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Evaluating Urinary MCP-1 Levels in Patients with Diabetic Nephropathy: A Cross-Sectional Study

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KEYWORDS

Urinary MCP-1, Diabetic nephropathy, Microalbuminuria, Macroalbuminuria, Urinary albumin creatinine ratio

ABSTRACT:

Background: MCP-1 levels in urine have been shown to increase with advancing stages of diabetic nephropathy and correlate with urinary albumin excretion, a key marker of renal impairment.

Objective: To determine urinary MCP-1 levels in patients with diabetic nephropathy.

Methods: This was a hospital-based cross-sectional study conducted in the Department of Biochemistry, Vinayaka Mission's Kirupananda Variyar Medical College and Hospital, a tertiary teaching healthcare facility in Salem, Tamil Nadu, India between January 2024 and June 2024. The study included 30 healthy individuals as controls (Group A) and 90 diabetic patients diagnosed with nephropathy, up to stage 3 classification based on urinary albumin levels [Group B, patients with normoalbuminuria (UACR <30mg/g); Group C, microalbuminuria (UACR between 30 and 300mg/g); and Group D, macroalbuminuria (UACR >300mg/g)].

Results: The study analysed clinical and biochemical parameters across four groups, revealing significant differences in BMI, and alcoholism prevalence. Group D had the highest alcoholism prevalence (37.5%, $p = 0.010$). BMI increased progressively from Group A (23.8 ± 2.5 kg/m²) to Group D (28.4 ± 3.9 kg/m², $p = 0.004$). Diabetes duration was longest in Group D (10.1 ± 3.1 years, $p < 0.001$). No significant differences were found in smoking, blood pressure, or hypertension. Blood glucose, microalbumin, and HbA1c levels were significantly higher in diabetic groups ($p < 0.001$), while insulin levels showed no significant difference ($p = 0.993$). Other biochemical markers, including blood urea, creatinine, uric acid, and lipid profile, did not differ significantly. Urinary MCP-1 levels were elevated in diabetic groups but showed no significant intergroup differences ($p = 0.209$). Correlation analysis found a significant association between MCP-1 and HbA1c ($r = 0.219$, $p = 0.001$) and serum creatinine ($r = 0.141$, $p = 0.043$). ROC analysis indicated MCP-1's predictive value for diabetes (AUC = 0.632, $p = 0.041$) and diabetic nephropathy with macroalbuminuria (AUC = 0.612, $p = 0.086$), though the latter was not statistically significant.

Conclusion: MCP-1 may be involved in the pathophysiology of diabetes and nephropathy, it is not a robust standalone biomarker for diagnosing or predicting the progression of diabetic nephropathy.

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Introduction

Diabetic nephropathy (DN) is a serious and common complication of diabetes mellitus, characterized by progressive kidney damage and increased risk of end-stage renal disease (ESRD).(1) It remains a leading cause of chronic kidney disease (CKD) and renal failure globally, reflecting the need for early detection and effective management strategies.(2) The pathogenesis of DN involves a complex interplay of hyperglycaemia, hypertension, and inflammation, which contribute to kidney injury and functional decline.(3)

Monocyte chemoattractant protein-1 (MCP-1), also known as chemokine (CC-motif) ligand 2 (CCL2), is a cytokine that plays a pivotal role in inflammatory responses by recruiting monocytes to sites of injury.(4) Elevated levels of MCP-1 have been associated with various inflammatory and fibrotic diseases, including diabetic nephropathy.(5) MCP-1 is produced by various cell types in the kidney, including tubular epithelial cells and mesangial cells, and its expression is upregulated in response to hyperglycaemia and other stressors.(2) This upregulation can exacerbate renal inflammation and contribute to disease progression in DN. Urinary MCP-1 has emerged as a potential biomarker for detecting and monitoring diabetic nephropathy.(6, 7) Several studies have demonstrated that urinary levels of MCP-1 are elevated in patients with diabetic nephropathy and correlate with the severity of kidney damage.(8, 9) For instance, MCP-1 levels in urine have been shown to increase with advancing stages of diabetic nephropathy and correlate with urinary albumin excretion, a key marker of renal impairment.(10) However, the predictive value of urinary MCP-1 for diagnosing diabetes and diabetic nephropathy, particularly at different stages of the disease, remains to be fully elucidated. Against this background, the aim of the present study was to determine urinary MCP-1 levels in patients with diabetic nephropathy.

