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# Correlation of microalbuminuria and serum CPK–MB in non–diabetic, non – hypertensive acute myocardial infarction patients

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**Abstract:** Microalbuminuria is known to occur independently in hypertension and diabetes mellitus. The cardiovascular implication of microalbuminuria (MA) in the non diabetic, non hypertensive patients has received focus recently yet little is known on the prognostic significance and pathophysiological mechanisms. The aim of our study was to assess occurence of microalbuminuria in non-diabetic, non hypertensive patients suffering from Acute Myocardial Infarction (AMI) and also to evaluate the correlation between Microalbuminuria and Creatine Phosphokinase - MB (CPK - MB) in non-diabetic, non hypertensive AMI patients. This case control study was conducted on 100 subjects (50 cases, 50 controls). The subjects were evaluated for urinary MA and serum CPK – MB. Mean level of MA and CPK – MB was significantly higher in cases as compared to controls. There was a weak positive correlation between MA and CPK – MB in cases.

**Keywords:** Microalbuminuria, CPK- MB, myocardial infarction, non diabetic, non hypertensive.

## Introduction

Myocardial Infarction is a common presentation of Atherosclerotic coronary vascular disease (ASCVD). The WHO estimated in 2004, that 12.2% of worldwide deaths were from ischemic heart disease, with it being the leading cause of death in high- or middle-income countries and second only to lower respiratory infections in lower income countries [1]. Worldwide, more than 3 million people have STEMI and 4 million have NSTEMI a year [2]. STEMI occurs about twice as often in men as women [3]. In India, IHD had become the leading cause of death in 2004, accounting for 1.46 million deaths (4%

of total deaths) and deaths due to IHD were expected to double during 1985 – 2015 [4]. As Indians are at a high risk for the developing IHD, it clearly demands more studies to understand pathophysiology, so that an effective prevention can be made possible.

Microalbuminuria has been reported as an independent predictor of cardiovascular disease in patients with diabetes [5] and hypertension [6], however, this association has been reported in the absence of those conditions [7]. Several studies suggest that microalbuminuria could be a surrogate for endothelial damage because it has been correlated with earlier stage of atherosclerosis [8].

In person with MA without DM, an increase in vascular permeability is caused by alterations in the extracellular matrix, this contributes to the development of endothelial dysfunction which promotes lipid influx into the vessel wall causing atherosclerotic changes [9].

The measurement of CK-MB level has long been used for the diagnosis of AMI, reaching their respected peak activities within 4-8 hours after the admission with acute symptoms [10].

The present study was undertaken to investigate the correlation of microalbuminuria with CK-MB. This would increase the understanding of the pathogenesis of the diseases and may help in prevention, diagnosis and management of MI.

**Materials and methods**

This was a prospective case control study carried out in the department of Biochemistry and Intracardiac care unit (ICCU) in Rohilkhand Medical College and Hospital, Bareilly, Uttar Pradesh contains 50 patients were admitted to the coronary care unit with the diagnosis of AMI between 15 January to 15 December 2015 with an equal number of age and sex matched controls were also included.

Patients with a history of diabetes, hypertension, systemic infection, urinary tract infection, arthritis, nephropathy with deranged KFT were also excluded from the study. Diagnosis of AMI was made based on detailed medical history, clinical examination especially cardiovascular system, ECG findings and level of cardiac markers. The study was approved by local ethical committee and written informed consent was obtained from all patients.

For the estimation of CK-MB level in serum 2 ml venous blood sample was collected in a serum separating tube under aseptic precaution, allow to clot then centrifuged to separate the serum and analyzed by Immunoinhibition/ Mod. IFCC method on ERBA EM 360 auto analyzer. Random mid – stream urine sample was collected in a sterile container for the determination of urinary microalbumin. Macroalbuminuria was excluded using ordinary dipstick testing. Negative samples were estimated for microalbuminuria by Turbidimetric Immunoassay with Erba Chem 5 plus analyzer. If required, the sample were stored at 2-

**Result**

The study included a total of 100 subjects out of which 50 were cases of AMI and another 50 were apparently healthy controls. Non – diabetic, non –hypertensive AMI group with mean age of 52.78 ± 8.50 years and in apparently healthy controls with a mean age of 51.43 ± 7.25 years (Table 1).

