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Microalbuminuria in Non Diabetic Acute Coronary Syndrome Patients.

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ABSTRACT

Background: Aim: To estimate microalbuminuria in non-diabetic patients with Acute Coronary Syndrome And assess the relationship between the two. **Methodology:** All patients age >18yrs, both sexes diagnosed as acute coronary syndrome based on history and relevant investigations and admitted in BLDEU'S Shri B.M PATIL Medical college hospital and research centre Vijayapur. microalbuminuria was measured at admission and compared with standard normal mean value. **Results**: This study was conducted on 60 patients, of the study group 70.0% were male and30.0% were female. The age ranged from 30 to 85 years of age. The mean age of thegroup was 55.5 ±13.19 SD. The known risk factors of ACS were studied and correlated,37.2 % of all patients were smokers, 31% were tobacco chewers, 24.7 % had diabetesmellitus, 31.8% were hypertensive and 8 % had family history of ACS. The meanmicroalbuminuria value in mg/dl for STEMI was 35 ± 0.30 SD, for NSTEMI it was 21±1.6 and for unstable angina it was 22 ± 1.0 SD. The mean microalbuminuria in patientswith ACS was 44.6 ± 3.2 SD mg/dl incompared to microalbuminuria levels of 30mg/l innormal population (p<0.0001). **Conclusion:** This study showed an correlation of microalbuminuria with ACS. This reinforcesthe fact that microalbuminuria acts as emerging potential risk factor marker.

Keywords: Acute Coronary Syndrome, Microalbuminuria, NSTEMI.

INTRODUCTION

Microalbuminuria is defined as an increased urinary albumin excretion which is detectable only by sensitive immunoassay it has been used for many years as a predictor of develop nephropathy in diabetic patients.[1,2] Recently it has been suggested that micro albuminuria may be a risk factor for the development of cardiovascular disease in thenon-diabetic patients and may therefore have role in screening programmes.

Atherosclerosis remains the leading cause of death and premature disability. Coronarydiseases Aute coronary Syndrome (ACS) is a common complication and this is associatedwith more than 2.5 million hospitalizations worldwide each year, Endothelialdysfunction seems to play a key role in non-diabetic glomerulosclerosis andatherosclerosis.

Increased permeability of the endothelium allows atherosclerotic lipoprotein particles (oxidized LDL and others) to penetrate into the vessel wall and promote the development of atherosclerotic plaques be associated with higher prevalence of coronary artery disease , microalbuminuria has as a early risk factor indicator for cardiovascular events.

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MATERIALS AND METHODS

Source of Data

ACS patients are admitted in to between November 2015 to July 2017with Acute coronary syndrome.

Method of collection of data

A total of 60 patients 42 were male and 18 in the age range group of 30 to 60years were selected on the basis of simple random sampling method , Information wasgathered in a pre–tested proforma .

The clinical history, clinical findings and laboratory findings were analyzed andrecorded in the proforma, patients were screened for systemic manifestations, renalfunction tests, lipid profile, urine tests, sugar, complete Haemogram .

Early morning urine samples were collected from patients in a sterile bottlewithout using any preservatives. Samples were tested for microalbuminuria byTurbidometric Immunoassay.

ACS was diagnosed on the basis of ECG findings and rasied cardiac enzymesand trop-t positive, patients included were those who presented with STEMI and NSTMIand UNSTABLE ANGINA.

Inclusion Criteria:

ACS patients who are positive for microalbuminuria by above mentioned method
Turbidometric Immunoassay.

1. Age > 18 yrs
2. Non diabetic patients with ACS diagnosis confirmed

Exclusion criteria:

ACS patients who had a past history of

1. Patients with Diabetes Mellitus
2. Urine showing
   - Macroalbuminuria
   - RBCs
   - Leucocytes
3. Patients on ACE Inhibitors

RESULTS

Table 1: Association Of Acs According To Urinary Albumin

ACS	Urinary albumin					p value
	<30		≥30		Total	
	N	%	N	%	N	
NSTEMI	8	50.0	15	34.1	23	0.533
STEMI	6	37.5	22	50.0	28	
Unstable	2	12.5	7	15.9	9	
Total	16	100.0	44	100.0	60	