Materials and Methods

This was a hospital-based cross-sectional study conducted in the Department of Biochemistry, Vinayaka Mission's Kirupananda Variyar Medical College and Hospital, a tertiary teaching healthcare facility in Salem, Tamil Nadu, India between January 2024 and June 2024. The study was approved by the Institutional Human Ethics Committee (IHEC). The participants were given the Participant Information Sheet (PIS) in their native language, and its contents were verbally explained to ensure their understanding and satisfaction. Enrolment into the study proceeded upon receipt of written informed consent.

The total number of study subjects was 120 – divided into groups based on urinary albumin creatinine ratio (UACR). The study included 30 healthy individuals (caregivers of patients, friends, staff members, and family) as controls (considered Group A). Also, the study included 90 diabetic patients diagnosed with nephropathy, up to stage 3 classification based on urinary albumin levels – Group B, 30 diabetic patients with normoalbuminuria (UACR <30mg/g); Group C, 30 diabetic patients with microalbuminuria (UACR between 30 and 300mg/g); and Group D, 30 diabetic patients with macroalbuminuria (UACR >300mg/g). However, patients with acute/chronic infection, hepatic dysfunction, severe heart failure, hypo or hyperthyroidism, pregnancy and malignancy were excluded. Pregnant and lactating women were also excluded from the study. The blood and urine samples of each study participant was analysed for fasting blood glucose, postprandial blood glucose levels, blood urea, serum creatinine, lipid profile including total cholesterol, serum triglycerides, high density lipoprotein (HDL), low density lipoprotein (LDL), and very low-density lipoprotein (VLDL) using a fully automated analyser. The glycated haemoglobin levels (HbA1c) were measured using nephelometry. MCP-1 (Monocyte chemoattractant protein-1), also known as Chemokine (CC-motif) ligand 2 (CCL2) was analysed using sandwich human ELISA KIT (Invitrogen Human MCP-1 ELISA Kit).

The data obtained was manually entered into Microsoft Excel and analysed using Statistical Package for Social Sciences (SPSS) v23. All the categorical variables were summarised using frequencies and percentages. Continuous variables were summarized using mean (standard deviation) and/or median (interquartile range) (based on the results of data normality, tested using Kolmogorov–Smirnov test and the Shapiro–Wilk test). To test for statistical significance, Chi square test or Fisher exact test (for categorical variables) and independent “t” test or Mann Whitney U test (for

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continuous variables) was used. Pearson's correlation coefficient was estimated to assess the correlation between two continuous variables. We used receiver operating characteristic (ROC) analysis to determine the area under the curve (AUC) for MCP-1 to predict diabetes and diabetic nephropathy. Statistical significance was considered at p value less than 0.05.

Results

The mean age of the study participants was 55.2 ± 7.9 years. The prevalence of alcoholism was significantly different among the groups, with the highest percentage in Group D (37.5%) and the lowest in Group A (7.5%), showing a significant p-value of 0.010. The mean BMI progressively increased from Group A (23.8 ± 2.5 kg/m²) to Group D (28.4 ± 3.9 kg/m²), with a statistically significant difference (p = 0.004). The duration of diabetes mellitus was also significantly different among the diabetic groups, with Group D having the longest duration (10.1 ± 3.1 years) and Group B the shortest (6.5 ± 2.3 years), with a highly significant p-value (<0.001). However, no significant differences were observed for smoking (p = 0.178), systolic blood pressure (p = 0.062), diastolic blood pressure (p = 0.095), or hypertension (p = 0.072) among the groups.

In this study, blood glucose levels were significantly higher in Groups B, C, and D (223.0 ± 79.3 mg/dL, 295.8 ± 117.7 mg/dL, and 278.4 ± 133.1 mg/dL, respectively) compared to Group A (106.0 ± 15.0 mg/dL), with a statistically significant difference (p < 0.001). Similarly, HbA1c levels were significantly elevated in Groups B, C, and D (7.4 ± 1.8%, 7.7 ± 2.6%, and 9.3 ± 3.5%, respectively) compared to Group A (6.2 ± 1.2%), also showing statistical significance (p < 0.001). Insulin levels, however, did not differ significantly between groups (p = 0.993). No significant differences were observed for blood urea (p = 0.378), serum creatinine (p = 0.505), uric acid (p = 0.193), total cholesterol (p = 0.217), triglycerides (p = 0.172), HDL (p = 0.981), LDL (p = 0.320), and VLDL (p = 0.876) across the groups. Microalbumin levels were significantly elevated in Groups B, C, and D compared to Group A (p < 0.001).

