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Association Between Urine Albumin Creatinine Ratio And Extended Lipid Profile In Diabetic And Hypertndive Patients To Predict Cardiovascular Morbidity And Mortality.

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Abstract

INTRODUCTION: Non-communicable diseases (NCDs) such as diabetes mellitus (DM) and hypertension (HTN) have emerged as global health challenges with rising prevalence, particularly in developing countries like India. ***Aim:*** To assess the association between extended lipid profile components and urinary albumin-creatinine ratio (UACR) in individuals with diabetes mellitus, hypertension, or both. ***Methodology:*** This was a hospital based cross-sectional observational study conducted at the Department of Medicine, S.P. Medical College and P.B.M. Associated Group of Hospitals, Bikaner. ***Result:*** Patients with diabetes mellitus and hypertension, particularly those with both conditions, had significantly higher UACR and a greater prevalence of macroalbuminuria. Apolipoprotein markers [Apo(A1), Apo(B), Apo(B)/Apo(A1) ratio, and Lp(a)] showed a stronger and more consistent association with albuminuria than the conventional lipid profile, while poor glycemic control (higher PPBG and HbA1c) was associated with increasing albuminuria. ***Conclusion:*** Early assessment of these markers, together with strict glycemic and blood pressure control, facilitate timely detection and reduce the risk of progression of diabetic kidney disease.

Keywords: Urine Albumin-to-Creatinine Ratio (UACR), Apolipoproteins, Diabetes Mellitus and Hypertension.

INTRODUCTION

Non-communicable diseases (NCDs) such as diabetes mellitus (DM) and hypertension (HTN) have emerged as global health challenges with rising prevalence, particularly in developing countries like India.1 In patients with DM or HTN, dyslipidemia is a common and critical metabolic abnormality. Traditionally, lipid abnormalities have been evaluated using parameters such as total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), and triglycerides (TG). However, recent research emphasizes the utility of extended lipid profiles, including markers such as Apolipoprotein B (ApoB), Apolipoprotein A1 (ApoA1), Lipoprotein(a) [Lp(a)], and small dense LDL (sdLDL). 2 Assessments of the conventional lipid profile including total cholesterol, triglycerides, HDL cholesterol and low-density lipoprotein (LDL) cholesterol do not always appropriately reflect the atherogenicity associated with dyslipidemia in the diabetic patients.

Instead, measurement of plasma ApoB may reflect the total number of atherogenic lipoprotein particles, including very low-density lipoprotein (VLDL), LDL, intermediate-density lipoprotein (IDL), and lipoprotein(a).Because ApoB is not always measured in clinical practice,3 the National Cholesterol Education Program-Adult Treatment Panel III (NCEP-ATP III) recommended the calculation of non-HDL cholesterol as a surrogate marker. However, Sniderman, Scantlebury, and Cianflone18 suggested that lipid measurement should include ApoB (rather than the surrogate non-HDL cholesterol) and patients should be classified based on triglyceride and ApoB levels because hypertriglyceride/hyper-ApoB phenotype is more atherogenic and is characterized by high triglyceride, low HDL cholesterol and increased numbers of small dense LDL particles, HDL:LDL ratio4.

Emerging studies suggest that the composition and size of lipoprotein particles may be more important than their absolute concentrations in predicting cardiovascular and renal risk. Traditional lipid profiles fail to detect subtle but clinically significant dyslipidemia seen in these patients. An extended lipid profile, including apolipoproteins, lipoprotein(a), and small dense LDL, provides more sensitive risk assessment. Despite this, extended lipid markers are not routinely evaluated in clinical settings, especially in resource-limited regions5,6. Identifying this correlation can promote early interventions and tighter control of metabolic parameters, ultimately reducing the burden of CKD and CVD. Hence, this study is required to explore the association between extended lipid parameters and UACR, aiming to stratify risk early in the disease process.

AIM

To assess the association between extended lipid profile components and urinary albumin-creatinine ratio (UACR) in individuals with diabetes mellitus, hypertension, or both.

METHODOLOGY

This was a hospital based cross-sectional observational study conducted at the Department of Medicine, S.P. Medical College and P.B.M. Associated Group of Hospitals, Bikaner. Study duration was from 1st July 2025 to 30th November 2025. Patients diagnosed with type 2 diabetes mellitus and/or hypertension attending outpatient/inpatient department. A consecutive sampling method was used, where all eligible patients attending the outpatient and inpatient departments during the study period were considered until the sample size was reached. Age between 30-70 years, Diagnosed with type 2 diabetes mellitus and/or hypertension and Willing to provide informed consent were included in the study. Exclusion criteria involved Known cases of chronic kidney disease (stage 3 or higher) ,Acute illness or infection within 4 weeks On lipid lowering medications, Urinary tract infection (Positive urine culture or pyuria) and Pregnancy or lactation.