Table 2: Distribution of cases according to Age

Age(yrs)	N	%
30-40	8	13.3
41-50	10	16.7
51-60	22	36.7
61-70	15	25
>60	5	8.3

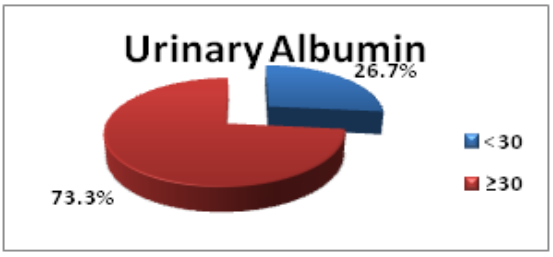

Figure 4: Distribution of cases according to Urinary Albumin

Table 3: Relation of risk factors with components of ACS

Risk factors	ACS								p value
	Unstable		NSTEMI		STEMI		Total		
	N	%	N	%	N	%	N	%	
CP	9	100.0	23	100.0	28	100.0	60	100.0	-
Sweating	6	66.7	12	52.2	23	82.1	41	68.3	0.072
Vomiting	2	22.2	7	30.4	9	32.1	18	30.0	0.851
HYPERTENSION	4	44.4	3	13.0	6	21.4	13	21.7	0.153
MI	1	11.1	2	8.7	0	0.0	3	5.0	0.241
ANGINA	0	0.0	0	0.0	0	0.0	0	0.0	-
HEPT	0	0.0	0	0.0	0	0.0	0	0.0	-
ACE inhibitors	1	11.1	0	0.0	0	0.0	1	1.7	0.056
Food habitis (NV)	3	33.3	12	52.2	17	60.7	32	53.3	0.355
Food habitis (V)	6	66.7	11	47.8	11	39.3	28	46.7	
Smoker	2	22.2	5	21.7	10	35.7	17	28.3	0.494
Alcoholic	0	0.0	10	43.5	3	10.7	13	21.7	0.004*
Total	9	100.0	23	100.0	28	100.0	60	100.0	

Table:

Parameters	Male				Female				Total			
	Min	Max	Mean	SD	Min	Max	Mean	SD	Min	Max	Mean	SD
AGE	30	85	55.7	12.6	38	80	58.6	10.4	30	85	56.5	12.0
Systolic BP	90	210	127.3	22.4	100	140	118.9	13.7	90	210	124.8	20.4
Diastolic BP	50	140	80.5	15.6	60	90	75.0	9.2	50	140	78.8	14.2
Urinary Albumin	13	98	43.5	21.3	12	90	47.4	24.3	12	98	44.6	22.1
CPK MB	10	300	74.3	66.0	23	133	58.1	31.8	10	300	69.4	58.1
Sr Creatinine	0.6	3.2	1.0	0.5	0.5	1.2	0.8	0.2	0.5	3.2	0.9	0.4
Hemoglobine	9	18	13.9	2.1	6.2	13.5	10.4	1.7	6.2	18	12.9	2.5
Total Cholesterol	90	319	180.5	48.9	115	252	171.1	37.9	90	319	177.7	45.8
Triglycerides	61	634	152.2	92.1	64	438	138.7	83.9	61	634	148.2	89.2
HDL-cholesterol	22	70	36.6	10.5	25	52	37.1	10.0	22	70	36.8	10.3
LDL-cholesterol	25	246	111.6	44.5	58	185	102.7	34.8	25	246	108.9	41.7
VLDL-cholesterol	12	126	30.8	18.5	13	87	27.9	16.7	12	126	29.9	17.9

In the 60 patients with Acute Coronary Syndrome (ACS) studied 70.0% were male and 30.0 % females. Males are more cases than female. The age ranged from 30 to 85 years of age. The mean age of the group was 55.5 + 13.19 and the ACS.

The know risk factors of ACS like smoking, tobacco chewing, diabetes mellitus, family history of ACS and hypertension were studied and correlated 37.2% of all ACS patients were smokers 31% chewed tobacco, 24.7% had diabetes mellitus, 8% had family

history of ACS and 31.8% were hypertensive. Hypertension had statistically significant correlation with ACS. All risk factors were more associated with STEMI compared to unstable angina or NSTEMI.