In this study, urinary MCP-1 levels were found to be higher in all diabetic groups compared to the control group (Group A). Group A had a mean urinary MCP-1 level of 390.4 ± 219.6 pg/mL, while Group B (diabetic patients with normoalbuminuria) had a mean level of 559.9 ± 278.0 pg/mL. Group C (diabetic patients with microalbuminuria) had the highest mean urinary MCP-1 level at 595.5 ± 638.5 pg/mL, and Group D (diabetic patients with macroalbuminuria) had a mean level of 457.9 ± 227.8 pg/mL. However, these differences in urinary MCP-1 levels across the groups were not statistically significant (p = 0.209).

Pearson's correlation coefficient analysis between urinary MCP-1 levels and various clinical parameters: There was a weak negative correlation between MCP-1 and the urinary albumin-creatinine ratio (UACR), with a coefficient of -0.103, which was not statistically significant (p = 0.378). MCP-1 also showed a weak positive correlation with blood glucose levels (r = 0.080, p = 0.233), but this association was not significant either. However, MCP-1 exhibited a significant positive correlation with HbA1c (r = 0.219, p = 0.001), suggesting a relationship between increasing HbA1c and higher MCP-1 levels. Similarly, there was a positive but weak correlation between MCP-1 and blood urea (r = 0.105, p = 0.131), which was not statistically significant. A statistically significant positive correlation was observed between MCP-1 and serum creatinine (r = 0.141, p = 0.043), indicating that higher serum creatinine levels were associated with elevated MCP-1. MCP-1 also had a weak and non-significant positive correlation with uric acid (r = 0.065, p = 0.337). Overall, MCP-1 was significantly correlated with HbA1c and serum creatinine, while other parameters did not show significant associations.

ROC analysis of urinary MCP-1 levels: The ROC analysis of urinary MCP-1 levels revealed its predictive value for diabetes and diabetic nephropathy with macroalbuminuria. For diabetes, MCP-1 demonstrated an area under the curve (AUC) of 0.632, with a 95% confidence interval (CI) of 0.511 to 0.753. The optimal cutoff value for MCP-1 to predict diabetes was >348.8 pg/mL, yielding a sensitivity of 68.4% and specificity of 53.8%, with statistical significance (p = 0.041). For diabetic patients with macroalbuminuria (UACR > 300 mg/g), MCP-1 had an AUC of 0.612, with a 95% CI of 0.484 to 0.740. The suggested cutoff value was >400.7 pg/mL, providing a sensitivity of 66.7% and a specificity of 52.0%. However, this result did not reach statistical significance (p = 0.086).

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Discussion

Aging is a well-documented risk factor for metabolic disorders, including T2DM, as it is associated with increased insulin resistance, pancreatic beta-cell dysfunction, and an overall decline in glucose metabolism.(11) Older adults with diabetes are also at higher risk for cardiovascular complications, neuropathy, and nephropathy due to prolonged exposure to hyperglycaemia and associated metabolic stress.(12) Alcohol consumption was significantly different among the groups, with the highest prevalence in Group D (37.5%) and the lowest in Group A (7.5%) ($p = 0.010$). Chronic alcohol consumption is associated with increased risks of insulin resistance, obesity, and liver dysfunction, all of which contribute to diabetes progression and complications.(13) Studies suggest that excessive alcohol intake exacerbates metabolic syndrome by influencing hepatic glucose production, lipid metabolism, and inflammatory pathways.(14) The mean BMI progressively increased from Group A (23.8 ± 2.5 kg/m²) to Group D (28.4 ± 3.9 kg/m²), demonstrating a statistically significant trend ($p = 0.004$). Obesity is a well-established risk factor for diabetes development and progression, with excess adiposity contributing to insulin resistance, chronic inflammation, and dyslipidaemia.(15) Higher BMI has been linked to poor glycaemic control and an increased risk of diabetic complications, including cardiovascular disease and kidney dysfunction.(16)