RESULTS

Table 1: Baseline characteristics.

				UACR (mg/gm)				f	P
		<30		30-300		>300			
		No.	%	No.	%	No.	%		
Age Group (Year)	≤30	1	3.7	5	2.4	6	6.9	1.775	0.171
	31-40	6	22.3	29	13.7	7	8.1		
	41-50	9	33.3	41	19.4	19	21.8		
	51-60	6	22.2	54	25.6	31	35.6		
	>60	5	18.5	82	38.9	24	27.6		
Cases	HTN	14	51.9	87	41.2	17	19.6	32.290	<0.001
	DM	0	-	76	36.1	31	35.6		
	DM+HTN	13	48.1	48	22.7	39	44.8		

Normal UACR (<30 mg/g) was most common in the 41–50 years age group, whereas microalbuminuria (30–300 mg/g) and macroalbuminuria (>300 mg/g) were predominantly observed in patients aged >60 years and 51–60 years, respectively.Among the 325 participants, hypertension alone was the most common comorbidity (36.3%), followed by diabetes alone (32.9%) and combined diabetes with hypertension (30.8%).

Table 2: Distribution of cases according to lipid profile relation to UACR (mg/gm)

			UACR (mg/gm)				f	P
	<30		30-300		>300			
	Mean	SD	Mean	SD	Mean	SD		
TC	185.44	32.93	177.71	28.81	171.99	32.53	2.308	0.101
TG (mg/dl)	122.33	29.18	130.95	31.60	121.37	30.43	3.358	0.036
HDL (mg/dl)	38.33	9.38	39.59	6.23	37.18	6.70	4.103	0.017
LDL (mg/dl)	122.00	32.54	114.33	27.05	107.74	31.56	2.992	0.052
HDL/LDL	3.47	1.43	2.92	1.06	3.04	1.23	2.840	0.060
VLDL (mg/dl)	27.70	6.41	25.08	5.50	26.64	6.13	4.037	0.019

The mean total cholesterol and LDL cholesterol levels showed a declining trend with increasing UACR, while the HDL/LDL ratio was lower in patients with elevated UACR; however, these differences were not statistically significant ($p > 0.05$). In contrast, triglyceride, HDL, and VLDL levels differed significantly across UACR categories ($p < 0.05$).

Table 3: Distribution of cases according to extended lipid profile in relation to UACR (mg/gm)

			UACR (mg/gm)				f	P
	<30		30-300		>300			
	Mean	SD	Mean	SD	Mean	SD		
APO(A1) (mg/dl)	136.37	26.68	119.65	23.64	110.10	23.27	13.269	<0.001
APO(B) (mg/dl)	87.52	14.40	99.00	19.77	106.17	16.93	11.097	
APO(B)/ APO(A1) Ratio	0.68	0.23	0.86	0.29	1.01	0.26	21.781	
LP (A)	32.26	20.55	52.12	27.31	81.25	34.50	43.064	

With increasing UACR, mean Apo(A1) levels decreased significantly, whereas Apo(B), Apo(B)/Apo(A1) ratio, and Lp(a) levels increased progressively across normal, microalbuminuria, and macroalbuminuria groups.

Table 4: Distribution of cases according to post prandial blood glucose (mg/dl),glycated hemoglobin (%) in relation to UACR (mg/gm)

			UACR (mg/gm)				f	P
	<30		**30-300**		**>300**			
	Mean	**SD**	**Mean**	**SD**	**Mean**	**SD**		
PPBG (mg/dl)	188.11	76.43	200.29	73.83	226.86	73.09	4.890	0.008
HbA1c (%)	6.77	1.41	6.96	1.51	7.63	1.53	7.058	0.001

The mean PPBG level showed a progressive increase with rising UACR (mg/gm), being 188.11±76.43 mg/dl in the normal UACR (mg/gm) group and 200.29±73.83 mg/dl in the microalbuminuria group. The highest mean PPBG level was observed in the macroalbuminuria group.

The mean HbA1c level increased progressively with increasing UACR (mg/gm), from 6.77 ± 1.41% in the normal UACR (mg/gm) group to 6.96 ± 1.51% in the microalbuminuria group and 7.63 ± 1.53% in the macroalbuminuria group.