The lipid profile between males and females in the total cholesterol mean is 177.7 ± 4.2 and in that males the mean is 180.5 and in females 171.1 and SD is 45.8 in both and in males 48.9 and in females 37.9.

In triglycerides the mean in both is 148.2 ± 5.0 and in that males the mean is 152.2 and in females the mean is 138.7 and SD is 89.2 in both in males is 92.1 in females is 83.9 .

In HDL the mean in both is 36.8 ± 2.1 and in that males the mean is 36.6 and in females the mean is 37.1 and SD is 10.3 in both in males is 10.5 in females is 10.0 .

In LDL the mean in both is 108.9 ± 3.2 and in that males the mean is 111.6 and in females the mean is 102.7 and SD is 41.7 in both in males is 44.5 in females is 34.8

DISCUSSION

This study was conducted in60 patients admitted to BLDE hospital with Acute coronary syndrome (ACS) and whotested positive for microalbuminiuria, of 60 cases 42 were male (70%) and 18 females(30%).

The age ranged from 30 to 85 years of age. The mean age of the group was 55.5+ 13.19 SD. The patients in the age group of 51 to 60 years had the highest incidence ofACS. This finding was similar to other studies and accepted fact that the incidence ofACS increases with age.

Previous study by Borsch-Johnsen et al also showed a male preponderance .The duration of disease was defined as the number of days the patient wassymptomatic before he or she finally arrived at the hospital.

The mean duration of disease in microalbuminuria positive ACS patients were1day.

The symptom of chest pain was either divided in to STEMI, NSTEMI andUnstable angina who were diagnosed to have ACS but free of chest pain .The majority ofmales came with chest painPatients of ACS who were microalbuminuria positive were analyzed for theirpast history, 6 patients had history of MI out this 3 were males and 3 were females .

There was a significant difference ($p<0.05$) between microalbuminuriapositive male and female ACS population.

Majority of males presented with myocardial infraction i.e. 82.4 % whereas themajority of females i.e. 68.6 % presented with ischaemia on ECG. There was significant statistical difference between males and females in these.

In our study the majority of patients had RBS of <150 and there was nosignificant statistical difference between the two groups ($p>0.05$). the patients weregrouped into <150 and >150 because the value for our hospital for normal RBS wasbetween 150-180.

The CPK-MB levels were taken as significant when there were more than 2 times

the normal levels our laboratory >40 U/L. the majority of males 77.6% and a majority of

females 93.6% had a cpkmb levels.

Microalbuminuria has been long postulated to have marker in the ischemiccoronary arteries diseases and thus its correlation with ACS is of interest. This study triedto find association of urinary microalbuminuria levels in patients with acute coronarysyndrome.

The mean microalbuminuria in patients with ACS was 44.6 + 22.1 SD mg/lcompared to microalbuminuria levels of 30mg/l in normal population.

There is statistically significance microalbuminuria of ACS than normal standardmean value (0.5) ($p<0.0001$). Hence our research hypothesis accepted.

Increased levels of microalbuminuria were shown to be associated with higherprevalence of coronary artery disease emerging as new potential risk factor marker.

Alternative markers of cardiovascular risk

There is a need for a marker that will more precisely identify those at higher risk. Most of the markers investigated have failed to find general application. For example the significance of lipoprotein (a) remains uncertain and of the ten largest prospective studies, only six concluded that it was an independent risk factor.[4] Less controversy surrounds raised plasma fibrinogen concentration as an independent risk factor, as indicated by early Framingham data and confirmed in subsequent large-scale studies.[5,6] Its failure to be widely adopted is partly due to the lack of a fibrinogen – reducing therapy, although there is evidence that some lipid-lowering agents have fibrinogen – lowering capability.[7] Denesh et al. have recently reviewed the clinical and epidemiological evidence linking chronic infection, either bacterial (helicobacter pylori, Chlamydia pneumonia) or viral (cytomegalovirus), with coronary artery disease.[8] despite over 50 studies, the association remains dubious.