The duration of diabetes significantly differed among the diabetic groups, with Group D having the longest duration (10.1 ± 3.1 years) and Group B the shortest (6.5 ± 2.3 years) ($p < 0.001$). Longer disease duration is associated with a higher risk of complications, including retinopathy, neuropathy, nephropathy, and cardiovascular diseases due to cumulative hyperglycaemic damage.(17) Chronic exposure to elevated blood glucose levels contributes to endothelial dysfunction, oxidative stress, and chronic inflammation, further exacerbating disease progression and comorbid conditions.(18)

The significantly higher blood glucose levels observed in Groups B, C, and D (diabetic patients) compared to Group A (healthy controls) are consistent with the pathophysiology of diabetes. Diabetic nephropathy is closely linked to poor glycaemic control, as chronic hyperglycaemia promotes the development of kidney damage via various mechanisms, including oxidative stress, inflammation, and the activation of the renin-angiotensin-aldosterone system (RAAS).(17) In this study, Group C (microalbuminuria) showed the highest mean glucose level (295.8 mg/dL), which is critical since microalbuminuria is an early marker of nephropathy.(19) Similarly, the significantly elevated HbA1c levels in Groups B, C, and D suggest poor long-term glycaemic control, which correlates with the progressive nature of diabetic nephropathy. HbA1c provides an average estimate of blood glucose levels over the preceding three months, and higher levels indicate sustained hyperglycaemia, a known risk factor for the development and progression of nephropathy.(20) This finding underscores the importance of maintaining optimal glycaemic control to prevent or delay the onset of diabetic nephropathy, as recommended by the ADA (2020). Interestingly, no significant difference in insulin levels was observed across the groups, which contrasts with expectations that insulin resistance, commonly associated with type 2 diabetes, might show variability. Insulin levels often depend on the severity of insulin resistance or beta-cell function. In individuals with type 1 diabetes or advanced stages of type 2 diabetes, insulin secretion may be low due to beta-cell failure. However, this study's finding could indicate that the diabetic patients were on insulin therapy or other antidiabetic treatments that might have normalized circulating insulin levels.(21) The lack of significant difference could also suggest that insulin levels alone may not directly correlate with nephropathy progression, highlighting the need to explore additional factors, such as inflammation or oxidative stress.

The progression of nephropathy typically leads to increased serum creatinine and urea as glomerular filtration rate (GFR) declines, but these parameters might not yet be drastically affected in the early to moderate stages of nephropathy.(22) Similarly, the lack of significant differences in lipid parameters (total cholesterol, triglycerides, HDL, LDL, and VLDL) could indicate that lipid abnormalities were managed through medications, such as statins, commonly prescribed to diabetic patients to reduce cardiovascular risks.(23) Although dyslipidaemia is prevalent in diabetic patients and contributes to nephropathy progression, its impact may have been mitigated by lipid-lowering therapies,

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thus explaining the non-significant findings. Microalbuminuria, an early indicator of diabetic nephropathy, was elevated in the diabetic groups compared to the controls.(19) Microalbuminuria is a well-known marker of endothelial dysfunction and a predictor of cardiovascular and renal outcomes in diabetic patients.(24) The study's non-significant results suggest that while trends are present, a larger sample size may be needed to confirm these findings definitively.

Urinary MCP-1 levels were found to be higher in all diabetic groups (B, C, and D) compared to the healthy control group (Group A), although the differences were not statistically significant ($p = 0.209$). The highest mean MCP-1 level was observed in Group C (microalbuminuria), which may suggest that MCP-1 plays a role in the early stages of nephropathy.(25) Microalbuminuria is considered an early marker of kidney damage, and the elevated MCP-1 levels in this group may indicate early inflammatory changes in the kidneys.(6) The lack of statistical significance in the differences between groups could be due to the small sample size or high variability in MCP-1 levels, as evidenced by the large standard deviation in Group C (595.5 ± 638.5 pg/mL). Previous research has shown that MCP-1 levels can fluctuate widely in diabetic patients depending on factors such as glycaemic control, the degree of kidney damage, and the presence of other inflammatory markers.(8) Therefore, larger studies may be needed to better understand the role of MCP-1 in different stages of diabetic nephropathy.

