

# Microalbuminuria in Non-diabetic, Non-hypertensive Myocardial Infarction in South Indian Patients with Relation to Lipid Profile and Cardiac Markers

SATHISHA T.G., MANJUNATHA GOUD B.K., AVINASH S.S., JEEVAN SHETTY, OINAM SARSINA DEVI, DEVAKI R.N.

## ABSTRACT

**Introduction and Objective:** The present study was carried out to compare the levels of urinary microalbumin, the lipid profile, the cardiac enzymes and troponin T in non-diabetic, non-hypertensive myocardial infarction patients and in healthy controls and to know the possible relationship between microalbuminuria and the lipid profile markers and the cardiac enzymes in myocardial infarction in patients from the southern part of India.

**Materials and Methods:** This study was carried out in 35 non-diabetic, non-hypertensive myocardial infarction patients and in 35 healthy, age matched controls. Urinary albumin, urinary creatinine, creatinine kinase-MB fraction (CK-MB), AST, LDH, troponin I and the lipid profile parameters were estimated by using an automated analyzer. The total cholesterol/HDL cholesterol ratio and the LDL cholesterol/HDL cholesterol ratio

were also calculated by using the total cholesterol and the LDL and HDL values.

**Results:** There was a significant increase in the levels of total cholesterol, LDL cholesterol, total cholesterol/HDL cholesterol ratio, LDL cholesterol/HDL cholesterol ratio, microalbumin, cardiac enzymes and troponin I ( $p < 0.001$ ) in patients with myocardial infarction as compared to those in the healthy controls. On applying Pearson's correlation, the microalbumin levels were found to correlate positively with the LDL cholesterol levels ( $p = 0.010$ ,  $r = 0.952$ ) and Troponin I ( $p = 0.025$ ,  $r = 0.885$ ) and this was found to be statistically significant.

**Conclusion:** Microalbuminuria can be used as a predictor for the early detection of cardiovascular and renal changes along with the lipid profile markers in the general population to prevent the mortality and morbidity which are associated with acute myocardial infarction

**Key Words:** Microalbuminuria, Myocardial infarction, lipid profile

## INTRODUCTION

Acute myocardial infarction (AMI) is one of the commonest diseases amongst hospitalized patients in industrialized countries. The mortality rate of AMI is approximately 30% and for every 1 in 25 patients who survive the initial hospitalization, dies in the first year after AMI [1]. Indians are four times more prone to AMI as compared to the people of other countries due to a combination of the genetic and lifestyle factors that promote metabolic dysfunction. The risk of cardiovascular disease is predicted by various factors such as age, sex, smoking, hypertension and dyslipidaemia. In most of the cases, the cardiovascular changes are detected only after a person exhibits the classical symptoms and the signs of acute myocardial infarction. This clearly indicates the need for a marker which can detect the risk of cardiovascular changes in the early stages, so that an effective prevention can be made possible.

Microalbuminuria (MA) is defined as the urine albumin to the urine creatinine ratio (UACR) of 30-300 mg/G of creatinine [2]. Microalbuminuria is considered to be a predictor of early renal damage in patients with diabetes mellitus. Previous studies have shown that MA is associated independently with cardiovascular morbidity and mortality in diabetic and hypertensive patients [3-8]. Accordingly, the national and international guidelines recommend the screening for MA in patients with diabetes or hypertension [9-11].

It is less clear, however, whether the screening for MA should be extended to the general population or to individuals who are at a

lower risk for cardiovascular disease (CVD), such as nondiabetics or non-hypertensives. Investigators have postulated that MA may be a marker of risk, even in apparently healthy people, because it reflects vascular damage in the kidneys and in the systemic endothelial dysfunction [12,13, 14]. As Indians are at a high risk for the development of cardiovascular events, it clearly demands the importance of cost-effective markers for the detection of the early cardiovascular changes.

The present study was undertaken to measure the levels of microalbumin with the cardiac enzymes and the lipid profile parameters in non-diabetic, non-hypertensive acute myocardial infarction patients, to know the relationship between MA and the lipid profile parameters and to compare their levels with those of the healthy controls.

