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MICROALBUMINURIA IN PATIENTS OF ACUTE CORONARY SYNDROME AND ITS CORRELATION WITH FASTING LIPID PROFILE

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ABSTRACT:

BACKGROUND: *Microalbuminuria has been detected in the urine of patients after myocardial infarction and is established as an independent risk factor for the development of ischemic heart disease. The association of microalbuminuria with dyslipidemia is significant and patients with dyslipidemia have underlying urine albumin excretion which puts them at risk of developing atherosclerotic coronary artery disease.*

AIMS & OBJECTIVE: *To observe the frequency of urinary albumin excretion in hospitalized patients with acute coronary syndrome and to establish its correlation with the fasting lipid profile.*

MATERIAL & METHODS: *This prospective analytical study was done at the Cardiology department of Mayo Hospital, Lahore over six months using a non-probability purposive sampling technique. A sample size of 139 patients was calculated by using a 90% confidence level, a 7% margin of error, and by taking an expected percentage of microalbuminuria in acute coronary artery disease patients as 50%. The levels of Microalbuminuria were compared with the lipid profile. The patients were followed at regular monthly intervals up to six months and their Microalbuminuria levels and fasting lipid profile were measured and analyzed. Patients presenting with Acute ST-Elevation myocardial infarction, NSTEMI, and unstable angina were included. Patients with a history of Diabetes mellitus, Systemic hypertension, Urinary tract infection, Nephropathy(serum creatinine >1.0mg/dl), Old MI and AMI following surgery and major trauma, Patients on Statin Therapy, Patients on ACE Inhibitors, and Patients with UAE > 300 mg were excluded from the study. Data entry and analysis were done with SPSS 23.*

RESULTS: *A total of 139 patients were included in the study. There were 100(71.9%) male and 39(28.1%) female cases with a male to female ratio of 2.56 :1.The mean age of patients was 51.51±11.97 years. The mean weight, height, and BMI were 76.58±8.70 kg, 166.43±6.24 cm, and 27.77±3.93 respectively. The mean Urea was 27.98±9.09 and mean creatinine was 1.01±0.23 with minimum and maximum of 0.10 and 1.40. More male patients with microalbuminuria had <200mg/dl cholesterol then females. However in negative microalbuminuria same number of male patients were observed in <200 and >200mg/dl cholesterol groups. At baseline, Microalbuminuria was diagnosed in 120(86.3%) of the cases then reduced to 62(44.6%) at 1st month, 51(36.7%) at 2nd month, and 45(32.4%) at 3rd month. To see a relationship between*

Microalbuminuria and Fasting Lipid profile so on applying Pearson correlation, we found a week positive correlation between Urea and cholesterol only i.e. r = 0.183, p-value 0.031 (<0.05). While no significant correlation was found in other parameters.The mean cholesterol at baseline was 190.85±32.48 mg/dl in the Microalbuminuria group and 188.78±27.01 mg/dl in the normal albumin group. The mean triglycerides in cases with and without Microalbuminuria were 169.82±37.11 mg/dl and 223.42±187.69 mg/ dl. The mean HDL in cases with and without Microalbuminuria was 39.92±6.01 mg/dl and 36.84±6.049 mg/ dl while mean LDL in cases with and without Microalbuminuria was 109.08±17.45 mg/dl and 114.10±35.24 mg/ dl. As data were not normally distributed so we applied Mann Whitney U-test to compare the median in cases with and without Microalbuminuria. The median HDL was higher in cases of Microalbuminuria as compared to those whose albumin level was normal, p-value = 0.027 (< 0.05).

CONCLUSION: *Microalbuminuria is found in higher number of patients admitted with the ACS. Microalbuminuria has statistically sigifiant association with and LDL and triglceride levels.*

KEY WORDS: *Acute coronary syndrome, Urinary albumin, Microalbuminuria, Fasting Lipid profile*

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INTRODUCTION:

The morbidity and mortality associated with ischemic heart disease (IHD) are frequent globally. IHD was among the leading single cause of deaths worldwide in the last decade. In 2004, cardiovascular diseases (CVD) caused an estimated 17 million deaths and led to 151 disability-adjusted life years (DALYs) lost about 30% of all deaths and 14% of all DALYs lost that year [1]. CVD leads to almost 16.7 million deaths per annum in low and middle-income countries and nearly 80% of the 35 million deaths yearly due to chronic diseases; defined as a gross national income per capita of less than $10066 US dollars per annum in 2004.[2] The incidence of CHD is also being increasingly reported in the developing countries like Pakistan, India, and Bangladesh. Data from studies done in the last decade showed a significant increase in the incidence of CHD in the South Asian region.[3]

