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Study of Microalbuminuria and Serum C Reactive Protein in Non hypertensive Acute Myocardial Infarction

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HUMAIRA KHAN [1]
ARCHANA DHAVALE [2]
AMOL R SHINDE [3]
A M SIDDIQUI [4]
1,4 Associate Professor
2 Junior Resident cum Tutor
3 Assistant Professor
Department of Biochemistry, Dr. Shankarrao Chavan Govt. Medical College, Nanded

Corresponding Author:

Dr. Archana Dhavale
23, Mahsool Nagar, Taroda BK road
Nanded-431605 (Maharashtra, India)
+91-7709299348
dr.archanadhavale@gmail.com

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Abstract:

Acute myocardial infarction is one of the commonest diseases and the leading cause of death for both men and women all over the world. Therefore this study was undertaken to assess microalbuminuria and serum C reactive protein (CRP) in non hypertensive AMI patients. The study was conducted in a group of 100 subjects consisting of 50 age and sex matched healthy control and 50 diagnosed cases of non hypertensive acute myocardial infarction (AMI) patients. There was a significant increase in level of microalbuminuria and serum CRP in non hypertensive AMI patients compared to healthy control. This study shows that microalbuminuria and serum CRP are useful biochemical parameter in non hypertensive acute myocardial infarction patients.

Keywords : acute myocardial infarction, AMI, C reactive protein, CRP, microalbuminuria

Abbreviations: CRP- C reactive protein, AMI- Acute myocardial infarction, MA- Microalbuminuria, LDL- Low density lipoprotein

Introduction:

Excretion of albumin in urine, in the range of 30-300 mg/day is called microalbuminuria. This range of albumin in urine cannot be detected by routine urine test.

Acute myocardial infarction is one of the commonest diseases in hospitalised patients in industrialised countries. In India, coronary artery disease is equally important and its incidence has increased in last 2 decades. Microalbuminuria was studied as a predictor of incipient nephropathy and coronary vascular disease in the diabetic patients.[1]

Microalbuminuria has also been implicated as a sensitive indicator of non renal disease. Microalbuminuria occurs in benign essential hypertension.[2] It was postulated that the microalbuminuria occurring in benign essential hypertension is due to increased transglomerular passage of albumin. It become evident that microalbuminuria is associated with increased risk of cardiovascular disease even in the non diabetic patients.[3,4] The association of microalbuinuria with cardiovascular disease is very important

owing to burden of cardiovascular disease morbidity and mortality.

Inflammation is an important factor in acute myocardial infarction. Myocardial infarction in human provokes an acute phase response where CRP, the classical acute phase plasma protein level is elevated, starting within 4-6 hrs of onset of symptoms. The human CRP and complement activation are the major mediators of ischemic myocardial injury and identify them as therapeutic targets in coronary hearts disease.

In this study an attempt has been made to estimate microalbuminuria and serum CRP level in non hypertensive acute myocardial infarction patient and to compare their levels with healthy control.

Material and Methods:

A present study was conducted in Department of Biochemistry, Dr. Shankarrao Chavan Government medical college and Hospital, Nanded. The study was approved by institutional Ethics Committee for research work.

The study is conducted in a group of 100 subjects consisting of 50 age and sex matched healthy control and 50 diagnosed cases of non hypertensive acute myocardial infarction patients. The sample size was calculated using statistical software.

The patients with history of diabetes, hypertension, urinary tract infection, inflammatory conditions like rheumatoid arthritis, nephropathy at the time of admission to the ICU, myocardial infarction following surgery and major trauma were excluded from the study as these conditions will affect independently the parameters to be estimated.

2 ml venous blood was collected in plain bulb for estimation of CRP. For estimation of microalbuminuria 24 hrs urine was collected in plastic bottle using thymol as a preservative. The subjects were asked to void the early morning urine sample and thereafter the urine was collected including the next day early morning sample. Albumin in urine was estimated by Pyrogallol red end point method on Erba Chem7 semi-autoanalyzer. Estimation of CRP was done on serum sample by immunoturbidometry method on Erba Chem7 semi-autoanalyzer.

The results were presented as mean±SD. Significance is assessed at 5% level of significance. For determination of p-value unpaired t-test was used on Graph pad prism. For all the tests, p-value of 0.05 or less was considered statistically significant.

Results:

The control group consisted of 44 males and 6 females with a mean age 53.96 ± 1.66 years. The mean age in non hypertensive AMI patients was 54.44 ± 1.72 years. The gender distribution was 45 males and 5 females. This is presented in **Table 1.**

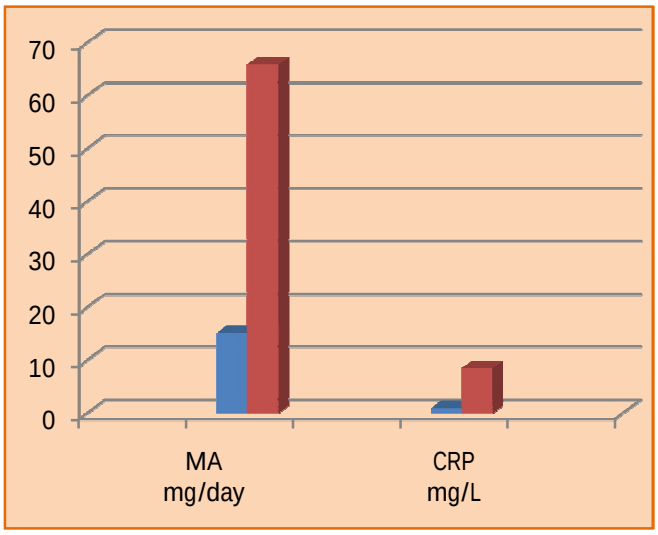
Table 1: Age and Gender distribution in control and cases