## MATERIALS AND METHODS

This study was carried out on patients who were admitted to the Cardiology Department, Kasturba Hospital, Manipal. The study group consisted of 35 patients with AMI and an equal number of age and sex matched controls were also included. The mean age of the patients was  $59 \pm 10$  years and that of the controls was  $52 \pm 10$  years. They were diagnosed to have AMI according to the clinical criteria; chest pain which lasted for up to 3 hours, ECG changes (ST elevation of 2mm or more in at least two leads), with elevated cardiac markers. Patients with a history of diabetes,

hypertension, systemic infection, urinary tract infection, arthritis, nephropathy (serum creatinine >1.0mg/dl), AMI following surgery and major trauma were excluded from the study.

After obtaining an ethical clearance from the institutional ethical committee, informed consent was obtained from all the subjects who were involved in the study. 5ml of blood samples were drawn into plain vacutainers from the antecubital vein of all the patients. Similarly, samples were also obtained from age and sex matched healthy controls. Early morning mid stream urine samples were collected under strict aseptic precautions. Fasting lipid profile, creatinine kinase MB isoform, aspartate transaminase, lactate dehydrogenase, troponin I and microalbumin levels were determined by using an automated analyzer, Hitachi 912, after proper processing and in accordance with the manufacturer's instructions. The total cholesterol: HDL cholesterol ratio was calculated by dividing the HDL cholesterol values from the total cholesterol values. The LDL: HDL ratio was calculated by dividing the HDL values from the LDL values.

## STATISTICAL ANALYSIS

The results were expressed as mean  $\pm$  standard error of the mean (SEM). p values which were <0.05 was considered to be statistically significant. Statistical analysis was performed by using the Statistical Package for Social Sciences (SPSS-16, Chicago, USA). The 'independent sample t test' was used to compare the mean values. Pearson correlation was applied to correlate between the parameters.

## RESULTS

As depicted in [Table/Fig-1], there was a significant increase in the levels of total cholesterol, LDL cholesterol, total cholesterol/HDL cholesterol ratio, LDL cholesterol/HDL cholesterol ratio, microalbumin, cardiac enzymes and troponin I ( $p < 0.001$ ) in patients with myocardial infarction as compared to those in the healthy controls. On applying Pearson's correlation, the microalbumin levels were found to correlate positively with the LDL cholesterol levels ( $p = 0.010$ ,  $r = 0.952$ ) and Troponin I ( $p = 0.025$ ,  $r = 0.885$ ) and this was found to be statistically significant.

|                           | Controls ( n=35)   | Cases ( n=35)         |
|---------------------------|--------------------|-----------------------|
| Age (Yrs)                 | 52 $\pm$ 10        | 59 $\pm$ 10           |
| Total Cholesterol (mg/dl) | 160.54 $\pm$ 24.22 | 209.57 $\pm$ 33.04*** |
| TG(mg/dl)                 | 148.98 $\pm$ 29    | 147.66 $\pm$ 57       |
| HDL (mg/dl)               | 39.66 $\pm$ 12.04  | 38.66 $\pm$ 12.45     |
| LDL(mg/dl)                | 95.03 $\pm$ 21.67  | 145.17 $\pm$ 30.88**  |
| VLDL(mg/dl)               | 30.42 $\pm$ 7.02   | 31.98 $\pm$ 11.08     |
| TC/HDL                    | 4.45 $\pm$ 1.6     | 5.75 $\pm$ 1.3*       |
| LDL/HDL ratio             | 2.6 $\pm$ 1.2      | 4 $\pm$ 1.2*          |
| CK-MB (U/L)               | 11 $\pm$ 2         | 78 $\pm$ 49*          |
| LDH (U/L)                 | 268 $\pm$ 53.88    | 1006 $\pm$ 526*       |
| Tropinin I                | 0.23 $\pm$ 0.007   | 15.98 $\pm$ 17.03*    |
| AST(U/L)                  | 24.88 $\pm$ 6      | 90 $\pm$ 68.03*       |
| MA (mg/g of creatinine)   | 19.24 $\pm$ 8      | 166 $\pm$ 89.4*       |

[Table/Fig-1: Lipid profile, cardiac enzymes and microalbumin levels in controls and cases (Values expressed in mean  $\pm$  SD).

