted to the high-risk population but also applies to the low-risk population. In a study by Hillege et al., findings showed that MAU predicted CVD and non-CVD mortality in a general population.²⁵ Patients with a high risk of adverse cardiovascular events as calculated by the TIMI risk score were more likely to be MAU-positive compared to patients

with low cardiovascular risk. A significant correlation was observed between MAU and the severity of the coronary artery. Patients with double or triple vessels disease had a higher level of MAU than patients single-vessel disease.

This study has given evidence that MAU as a predictor of cardiovascular risk among the non-diabetic population.

CONCLUSION

MAU can be done at a low-cost setting which can be used as a non-invasive technique to assess the cardiovascular disease risk factor. The application of MAU into the non-diabetic general population can improve the detection rate of cardiovascular diseases.

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