Parameter	Control	Non hypertensive AMI
Male	44	45
Female	6	5
Mean age ± SD	53.96 ± 1.66	54.44 ± 1.72

Table 2: Comparison of MA and CRP between controls and hypertensive AMI

Sr. no.	Parameters	Control	Non hypertensive AMI	P value
1	MA (mg/day)	15.16 ± 0.36	65.88 ± 8.41	<0.0001
2	CRP (mg/L)	0.99 ± 0.07	8.47 ± 0.45	<0.0001

Figure 1: Bar diagram showing comparison of parameters between controls and non hypertensive AMI



As depicted in Table 2 and Figure 1, there was an increase in level of microalbuminuria and serum CRP in non hypertensive AMI patients compared to healthy control. It is found that the difference between the groups is significant with respect to all the parameters.

Discussion:

In our study there is highly significant microalbuminuria in non hypertensive AMI patients when compared to that of controls. This is in accordance with study of Hillege et al[5], Klauser et al[6] who also found microalbuminuria in non hypertensive group.

Microalbuminuria is non-specific for acute myocardial infarction. Any inflammatory condition, medical or surgical can cause microalbuminuria.[7] Even though it is non-specific; the marker value of microalbuminuria in acute myocardial infarction has been considered. Our study agrees with some of the recent studies like the Klauser et al[6] study, where they found microalbuminuria to be an independent risk factor of cardiovascular diseases.

All the cases in the present study had a normal renal function. Microalbuminuria in these patients was therefore not related to renal dysfunction. Our study in this respect agrees with the views of Gosling P[2], who considered it to be a sensitive indicator of non renal disease.

In our study we found microalbuminuria to be related to acute myocardial infarction independent of renal disease and diabetes. In this respect our study agrees with some of the recent studies.[6] We excluded not only diabetes mellitus but also renal insufficiency. We therefore excluded any gross kidney damage, diabetic causes of microalbuminuria. Thus the major pathophysiologic cause of microalbuminuria in our clinical setting seems to be a systemic inflammatory response. This leads to an increased capillary permeability to proteins. This effect is amplified by the kidneys and manifests as microalbuminuria. However a tubular dysfunction leading to decreased tubular reabsorption cannot be ruled out. It is therefore suggested that more such studies be taken up in this clinical setting with a larger sample size to elucidate the exact pathophysiology.

In our study there is highly significant increase in serum CRP level in non hypertensive AMI patients when compared to that of controls. The result of our study is consistent with finding in previous studies by Ridker et al[8], Pearson et al[9], Pasupathi et al[10], Nelakantan et al[11], Mishra CK et al [12].

The principle cause of elevated levels of CRP seems to be the persistent and subclinical chronic inflammation leading to atherosclerosis. There is growing evidence that CRP is not merely a marker of inflammation but also plays an active role in atherogenesis.

Pepys and Hirschfield[13] suggested a direct role for CRP in the pathogenesis of atherosclerosis and post myocardial infarction inflammation. CRP, present in atherosclerotic plaques, binds to oxidized LDL and enhances of the ability of macrophages to phagocytose LDL and form foam cells through the CRP receptor CD32. Furthermore, the presence of CRP within most atherosclerotic plaques and all AMI lesions, coupled with binding of CRP to lipoproteins, and its capacity for pro-inflammatory complement activation, suggest that CRP may contribute to the

pathogenesis and complications of cardio vascular disease.[14, 15]

Blake and Ridker[16] suggested that the CRP concentration might reflect the vulnerability of atheromatous lesion and the likelihood of a plaque to rupture. It has been reported that CRP correlate with number of vulnerable atherosclerotic plaques with superficial foam cells, large necrotic cores and thin fibrous cap atheroma.[17] These finding indicate that serum CRP levels are a marker of coronary artery disease activity and may be a biochemical marker of the diffuse inflammatory process that leads to multifocal plaque instability. Moreover, it has been suggested that CRP may not only be a marker of generalized inflammation but directly participates in both atherogenesis and atheromatous plaque disruption.[18,19]

Conclusion:

In our study we found a significantly high microalbuminuria and serum CRP in non hypertensive AMI patients. Microalbuminuria and serum CRP are the non specific yet sensitive markers of myocardial infarction. Since microalbuminuria and serum CRP are the simple investigations and relatively inexpensive tests we propose the use of microalbuminuria and serum CRP as adjunct biochemical parameters in non hypertensive AMI patients. However, more studies are required with a large sample size to ascertain whether microalbuminuria and serum CRP can predict in-hospital mortality and its pathophysiology in this clinical setting.

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