\*P<0.05

\*\*P<0.01

\*\*\*p<0.001 compared to healthy controls

## DISCUSSION

In line with previous studies, we found a significant rise in the cardiac enzymes CK-MB, AST, LDH and troponin I in AMI patients. After infarction, the enzymes leak from the cytosol due to myocardial tissue injury. We found a significant increase in the total cholesterol, LDL cholesterol, the TC/HDL ratio and the LDL/HDL ratio. LDL has an important role in the transport of cholesterol from the liver to the peripheral tissues. Previous studies have shown that elevated LDL levels increased the risk for the development of atherosclerosis. LDL cholesterol undergoes oxidation due to various factors and oxidized LDL plays a key role in the initiation and development of atherosclerotic plaque in the coronary arteries [15]. An original paper described that the increase in the LDL/HDL ratio was a better indicator of the atherogenic tendency [16] and this was in agreement with our study, which also found an increase in the LDL/HDL ratio. The total cholesterol/HDL cholesterol ratio can further confirm the atherogenic risk, which was evident in our study group in comparison with the healthy controls.

Previous studies have shown the increase in the microalbumin levels in diabetic and/or hypertensive patients and also in non diabetic and non hypertensive patients. Several studies have shown that microalbuminuria is a good predictor of early renal damage and this helps to initiate preventive measures for renal damage [17]. In agreement with previous studies, we found a significant increase in the microalbumin levels in patients with AMI as compared to those in healthy controls. This may be due to dyslipidaemia induced renal glomerular damage or in some cases, due to ischaemia and reperfusion.

In comparison to earlier studies, we found a positive correlation between microalbuminuria and cardiac troponin I, which was statistically significant. A positive correlation may indicate that the extent of microalbuminuria is proportional to the size of the infarct and the severity of the coronary artery damage. Similarly, we found a positive correlation between microalbuminuria and LDL cholesterol and total cholesterol, which was statistically significant. A positive correlation between the atherogenic lipid profile parameters and microalbuminuria clearly indicated the role of the latter in the development of atherosclerosis. However, the exact mechanism of the accelerated atherosclerosis in microalbuminuria is not clear. Abnormal vasodilatation, endothelial dysfunction, inflammation and abnormal coagulation may be involved in this process.

In conclusion, microalbuminuria can be used as a predictor for the early detection of cardiovascular renal changes along with the lipid profile markers in the general population, to prevent the mortality and morbidity which are associated with acute myocardial infarction. However, due to its non-specific nature, there should be proper selection criteria, so that it can be used as an effective screening tool in the general population.

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**AUTHOR(S):**

1. Dr. Sathisha T.G.
2. Dr. Manjunatha Goud B.K.
3. Dr. Avinash S.S.
4. Dr. Jeevan Shetty
5. Dr. Oinam Sarsina Devi
6. Dr. Devaki R.N.

**PARTICULARS OF CONTRIBUTORS:**

1. Department of Biochemistry, KMC, Manipal University, Manipal, Karnataka, India.
2. Department of Biochemistry  
Ras Al Khaimah Medical and Health Sciences University,  
Ras Al Khaimah, U.A.E
3. Department of Biochemistry, FMMC, Mangalore,  
Karnataka, India
4. Department of Biochemistry, KMC, Manipal University,  
Manipal, Karnataka, India.

5. Department of Nursing, Vidya Nursing College Udupi,  
Karnataka, India.
6. Department of Biochemistry, JSS Medical College, JSS  
University.  
Mysore, India.

**NAME, ADDRESS, TELEPHONE, E-MAIL ID OF THE CORRESPONDING AUTHOR:**

Dr.B.K.Manjunatha Goud  
Asistant Professor of Biochemistry  
Ras Al Khaimah Medical and Health Sciences University, Ras Al  
Khaimah, U.A.E-11172  
Mobile:+971554195204  
E-Mail:drmanjunathag@gmail.com

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