One of the key findings of this study is the significant positive correlation between urinary MCP-1 levels and HbA1c ($r = 0.219$, $p = 0.001$), indicating that higher levels of HbA1c are associated with increased MCP-1. This suggests a potential link between chronic hyperglycaemia and inflammation in diabetic patients. Chronic hyperglycaemia is known to induce oxidative stress and the production of advanced glycation end products (AGEs), which in turn activate inflammatory pathways, including the expression of MCP-1.(2) MCP-1 plays a role in recruiting monocytes and macrophages to sites of inflammation, contributing to kidney damage in diabetes. Thus, the positive correlation between HbA1c and MCP-1 underscores the importance of maintaining glycemic control to prevent or reduce inflammation and its associated complications in diabetic nephropathy.(3) Another significant correlation was found between MCP-1 and serum creatinine levels ($r = 0.141$, $p = 0.043$), suggesting that higher serum creatinine, a marker of kidney dysfunction, is associated with increased MCP-1 levels. Elevated serum creatinine indicates reduced kidney function, and the positive correlation with MCP-1 suggests that inflammation, as indicated by MCP-1, may worsen as kidney function declines.(26) MCP-1 is involved in the recruitment of inflammatory cells to the kidney, leading to fibrosis and further deterioration of kidney function.(27) This correlation supports the role of MCP-1 as a potential biomarker for kidney damage in diabetic nephropathy. The relationship between MCP-1 and serum creatinine has been documented in other studies. For example, Tashiro et al. (2002) found that MCP-1 levels were significantly elevated in diabetic patients with more advanced kidney disease, further supporting the link between MCP-1 and renal impairment.(8)

The ROC analysis demonstrated an AUC of 0.632 for MCP-1 in predicting diabetes, with a statistically significant result. This suggests that urinary MCP-1 levels could be a modest predictor of diabetes. An AUC value between 0.6 and 0.7, as observed here, generally indicates poor-to-fair discriminatory power.(28) Although the AUC is not particularly high, MCP-1's predictive ability may still offer some clinical value, particularly when used in combination with other diagnostic markers. The optimal cutoff value for MCP-1 to predict diabetes was identified as >348.8 pg/mL, with a sensitivity of 68.4% and a specificity of 53.8%. Sensitivity refers to the ability of MCP-1 to correctly identify patients with diabetes, while specificity indicates its capacity to correctly exclude those without the condition.(29) The sensitivity of 68.4% means that MCP-1 can correctly identify approximately two-thirds of diabetic patients based on the chosen cutoff value. However, its specificity is relatively low (53.8%), indicating a risk of false positives. While these results are statistically significant, the modest AUC and specificity suggest that MCP-1 alone may not be sufficient as a standalone diagnostic marker for diabetes but could contribute to a panel of markers for better diagnostic accuracy. Previous studies have highlighted the role of MCP-1 in the inflammatory response in diabetes. For instance, increased MCP-1 levels have been linked to insulin resistance, chronic hyperglycaemia, and systemic

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inflammation, all of which contribute to the development of type 2 diabetes.(30) Therefore, MCP-1 may serve as an inflammatory biomarker reflecting underlying processes related to diabetes pathogenesis, although its modest predictive performance indicates that other factors must be considered in diagnosis.

For diabetic nephropathy with macroalbuminuria (UACR > 300 mg/g), the ROC analysis yielded an AUC of 0.612, which falls within the same poor-to-fair discriminatory range as the AUC for diabetes. Although the result did not reach statistical significance ($p = 0.086$), it is worth noting that the AUC of 0.612 suggests some potential for MCP-1 as a predictor of macroalbuminuria, albeit with limitations. The optimal cutoff value for MCP-1 to predict diabetic nephropathy with macroalbuminuria was found to be >400.7 pg/mL, with a sensitivity of 66.7% and specificity of 52.0%. Similar to the findings for diabetes, the sensitivity indicates that MCP-1 could correctly identify two-thirds of patients with macroalbuminuria, while the specificity suggests that about half of the patients without macroalbuminuria could be correctly excluded. This relatively low specificity raises concerns about the potential for false positives, which could reduce the clinical utility of MCP-1 as a predictor of macroalbuminuria when used independently. The lack of statistical significance for MCP-1 in predicting macroalbuminuria ($p = 0.086$) indicates that while MCP-1 may play a role in the pathophysiology of diabetic nephropathy, its utility as a reliable predictor is limited. Diabetic nephropathy is a multifactorial condition characterized by chronic hyperglycaemia, glomerular hypertension, and oxidative stress, leading to kidney inflammation and fibrosis.(3)