Table 5: Comparison of cases with lipid profile

Lipid Profile	**Cases**						**f**	**p**
	DM+HTN		**DM**		**HTN**			
	Mean	SD	Mean	SD	Mean	SD		
TC	172.72	29.51	180.20	28.85	176.25	30.00	1.667	0.190
TG	127.02	34.35	131.28	29.05	124.95	30.58	1.178	0.309
HDL	38.05	6.41	39.11	6.78	39.27	6.95	1.018	0.363
LDL	109.98	29.27	116.66	27.19	112.58	29.80	1.425	0.242
VLDL	25.85	5.59	25.91	5.66	25.43	6.13	0.225	0.799

Mean lipid profile parameters showed no statistically significant differences among patients with DM, HTN, and DM+HTN (all $p > 0.05$). Although patients with DM had slightly higher mean TC, TG, and LDL levels, while those with HTN had marginally higher HDL levels, these variations were not significant. VLDL levels were also comparable across all three groups, indicating a similar lipid profile irrespective of comorbidity status.

DISCUSSION

In our study, the majority of patients belonged to the age group above 60 years (34.2%), followed by 51–60 years (28.0%) and 41–50 years (21.2%). In our study, 36.3% of the patients had isolated hypertension, 32.9% had type 2 diabetes mellitus alone, and 30.8% had both diabetes mellitus and hypertension. Among patients with macroalbuminuria (UACR >300 mg/g), the highest proportion was observed in those having both diabetes and hypertension (44.8%), followed by those with diabetes alone (35.6%), whereas only 19.5% of patients with isolated hypertension had macroalbuminuria. Similar findings reported by Varghese et al7 in the Chennai Urban Rural Epidemiology Study observed a prevalence of microalbuminuria of 36.3%, and the presence of hypertension was significantly associated with albuminuria.

In our study, the mean total cholesterol levels among patients with UACR <30 mg/g, 30–300 mg/g, and >300 mg/g were 185.44±32.93 mg/dl, 177.71±28.81 mg/dl, and 171.98±32.53 mg/dl, respectively. A statistically insignificant association was observed.Hameed et al8 found a significant association between dyslipidaemia and diabetic kidney disease and concluded that lipid abnormalities contribute to renal damage and progression of albuminuria. In our study, 24.9% of patients had triglyceride levels >150 mg/dl, whereas 75.1% had triglyceride levels below 150 mg/dl. Khadka et al9 found that patients with albuminuria had significantly higher triglyceride levels compared with normoalbuminuric individuals (184.7±76.8 mg/dl vs. 149.2±57.4 mg/dl, p<0.05). In our study, 55.7% of patients had HDL cholesterol levels below 40 mg/dl, while only 3.4% had HDL levels above 50 mg/dl. In our study, the mean LDL cholesterol levels among patients with UACR <30 mg/g, 30–300 mg/g, and >300 mg/g were 122.00±32.54 mg/dl, 114.33±27.05 mg/dl, and 107.73±31.56 mg/dl, respectively. A statistically insignificant association was observed between LDL cholesterol and UACR categories (F=2.992, p=0.052).

In our study, the mean HDL/LDL ratio among patients with UACR <30 mg/g, 30–300 mg/g, and >300 mg/g was 3.47±1.43, 2.92±1.06, and 3.04±1.23, respectively. Most patients (69.2%) had an HDL/LDL ratio below 3.5, whereas only 4.6% had a ratio greater than 5.1. Similar findings were reported by Kumar et al10 in a study involving 48 patients with type 2 diabetes mellitus that evaluated the relationship between extended lipid profile and urine albumin-creatinine ratio. In our study, the mean VLDL levels among patients with UACR <30 mg/g, 30–300 mg/g, and >300 mg/g were 27.70±6.41 mg/dl, 25.08±5.50 mg/dl, and 26.64±6.13 mg/dl, respectively. Hameed et al11 in a study found that patients with diabetic kidney disease exhibited significantly higher triglyceride levels and more pronounced dyslipidaemia than normoalbuminuric individuals. In our study, a progressive decline with increasing severity of albuminuria, being 136.37±26.68 mg/dL in patients with normoalbuminuria (UACR <30 mg/g), 119.65±23.64 mg/dL in those with microalbuminuria (30–300 mg/g), and 110.10±23.27 mg/dL among patients with macroalbuminuria (>300 mg/g). Moreover, lower Apo(A1) categories (<107 mg/dL) were increasingly prevalent in patients with higher UACR values, whereas higher Apo(A1) concentrations (145–154 mg/dL and above) were observed in patients with normoalbuminuria. Comparable findings were reported by Chung et al12 in a study demonstrating that individuals with diabetic complications had significantly lower serum ApoA-I levels, and these associations were markedly influenced by urinary albumin excretion.