Many studies have reported on multiple new biomarkers and inflammatory markers of CHD such as increased lipoproteins (a) levels, total plasma homocysteine, elevated plasma fibrinogen levels, plasminogen activator inhibitor (PAI), C-reactive protein (CRP), different cytokines, and microalbuminuria (MA).[4] Microalbuminuria has implications on the development of coronary artery disease (CAD) and leading to myocardial infarction and it is emerging as an individualistic risk factor for CAD[5].

Microalbuminuria is now widely considered as a cause of atherogenesis and many studies indicated that risk factors for IHD and other CVD such as advanced age, gender, smoking, hypertension, dyslipidemia, and diabetes mellitus are often associated with microalbuminuria.[5]

Microalbuminuria is associated with endothelial dysfunction in coronary arteries and other vasculature and therefore predicts the development of atherothrombosis and CAD.[6] It has also been stated in Steno's hypothesis that MA is a marker of endothelial dysfunction. However, the strong association between endothelial dysfunction and the development of atherosclerosis leading to CAD remains poorly understood.

Evidence shows that the pathophysiology of MA and premature atherosclerosis is nearly the same. Heparin sulfate in endothelial cells tends to be decreased which results in decreased lipoprotein lipase binding and thus decreased clearance of VLDL and leads to hyperlipidemia.[7]

Although MA is frequently associated with atherosclerosis, it has also been compared and association is reported with dyslipidemia. Most of the patients presenting with ACS have atherosclerotic CAD and dyslipidemia is almost always present in them. In one study it has been studied and concluded that MA measurement in the general population can be used as an identification parameter for cardiovascular disease in patients of ACS together with fasting lipid profile to prevent morbidity and mortality.[8] Microalbuminuria is consistent with various other cardiac abnormalities in patients of IHD including ECG abnormalities, Left ventricular (LV) dysfunction and hypertrophy.[9] Acute coronary syndrome has been linked with Microalbuminuria and it is predictive of one-year mortality after acute MI[10].It has been studied and concluded that mortality associated with microalbuminuria is higher among high-risk individualsand that the risk remains among healthy non-diabetic and non-hypertensive individuals who have urine albumin excretion below normal threshold level[11, 12]. The screening of individuals at high risk should be done much earlier because it reflects vascular damage and endothelial dysfunction and premature atherosclerosis.

In a study conducted on patients of STEMI, the incidence of MA was 52%.[13] One study reported frequency and association of MA in patients of IHD which ranges from 33%-45% and varies according to age group.[14] The prevalence of microalbuminuria has been reported in various studies conducted on patients after acute MI and the results were (33.6%, 45.5%, 52.1%, and 54.2%).[15-18] The prevalence of MA in the general population is reported to fall between 4%-28% in various studies.[19, 20] Whereas an International, observational, and cross-sectional study of 22,282 patients with 5,605 attendees in Germany and Switzerland at 444 cardiology centers reported the prevalence of MAU in Germany and Switzerland (53.1%).[21]

Microalbuminuria has recently been linked with the development of atherosclerosis and coronary artery disease. It has been detected in the urine of patients after Myocardial Infarction andis established as an independent risk factor for the development of ischemic heart disease and even clinically negligible levels of microalbuminuria i.e., below 1mg/mmol, are associated with increased cardiovascular risk.The association of microalbuminuria with dyslipidemia is significant and patients with dyslipidemia have underlying urine albumin excretion which puts them at risk of developing atherosclerotic coronary artery disease. Therefore,if asymptomatic patients with dyslipidemia are screened for microalbuminuria, prevention of major adverse cardiovascular events (MACE) can be done effectively.

The present study was undertaken to measure the levels of MA and fasting lipid profile in non-diabetic and non-hypertensive individuals with acute MI and angiographically documented CAD, furthermore the correlation between MA and fasting lipid profile was analyzed and compared to the levels in healthy controls.