Against this background, microalbuminuria has been suggested as a risk factor indicator for cardiovascular events.

Microalbuminuria is used clinically to monitor diabetic nephropathy, but it is also know to be a non-specific marker of inflammation, as both systemic and local and appears to be useful as a predictor of outcome in several clinical situation.[9-16] An increase in urinary albumin excertion is indicative of increased glomerular permeability. The normal

urinary albumin excertion of albumin is less than 30 mg/day.

Albuminuria normally refers to greater than 300mg/day of albuminuria, with the term Microalbuminuria being used to describe smaller degrees of albuminuria.

Microalbuminuria is defined as urinary albumin excretion rate of more than 20mg/l and less than or equal to 200mg/l.[18] or 30 to 300mg/day in a 24hr collection.

Endothelial dysfunction seems to play a key role in non-diabetic glomerulosclerosis and atherosclerosis. Increased permeability of the endothelium allows atherosclerotic lipoprotein particles (oxidized LDL and others) to penetrate into the vessel wall and promote the development of atherosclerotic plaques.

Microalbuminuria is thought to reflect the glomerular component of a systemic capillary leak is fundamental to the pathogenesis of multiple organ failure. In the healthy kidney over 99% of filtered albumin is recollected by mechanisms that are close to saturation. A small increase in glomerular vascular permeability resolves in an increase in filtered albumin presented to the renal tubules. This cannot be reabsorbed and results in large increases in urinary albumin.

This amplification resolves in sudden rise in albumin excertion after the occurance of systemic inflammation, maximum microalbuminuria find up to 2 days before than the other markers of systemic inflammation such as C reactive protein.

Aute coronary Syndrome (ACS) is a common complication and this is associated with more than 2.5 million hospitalizations worldwide each year. In the United states, for example, Aconservative estimate for the number of discharges with ACS from hospital data in 2006

was 13,65000 unique for ACS.

India has the highest number of cases ACS in the world. Treatment and outcomesof Acute Coronary Syndromes in India (CREATE) in order data base has providedcontemporary data on 20,468 patients with ACS from 89 centers from 10 regions and 50 cities in India and found higher 30 day mortality than developed countries.

Cardiovascular risk factors for ACS are on the rise in people of Indian origin and ACS isnow the leading cause of death.[6-10]

CONCLUSION

There are many studies that showed a positive correlation betweenmicroalbuminiuria and acute coronary syndrome (ACS) hence many researcherspostulated that microalbuminiuria was a marker of ACS. This view was howeverdisputed by many others.

In our study patients with symptoms of ACSand were subsequently proved to the ACS patients were tested for microalbuminiuriaand those who tested for positive were included in the study.

Microalbuminiuria is known to be associated with diabetes, so we to includepatients who are non-diabetic, hypertensive and non hypertensive and aimed at forming aclinical profile of patients who are microalbuminiuria positive ACS patients.

Majority of the patients had a significant family history of hypertension andACS, smoking and alcoholism was a significant part of their personal history. Majority of them had a microalbuminiuria level of >30mg.

Patients of ACS were naturally tested for cardiac enzymes and in our group ofmicroalbuminiuria positive ACS patients, CPKMB and Trop t positive. Maximum60number of ACS patients are included in our study was admitted for chest pain, followedby admissions due to ACUTE CORONARY SYNDROME.

Our aim was to study the clinical profile of the patients with slightly elevatedurinary albumin excretion and whether consequently it may be a clinically relevant riskfactor.

Our study demonstrated the presence of microalbuminiuria independent ofother classic risk factor for ACS.

We also found a definitive correlation between LDLCholesterol and microalbuminiuria; a positive correlation between microalbuminiuria andTGL; and a positive correlation between microalbuminiuria and creatinine, which maybecome significant in a study based on a larger sample.

Thus individuals with elevated microalbuminiuria levels should get their LDL,Triglycerides classification of the individual as a high risk or high susceptibilityindividual.

It is unknown wheather individuals with microalbuminiuria will benefit morefrom intervention, but we would recommend that future controlled clinical trials shouldfocus on answering this question as it could to a more targeted and focused strategy forthe prevention of ACS.

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