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SIGNIFICANCE OF MICRO ALBUMINURIA AS A PREDICTIVE FACTOR IN ACUTE MYOCARDIAL INFECTION

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ABSTRACT: OBJECTIVES: A study was undertaken among 50 non-diabetic patients of acute myocardial infarction (AMI) admitted within 24 hours of the onset of pain for investigation of the prevalence and significance of microalbuminuria (MA) as a predictor of in-hospital mortality and also to correlate it with other well-established prognostic markers. **METHODOLOGY:** Spot urinary albumin to creatinine ratio (ACR) was measured in first morning sample on day 1 (D₁). Day 4 (D₄) and /or day 7 (D₇) hemodynamic status was assessed in first clinical by Killip's class and the ejection fraction was measured by echocardiography on D₁, D₄ and /or D₇ total 7 days mortality was recorded. **RESULTS:** MA (>30µg/mg) was found in 95% of patients on D₁ in the group with higher value of MA (>100µg/mg) on D₁ there was significantly more deaths (p<0.01). Also there was significantly more death increasing or static MA value from D₁ to D₄/D₇ (p<0.01). Increasing or static MA had a positive correlation with deteriorating Killip's class in non-survivors, and also there was a correlation between decrease in left ventricular ejection fraction from D₁ to D₄/D₇ and an increasing trend in MA over that period. **CONCLUSION:** MA was found to be a reliable predictor of short-term in-hospital mortality in AMI.

KEYWORDS: Micro-albuminuria, Acute MI, Nephropathy.

INTRODUCTION: Micro-albuminuria (MA) is thought to be a marker of widespread vascular damage. A transient increase in stress and is associated with increased mortality.¹

The aim of this study was to evaluate the incidence of MA in hospitalized patients with acute myocardial infarction (AMI) without other confounding stress, and also to identify any association of MA on day 1 and its subsequent any association trend (from day 1 to 4 or 7) with 7-day mortality. Attempts were also made to find out the correlation between urinary albumin excretion (UAE) and two well-established prognostic markers of AMI namely, Killip's class for hemodynamic status and left ventricular ejection fraction (LVEF).^{2,3}

MATERIAL AND METHODS: A total 50 patients (35 male and 15 female, aged 34-67 years with a mean of 52.05±9.6 years) of first known AMI, diagnosed as per the ACC/AHA criteria,⁴ were included. Excluded were those patients with any primary or secondary renal disease, diabetic mellitus, abnormal urinalysis, hepatic or renal failure, clinical signs or symptoms of infection, neoplastic disease, and those cases of AMI who died due to unrelated causes.

All the patients were thrombolysed and routinely given aspirin (150mg daily) and clopidogrel (75mg daily). For comparison, 38 patients (33 males, 25 females, aged 37-82 years mean 52.7±10.1) with non-AMI cardiovascular disease and 30 (21 males, 9 females, age 42-85 years; 51.9±9.58) age and sex matched controls were also included.

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Spot urine sample was collected on day 1 (D_1), within 24 hours of entry into study protocol to measure UAE by immunoturbidimetry, and creatinine by colorimetry using Benedict-Behre reaction. Albumin to creatinine ratio (ACR) value less than $30\mu\text{g}/\text{mg}$ was considered normal and the value between $30\text{--}300\mu\text{g}/\text{mg}$ was taken as MA. Subsequent measurement was done on day 4 (D_4) and day 7 (D_7), and the mean of the two values taken.

Those survived less than 7 days, only D_4 value was taken. Based on the level of MA on D_1 , patients were divided into two groups- those with ACR value $>100\mu\text{g}/\text{mg}$ and those with, $100\mu\text{g}/\text{mg}$, and mortality between these two groups was compared. Mortality data was also analyzed between those having decreasing MA from D_1 to D_4/D_7 with those having static or increased values.

Similar to MA, Killip's core and echocardiography LVEF were determined on D_1 and subsequently and D_4/D_7 . Changes in the Killip's score (her increasing or decreasing) and LVEF were compared with the trend in the change in MA.

Variable were expressed as mean $\pm$ standard deviation (SD). The Student's 't' test, binomial proportion test, standard error of proportions and Spearmans' rank correlation were performed where appropriate. A p-value of <0.05 was considered statistically significant.