MATERIAL AND METHODS:

This prospective analytical study was done study was conducted in the Department of Cardiology, Mayo Hospital, Lahorefor six months. The sample size of 139 patients is calculated by using a 90% confidence level, 7% margin of error, and by taking an expected percentage of microalbuminuria in acute coronary artery disease patients as 50% 17.Patients presenting with Acute ST-Elevation myocardial infarction, NSTEMI, and unstable angina were included. Patients with a history of Diabetes mellitus, Systemic hypertension, Urinary tract infection, Nephropathy(serum creatinine >1.0mg/dl), Old MI and AMI following surgery and major trauma, Patients on Statin Therapy, Patients on ACE Inhibitors, and Patients with UAE > 300 mg were excluded from the study. A non-Probability purposive sampling technique was used.

n=139

P= Expected percentage of microalbuminuria in ACS patients = 50%

d= Absolute precision required = 7%

Z1-α= Confidence Interval 90% = 1.645

$$n = \frac{z^2_{1-\alpha/s} P(1-P)}{d^2}$$

MYOCARDIAL INFARCTION:

Based on a consensus document of the European Society of Cardiology (ESC) and American College of Cardiology (ACC), the term acute myocardial infarction (AMI) should be used when there is evidence of myocardial necrosis in a clinical setting consistent with acute myocardial ischemia.

One of the following must be present to establish and diagnose MI:

•Serial Elevation of cardiac biomarkers followed by normalization.
•Symptoms of ischemia. (Central chest pain lasting longer than 30 minutes).
•New-onset or apparent new significant ST Segment + T wave (ST–T) changes (elevation/ depression) or new-onset left bundle branch block (LBBB).
•Finding of pathological Q waves in the ECG.
•Imaging evidence of new loss of viable myocardium or new regional wall motion abnormality.
MICROALBUMINURIA
•Spot urinary albumin excretion rate of 30 – 300 mg.
•Albumin/Creatinine Ratio > 2.5 mg/mmol in males and >3.5mg/mmol in females.

DATA COLLECTION PROCEDURE:

After approval from the hospital ethical committee, informed consent was obtained from subjects included in the study who wereadmittedtothecoronary care unit of the cardiology department, Mayo Hospital, Lahore (MHL). Demographic and anthropometric data of each patient including name, age, sex, BMI, and contact and address wererecorded. The first-morning venous sample was obtained after overnight fasting of 10-12 hours. 24-hour urine specimen was collected and patient and his/her attendants wereinstructed to collect urine in sterile container provided to them. Fasting blood sugar (FBS), Total cholesterol, Triglycerides (TG), High-density lipoprotein (HDL), Low-density lipoprotein (LDL), and very-low-density lipoprotein (VLDL) weremeasured by automated enzymatic method. Serum and urine creatinine concentrations were measured by the kinetic calorimetric method. Microalbuminuria was measured by urine dip stick method. Albumin creatinine ratio was calculated. The levels of Microalbuminuria were compared with the lipid profile. The patients were followed at regular monthly intervals up to six months and their Microalbuminuria levels and fasting lipid profile weremeasured and analyzed.

DATA ANALYSIS PROCEDURE:

Data entry and analysis were done using SPSS 23. Quantitative data like age was presented by using mean ± SD. Qualitative data like gender was presented by using a frequency table, percentages, and appropriate graphs where applicable.

RESULTS:

The mean age of patients was 51.51 ± 11.97 years with an age range of 52 (28 and 80 as a minimum and maximum value). The mean weight, height, and BMI were 76.58 ± 8.70 kg, 166.43 ± 6.24 cm, and 27.77 ± 3.93 respectively. (Table 1)

The mean Urea was 27.98 ± 9.09 with minimum and maximum values as 12.60 and 55 and mean creatinine was 1.01 ± 0.23 with minimum and maximum of 0.10 and 1.40. Table 1

More male patients with microalbuminuria had <200mg/dl cholesterol then females. However in negative microalbuminuria same number of male patients were observed in <200 and >200mg/dl cholesterol groups. (Table 2)

More male patients with microalbuminuria had >150mg/dl triglycerides then females. However in negative microalbuminuria more number of male patients were observed in <150mg/dl triglyceride groups. (Table 2)

More male patients with both groups of microalbuminuria had >100mg/dl LDL then females. (Table 2)

At baseline, Microalbuminuria was diagnosed in 120(86.3%) of the cases then reduced to 62(44.6%) at 1st month, 51(36.7%) at 2nd month, and 45(32.4%) at 3rd month. (Table 3)

At admission the mean cholesterol was 190.57 ± 31.71, at 1st month it was 160.95 ±14.06 at 2nd month the mean cholesterol was 164.19 ± 12.97 and at 3rd month the mean cholesterol was 165.73 ± 13.29. (Table 4)