As shown in Table – 2, the controls had a mean CK – MB ± standard deviation (SD) of 14.12 ± 3.16 whereas, in cases of AMI, the mean CK-MB level was 77.88 ± 68.37 U/L. The CK-MB level between the non – diabetic,

non – hypertensive AMI patients was statistically significant ($p < 0.001$) when compared with controls.

Urinary microalbumin levels were also determined for both the cases and controls. The mean urinary microalbumin level in non – diabetic, non – hypertensive AMI cases was 94.12 ± 70.76 mg/L. The mean urinary microalbumin excretion in controls was 12.42 ± 4.50 mg/L. this increased excretion of microalbumin in AMI patients was clinically and statistically significant ($p < 0.001$) when compared with controls. (Table - 3)

The coefficient of correlation between urinary MA and CPK – MB was 0.101. However the correlation was not statistically significant as the p value was > 0.05 (Table - 4)

**Discussion**

Many studies have been done to establish microalbumin excretion in the urine in non – diabetic, non hypertensive patients with AMI. Haffner et.al considered MA to be arker of cardiovascular risk in non – diabetic patients [11]. The result of our study also indicate that there is highly significant microalbuminuria in non – diabetic, non – hypertensive AMI patients. The level of microalbuminuria in acute myocardial infarction patients in our study was highly significant ($p < 0.001$) when compare to the controls. We however did not find any significant correlation between CPK –MB and microalbuminuria of the cases in our study group. This does not agree with Gosling et. al., study who found significant correlation between microalbuminuria and CPK – MB remarked that MA can predict the area of infarction [12]. In a recent study microalbuminuria was considered as a marker for cardiovascular diseases [13]. However in this study microalbuminuria was found to be significantly elevated in the study group taken as a whole but not in the non – hypertensive group taken separately. Our study is not in agreement with this because we found significant MA in AMI patient who were non – hypertensive. Our study agrees with Klauser et. al., where they found microalbuminuria to be an independent risk factors of cardiovascular diseases and death, independent of renal insufficiency, diabetes and hypertension [14]. We excluded not only hypertension but also patients with renal insufficiency. Thus the major pathophysiologic cause of microalbuminuria in this patient group remains to be unrevealed but an inflammatory response remains an important suspect. Inflammation leads to an increased capillary permeability to proteins. This effect is amplified by the kidneys and manifests as MA. However a tubular dysfunction leading to decreased tubular reabsorption cannot be ruled out.

| | Cases | Controls |
|---|---|---|
| Age in years | 52.78± 8.50 | 51.43 ± 7.25 |
| p value > 0.05 when compared with controls | | |
| Mean age (mean ± SD) among cases of AMI and healthy controls | | |

Table 1

| | Cases | Controls |
|---|---|---|
| CK – MB (IU/L) | 77.88 ± 68.37 | 14.12 ± 3.16 |
| P value < 0.001 when compared with controls | | |
| Comparision of CK – MB between cases and controls (expressed as mean ± S.D) | | |

Table 2

| | Cases | Controls |
|---|---|---|
| Urinary Microalbumin level (mg/L) | 94.12 ± 70.76 | 12.42 ± 4.50 |
| p value < 0.001 when compared with controls | | |
| Urinary microalbumin levels in case and controls | | |

Table 3

| | |
|---|---|
| Correlation between MA and CPK –MB | 0.101 |
| p value for r | p > 0.05 |

Table 4

**Conclusion**

In our study we found a significantly high microalbuminuria in nondiabetic, non hypertensive acute myocardial infarction patients. Since test for microalbuminuria is simple and relatively inexpensive, we propose the use of test for microalbuminuria as a biochemical parameter to predict future increased risk for myocardial infarction in non – diabetic, non – hypertensive patients. However it is not possible to explain because effect relationship in a case control study and also the pathogenesis of microalbuminuria in the above patient group remains to be elucidated, for which longitudinal studies with larger sample size are warranted.

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