OBSERVATIONS: Data is represented in four (5%) out of 50 patients of AMI did not have any A on D_1 , rest 95% (46/50) showed MA, out of them, 18% ($n=9$) had $\text{ACR}>100\mu\text{g}/\text{mg}$ and rest 82% ($n=41$) had the value $<100\mu\text{g}/\text{mg}$, 71% ($n=10$) died in the hospital and 29% ($n=4$) survived. In 90% of those who died, (9 out of 10), the value of MA remained in all the patient with high value ($>100\mu\text{g}/\text{mg}$) who survived the value decline on D_4/D_7 from D_1 . Only 24% patients (10 out of 41) $<100\mu\text{g}/\text{mg}$ died in hospital.

There was statistically significant ($p<0.01$) increase in the number of death in those with $\text{MA}>100\mu\text{g}/\text{mg}$ on D_1 .

In 83% (42 out of 50) of patients, the level of MA decreased from D_1 to D_4/D_7 ; rest 17% (8 out of 50) showed a static or increasing value. In the first group, 22% ($n=11$) died. In comparison, 85% (11 out of 13 patients) died in the group showing static / increasing MAs from D_1 to D_4/D_7 . the 2 survived patients in this alter group actually had ACR on $D_1<100\mu\text{g}/\text{mg}$. there was significantly more change of death with static or increasing level of MAS from D_1 to D_4/D_7 ($p<0.01$).

Comparison was done for demographic parameters (age, sex) clinical characteristics (anterior versus inferior infraction). Lipid status, smoking habits, and drug history (beta-blockers, angiotensin converting enzyme inhibitors) did not reveal significant difference between those with D_1 ACT $<100\mu\text{g}/\text{mg}$ or $>100\mu\text{g}/\text{mg}$.

At admission, Killip's class was high (means worse) in the group with MA ($p<0.05$). The Killip's class remained static or increased on D_4/D_7 in 16% (8 out of 50); 67% ($n=8$) of them ultimately died. However, only 27% (9 out of 50) of patients whose killip's class improved from D_1 to D_4/D_7 expired. There was a positive correlation between increase in Killip's class and increased/ static MA level amongst those who died.

LVEF improved in 77% (39 out of 50) and decline or remained static in 23% (11 out of 50) from D_1 to D_4/D_7 . Only 25% (10 out of 39) in the former group and 56% (7 out of 11) in the later group died. Comparing changes in the LVEF and changes in UAE, there was a positive correlation ($r=+0.78$) between decrease in LVEF and increase in level of MA.

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DISCUSSION: MA predicts cardiovascular diseases in both diabetics and non-diabetics. Assessment of MA could be an instrument to identify those at an increased risk for coronary artery disease (CAD) in an early stage.⁵ MA is associated with increased risk of in-hospital on-year, and three year mortality in MI.⁶

Our study demonstrated that MA is found in high percentage of hospitalized patients with AMI (95%) with a rapid fall over next one week (from 62% on D₁ to 28% on D₄ and 14.5% on D₇). This suggests that rise in UAE in AMI is essentially an acute phase response.

Patients with MA on D₁ (>100µg/mg) had higher mortality. Furthermore, this increased risk was largely confined to the group with higher level of MA (>100µg/mg) indicating that with higher MA, the inflammatory insult was greater resulting in more death^{7,8}. Again a decline in the value of MA from D₁ to D₄/D₇ was associated with less mortality while increasing / static value had the opposite effect. An increasing or static value had the opposite of MI may be due to intrinsic perpetuation of disturbed vascular hemodynamic once triggered by a vascular insult or difficult in effective clearance of inflammatory response- both indicating a detrimental state of underlying vasculopathy prior to the insult¹. Expectedly this group had more mortality.

Independent clinical and echocardiography predictors of poor prognosis after MI are Killip's class (> or =2 on admission) and LVEF (<40%) respectively.^{2,3} By showing a good correlation between increasing or continued MA and deteriorating Killip's class, and also a positive correlation between increasing MA with deteriorating LVEF value, MA in this study was further validated as reliable prognostic marker in AMI.

Thus MA predicts 7 days mortality in hospitalized thrombolysed AMI patients and can be used as a reliable prognostic marker. ACR value of more than 100µg/mg on D₁ and a static or increased value from D₁ to D₄/D₇ are strongly associated with increased mortality.

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