At admission the mean triglycerides were 177.14 ± 78.26 mg/dl, at the first month was 139.53 ± 30.17 mg/dl, at 2nd month it was 140.13 ± 30.33 mg/dl and at 3rd month the mean triglycerides were 152.84 ± 21.84 mg/dl. (Table 4)

The mean HDL at admission was 39.50 ± 6.06, at 1st, 2nd and 3rd month the mean HDL was 38.79 ± 6.40, 43.96 ± 9.33, and 44.95 ± 4.11 mg/dl respectively. (Table 4)

The mean LDL at admission, 1st month, 2nd month, and 3rd month was 109.77± 20.68, 99.15 ± 7.44, 97.27 ± 6.44, and 98.68± 11.98 mg/dl respectively. (Table 4)

As our second objective was to see a relationship between Microalbuminuria and Fasting Lipid profile so on applying Pearson correlation, we found a week positive correlation between Urea and cholesterol only i.e. r = 0.183, p-value 0.031 (<0.05). While no significant correlation was found in other parameters. (Table 5)

The mean cholesterol at baseline was 190.85 ± 32.48 mg/dl in the Microalbuminuria group and 188.78 ±27.01 mg/dl in the normal albumin group. The mean triglycerides in cases with and without Microalbuminuria were 169.82 ± 37.11

Table-1: Distribution of baseline characteristics among the study population.

Baseline Characteristics	Numbers (Percentages) n=139
Age	
Mean years	51.5±11.9
<40 years	19(13.675%)
40-60 years	93(66.91%)
>60 years	27(19.42%)
Gender	
Male	100(71.94%)
Female	39(28.06%)
Weight	
Mean kgs	76.6±8.7
Normal	31(22.3%)
Overweight	70(50.36%)
Obese	38(27.34%)
Height mean Cms	166.4±6.2
BMI mean kg/m^2	27.7±3.93
Urea mean	27.9±9.1
Creatinine	1.01±0.23

Table-2. Distribution of Lipid Profile in patients of ACS with Microalbuminuria distributed according to gender

Parameter	Cut off Values	Microalbuminuria Positive n = 120		Microalbuminuria Negative n = 19	
Total Cholesterol mg/dL	> 200 mg/dL	39(32.5%)	Males 28(23.33%) Females 11(9.16%)	8(42.1%)	Males 8(42.10%) Females 0(0%)
	< 200 mg/dL	81(67.5%)	Males 46(46.66%) Females 25(20.83%)	11(57.9%)	Males 8(42.10%) Females 3(15.7%)
Triglycerides mg/dL	>150 mg/dL	75(62.5%)	Males 49(40.83%) Females 26(21.66%)	7(36.8%)	Males 7(36.84%) Females 0(0%)
	<150 mg/dL	45(37.5%)	Males 35(29.16%) Females 10(8.33%)	12(63.2%)	Males 9(47.36%) Females 3(15.78%)
LDL	> 100 mg/dL	84(70.0%)	Males 59(49.16%) Females 25(20.83%)	15(78.9%)	Males 13(68.42%) Females 2(10.52%)
	<100 mg/dL	36(30.0%)	Males 25(20.83%) Females 11(9.16%)	4(21.0%)	Males 3(15.78%) Females 1(5.26%)
HDL	>40 mg/dL	60(50%)	Males 4(36.66%) Females 6(13.33%)	6(31.6%)	Males 5(26.31%) Females 1(5.26%)
	< 40 mg/dL	60(50%)	Males 40(33.33%) Females 20(16.66%)	13(68.4%)	Males 11(57.89%) Females 2(10.52%)

(Lab Value Reference Mayoclinic.org / American association of clinical chemistry).

Table -3: Frequency of Microalbuminuria at baseline, 1st month, 2nd month, and 3rd month		
Microalbuminuria		Frequency (percentage) n=139
Baseline	*Positive*	120(86.3%)
	Negative	19(13.7%)
1st month	*Positive*	62(44.6%)
	Negative	77(55.4%)
2nd month	*Positive*	51(36.7%)
	Negative	88(63.3%)
3rd month	*Positive*	45(32.4%)
	Negative	94(67.6%)