In our study, the mean Apo(B) level increased progressively from 87.52±14.40 mg/dL in patients with normoalbuminuria (UACR <30 mg/g) to 99.00±19.77 mg/dL in those with microalbuminuria (30–300 mg/g), and further to 106.17±16.93 mg/dL among patients with macroalbuminuria (>300 mg/g). Lower Apo(B) concentrations (64–84 mg/dL) were predominantly observed in patients with normoalbuminuria, whereas higher Apo(B) values (>114 mg/dL) were increasingly common among patients with severe albuminuria. Jenkins et al12, who evaluated Elevated Apo(B) levels were strongly associated increased urinary albumin excretion and a higher risk of cardiovascular events. In our study, the mean Apo(B)/Apo(A1) ratio increased progressively with worsening albuminuria, from 0.68±0.23 in patients with normoalbuminuria (UACR <30 mg/g) to 0.86±0.29 in patients with microalbuminuria (30– 300 mg/g), and further to 1.01±0.26 in patients with macroalbuminuria (>300 mg/g). Lower ratios (0.40–0.60) were predominantly observed among patients with normoalbuminuria, whereas higher ratios (>1.00) were markedly more common in patients with macroalbuminuria, accounting for 43.7% of cases. Jung et al13, who investigated the relationship between the Apo(B)/Apo(A1) ratio and diabetic nephropathy in patients with type 2 diabetes mellitus. In our study, the mean Lp(a) concentration increased progressively with increasing severity of albuminuria, being 32.26±20.55 mg/dL among patients with normoalbuminuria (UACR <30 mg/g), 52.12±27.31 mg/dL in patients with microalbuminuria (30–300 mg/g), and 81.25±34.50 mg/dL in those with macroalbuminuria (>300 mg/g). Furthermore, elevated Lp(a) levels (>50 mg/dL) were observed in 11.1% of normoalbuminuric patients, 37.4% of patients with microalbuminuria, and as many as 79.3% of patients with macroalbuminuria. Hiraga et al14, observed mean Lp(a) concentrations of 20.7±15.8 mg/dL in normoalbuminuric patients, 38.6±23.4 mg/dL in patients with microalbuminuria, and 67.5±35.2 mg/dL in those with overt nephropathy, indicating a strong positive correlation between albuminuria and Lp(a) levels.

In our study, the mean PPBG values increased progressively with worsening albuminuria, being 188.11±76.43 mg/dL in patients with normoalbuminuria (UACR <30 mg/g), 200.29±73.83 mg/dL in patients with microalbuminuria (30–300 mg/g), and 222.68±73.09 mg/dL among those with macroalbuminuria (>300 mg/g). Furthermore, elevated PPBG levels (>200 mg/dL) were observed in 44.4% of normoalbuminuric patients, 50.2% of patients with microalbuminuria, and as many as 67.8% of patients with macroalbuminuria. In our study, the mean HbA1c values increased progressively with worsening albuminuria, being 6.77±1.41% in patients with normoalbuminuria (UACR <30 mg/g), 6.96±1.51% in patients with microalbuminuria (30–300 mg/g), and 7.63±1.53% among those with macroalbuminuria (>300 mg/g). Moreover, elevated HbA1c levels (>6.4%) were present in 51.9% of normoalbuminuric subjects, 60.7% of patients with microalbuminuria, and as many as 81.6% of patients with macroalbuminuria.

In our study, the mean total cholesterol levels were 172.72±29.51 mg/dL, 180.20±28.85 mg/dL, and 176.25±30.00 mg/dL in the DM+HTN, DM, and HTN groups, respectively (F=1.667, p=0.190). Similarly, mean triglyceride levels were

127.02±34.35 mg/dL in the DM+HTN group, 131.28±29.05 mg/dL in the DM group, and 124.95±30.58 mg/dL among hypertensive patients (F=1.178, p=0.309). The mean HDL cholesterol levels were 38.05±6.41 mg/dL, 39.11±6.78 mg/dL, and 39.27±6.95 mg/dL, respectively (F=1.018, p=0.363). Likewise, no significant differences were observed in LDL cholesterol (F=1.425, p=0.242) or VLDL cholesterol (F=0.225, p=0.799).

CONCLUSION

The present study concludes that urinary albumin-creatinine ratio is a simple, reliable, and effective marker of early renal damage and is closely associated with adverse alterations in the extended lipid profile among diabetic and hypertensive patients. Measurement of UACR together with extended lipid profile parameters, particularly Apo(B), Apo(A1), Apo(B)/ Apo(A1) ratio, and Lipoprotein(a), facilitate early identification of individuals at high risk of cardiovascular morbidity and mortality. Incorporating these biomarkers into routine clinical evaluation improve cardiovascular risk stratification, enable timely therapeutic intervention, and ultimately reduce the burden of cardiovascular and renal complications in this high-risk population.

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