Table-4: Descriptive Statistics of various parameters at admission, 1st month, 2nd month, and 3rd month				
Parameter	Mean (SD) Admission	Mean (SD) at 1st Month	Mean (SD) at 2nd Month	Mean (SD) at 3rd Month
Cholesterol mg/dl	190.5±31.71	160.±14.1	164.2±12.9	165.7±13.3
Triglycerides mg/dl	177.2±78.3	139.5±30.2	140.13±30.33	152.84±21.84
HDL mg/dl	39.5±6.1	38.8±6.4	43.9±9.3	44.9±4.1
LDL mg/dl	109.7±20.7	99.2±7.4	99.3±7.4	98.7±11.98

Table-5: Correlation between different parameters			
At admission		Urea	Creatinine
Cholesterol mg/dl	*Pearson Correlation*	0.183*	0.139
	p-value	0.031	0.103
Triglycerides mg/dl	*Pearson Correlation*	0.051	-0.002
	p-value	0.552	0.982
HDL mg/dl	*Pearson Correlation*	-0.015	-0.038
	p-value	.863	0.654
LDL mg/dl	*Pearson Correlation*	0.004	-0.104
	p-value	0.965	0.223

mg/dl and 223.42 ± ± 187.69 mg/ dl. The mean HDL in cases with and without Microalbuminuria was 39.92 ± 6.01 mg/dl and 36.84 ± 6.049 mg/ dl while mean LDL in cases with and without Microalbuminuria was 109.08 ± 17.45 mg/dl and 114.10 ± 35.24 mg/ dl. As data were not normally distributed so we applied Mann Whitney U-test to compare the median in cases with and without Microalbuminuria. The median HDL was higher in cases of Microalbuminuria as compared to those whose albumin level was normal, p-value = 0.027 (< 0.05). (Table 6).

DISCUSSION:

Cardiovascular diseases are the most prevalent serious disorders among the industrialized nations and are rapidly growing among developing nations like Pakistan and India. Cardiovascular disease is responsible for 12 million deaths per year globally and is the commonest cause of death. Previously it was believed to be a disease of rich people but in the last three decades the incidence and prevalence of Coronary Artery Disease (CAD) have

Table-6: Comparison of Cholesterol, Triglycerides, HDL and LDL in positive and negative Microalbuminuria					
Microalbuminuria		Cholesterol mg/dl	Triglycerides mg/dl	HDL mg/dl	LDL mg/dl
Positive	*Mean*	190.85	169.82	39.92	109.08
	S.D	32.48	37.11	6.01	17.45
	Median	182.50	161.00	39.50	106.00
	IQR	54.20	36.80	8.00	21.00
Negative	*Mean*	188.78	223.42	36.84	114.10
	S.D	27.01	187.69	6.049	35.24
	Median	192.00	149.00	36.00	103.00
	IQR	58.00	40.00	6.00	16.00
Overall	*Mean*	190.56	177.14	39.50	109.77
	S.D	31.71	68.26	6.08	20.68
	Median	183.00	160.00	39.00	106.60
	IQR	53.0	37.00	9.00	20.0
p-value		0.785	0.279	0.027	0.785

declined in the western nations, but it is now rapidly increasing to become epidemic magnitudes in thedevelopingcountries. Ischemic heart disease is a state of compromised supply of blood and oxygen to the myocardium. It usually results when there is decreased oxygen supply to the myocardium; supply-demand mismatch. The blood flow to the myocardium perfused by a specific coronary artery is reduced if there is underlying atherosclerosis of the artery which causes inadequate myocardial tissue perfusion and subsequently ischemia.[22]

In our study, a total of 93(66.91%) cases were between 40-60 years of age, 27(19.42%) cases were > 60 years of age and 19(13.675%) cases were < 40 years of age. The overall mean age of patients was 51.51 ± 11.97 years with an age range of 52 (28 and 80 as a minimum and maximum value). Our findings were comparable to an observational cross-sectional study in which the majority of subjects were in the age group 51 to 60 years (40%), followed by > 60 years (30%), 41 to 50 years (20%), and < 40 years (10%), depicting the fact that risk of developing CAD is more common after 50 years.[23]

In our study,microalbuminuria was found in120(86.3%) of cases and this is also comparable to the values reported in an above-mentioned study which reported the prevalence of microalbuminuria among ACS patients without diabetes in 88.3% of cases.[23]

Moreover, in our study the levels of LDL and TGs in microalbuminuria positive cases were 84(70%) and 75(62.5%) respectively which were similar to the values reported in a study conducted in India.[24]However, both studies have shown an overall lower frequency of raised TC in the study population with no microalbuminuria.These findings were also similar to a study conducted in Nigeria on the hypertensive population.[25]

In 2015, another study has assessed the prevalence of UAE in the non-diabetic patients who have had UA/NSTEMI, and in these patients, the correlation of UAE to the severity of coronary artery disease was studied. There was a significant correlation of UAE with the signs of ischemia as evident on echocardiography, n = 20 (38 %), p < 0.01).[26] Khosravi conducted a study in Iran in 2009 to study the implications of urine albumin excretion and subclinical IHD. The 999 subjects in the trial were selected randomly who were between 35 to 70 years of age, 40.8% were male. Microalbuminuria was found in 8% of individualsand subclinical ischemic ECG changes were present in 23.4% of individuals.[27] In our Study, Subclinical ECG changes were present in 32.37% of patients with unstable angina.

In our study microalbuminuria was found in 120(86.3%) of the cases. This frequency differs from the results reported in a study in which the frequency of microalbuminuria in 22% of cases.[25] Moreover, in our study total cholesterol (TC) was raised in only 32.5% of microalbuminuria positive

cases which is in contradiction to the Indian study which reported raised TC in 50% of cases¬.[24]
Recently a local study demonstrated the role of microalbuminuria in the development of atherosclerotic coronary artery disease and its identification as a risk factor and the results had shown that microalbuminuria was found in 66 (22%) patients while 234 (78%) patients had no microalbuminuria.[25]
In a case-control study done by Sathisha T.G et al, the levels of MA in patients of acute MI were associated with a considerable rise in the levels of cholesterol (total), Low-density lipoproteins, Cholesterol to HDL ratio, LDL/HDL ratio, microalbuminuria, cardiac biomarkers, and cardiac troponin I ($p<0.001$) as compared to controls. The microalbuminuria levels were associated correspondingly with the Low Density lipoproteins (LDL) levels ($p = 0.010$, $r = 0.952$) and cardiac Troponin I ($p=0.025$, $r = 0.885$) and statistically it was significant.[24]
Recently a study conducted in Rawalpindi, Thirty controls (groups A) and fifty CHD patients (group B) included in this study were non-diabetic and non-hypertensive. When the values of microalbuminuria of group-A were compared with groupB a significant difference was found with $p < 0.05$. The levels of MA in Patients (group B) and controls (group A) were 36.58 μg/mg $\pm$3.78 and 21.78 μg/mg $\pm$ 1.01 respectively.[28]
Although MA is an early response following acute MI and is reported in the first study by Gosling, Hughes, Reynolds, Fox, et al.[29] MA prevalence was also increasingly reported in patients with MI. In one study, it has been reported to be a strong risk factor for developing complications in patients of Acute Myocardial Infarction.[30] In clinically healthy subjects the levels of atherogenic risk factors are increased if they have associated problems of microalbuminuria.[31] Microalbuminuria was reported to be significantly higher in angiographically reported CAD than disease-free individuals (28 versus 10mg/g; $P< 0.001$) and that urinary albumin excretion (UAE) increased progressively with the severity of CAD.[32]
Microalbuminuria has emerged as an independent and robust risk factor for cardiovascular diseases. It is well accepted that microalbuminuria reflects micro and macrovascular damage in patients having diabetes mellitus but still many more studies are accumulating evidence of its afflictions with progressive vascular diseases and cardiovascular diseases in the general community.[33]
Microalbuminuria has critical importance among cardiologists because of its detrimental effects in post-acute myocardial infarction state and more importantly, it has long-term extrapolative importance in patients of cardiovascular diseases. Even some studies have found subclinical levels of MA in patients who had otherwise normal coronary angiograms.[34] Consequently, the prognosis of patients with CAD and UAE is worse when compared to patients without any coronary disease. Generally, it is statistically recognized that UAE is more relevant in the establishment of cardiovascular diseases and complications than other risk factors. [35]
A recent multivariate analysis from the HOPE trial has shown that the probability of myocardial infarction, stroke, and cardiovascular deaths are higher in patients with UAE as compared to those only having standalone peripheral arterial disease or diabetes or male sex. The analyses further support the notion that, in the prospective assessment and probability of cardiovascular risk, the sub-threshold albumin excretion level (low-grade albuminuria) reflected anincreasedlikelihood of cardiovascular disease. [36]

CONCLUSION:

Microalbuminuria is found in higher number of patients admitted with the ACS. Microalbuminuria has statistically sigifiant association with and LDL and triglceridelevels.

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