Conclusion

This study highlights the clinical and biochemical differences among diabetic and non-diabetic groups, with significant variations in BMI, diabetes duration, blood glucose, microalbumin, and HbA1c levels. While urinary MCP-1 levels were elevated in diabetic patients, they did not show statistically significant differences across the groups. However, MCP-1 demonstrated a significant positive correlation with HbA1c and serum creatinine, suggesting its potential role in glycaemic control and kidney function. ROC analysis indicated that urinary MCP-1 may serve as a predictive biomarker for diabetes, with moderate diagnostic accuracy. However, its ability to predict diabetic nephropathy with macroalbuminuria was not statistically significant. These findings suggest that while MCP-1 may be associated with diabetes and renal dysfunction, further research with larger sample sizes is required to establish its clinical utility as a diagnostic or prognostic marker.

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Table 1: Comparison of study groups, by demographic and clinical characteristics

	Group A N = 40	Group B N = 40	Group C N = 40	Group D N = 40	P value
	Mean (SD)	Mean (SD)	Mean (SD)	Mean (SD)	
Age (in years)	55.2 (9.5)	53.8 (8.1)	54.3 (6.8)	57.6 (7.2)	0.625
Smoking, n (%)	5 (12.5)	8 (20.0)	10 (25.0)	12 (30.0)	0.178
Alcoholism, n (%)	3 (7.5)	10 (25.0)	12 (30.0)	15 (37.5)	0.010*
SBP (in mmHg)	118.5 (8.2)	126.2 (9.1)	132.8 (10.4)	138.6 (11.2)	0.062
DBP (in mmHg)	76.1 (5.4)	78.9 (6.2)	80.5 (6.8)	82.2 (7.1)	0.095
Hypertension, n (%)	6 (15.0)	12 (30.0)	18 (45.0)	22 (55.0)	0.072
BMI (in kg/m2)	23.8 (2.5)	25.6 (3.1)	27.1 (3.6)	28.4 (3.9)	0.004*
Duration of DM (in years)	–	6.5 (2.3)	8.2 (2.6)	10.1 (3.1)	<0.001*

*Statistically significant at p<0.05
SBP, Systolic blood pressure; DBP, Diastolic blood pressure; BMI, Body mass index; DM, Diabetes Mellitus

Table 2: Comparison of blood glucose, insulin, renal parameters, lipid parameters, and electrolytes between the study groups

	Group A	Group B	Group C	Group D	P value
Blood glucose	106.0 (15.0)	223.0 (79.3)	295.8 (117.7)	278.4 (133.1)	<0.001*
HbA1c	6.2 (1.2)	7.4 (1.8)	7.7 (2.6)	9.3 (3.5)	<0.001*
Insulin	54.0 (60.6)	51.2 (51.2)	54.5 (75.5)	50.8 (53.6)	0.993
Blood Urea	20.5 (6.1)	23.4 (9.7)	25.1 (15.4)	23.4 (6.9)	0.378
Serum creatinine	0.8 (0.3)	0.8 (0.2)	0.8 (0.2)	0.8 (0.3)	0.505
Uric acid	4.9 (1.7)	5.2 (1.5)	5.0 (1.9)	4.3 (1.3)	0.193
Total cholesterol	173.6 (36.1)	193.9 (40.8)	178.2 (38.8)	178.2 (42.9)	0.217
Triglycerides	147.8 (74.5)	205.6 (158.7)	181.0 (99.9)	198.3 (79.3)	0.172
HDL	44.3 (7.1)	44.0 (9.1)	44.6 (9.3)	43.7 (9.7)	0.981
LDL	100.9 (29.6)	113.4 (28.9)	101.9 (29.6)	102.2 (30.6)	0.320
VLDL	36.7 (19.8)	37.4 (16.3)	41.1 (21.5)	37.7 (30.3)	0.876
Microalbumin	13.8 (26.6)	21.0 (7.6)	152.1 (57.1)	346.7 (30.1)	<0.001*

*Statistically significant at p<0.05
HbA1c, Glycated haemoglobin; HDL, High density lipoprotein; LDL, Low density lipoprotein; VLDL, Very low-density lipoprotein

Table 3: Comparison of urinary MCP-1 between the study groups

	Group A	Group B	Group C	Group D	P value
Urinary MCP-1	390.4 (219.6)	559.9 (278.0)	595.5 (638.5)	457.9 (227.8)	0.209

*Statistically significant at p<0.05
MCP-1, Monocyte chemoattractant protein-1

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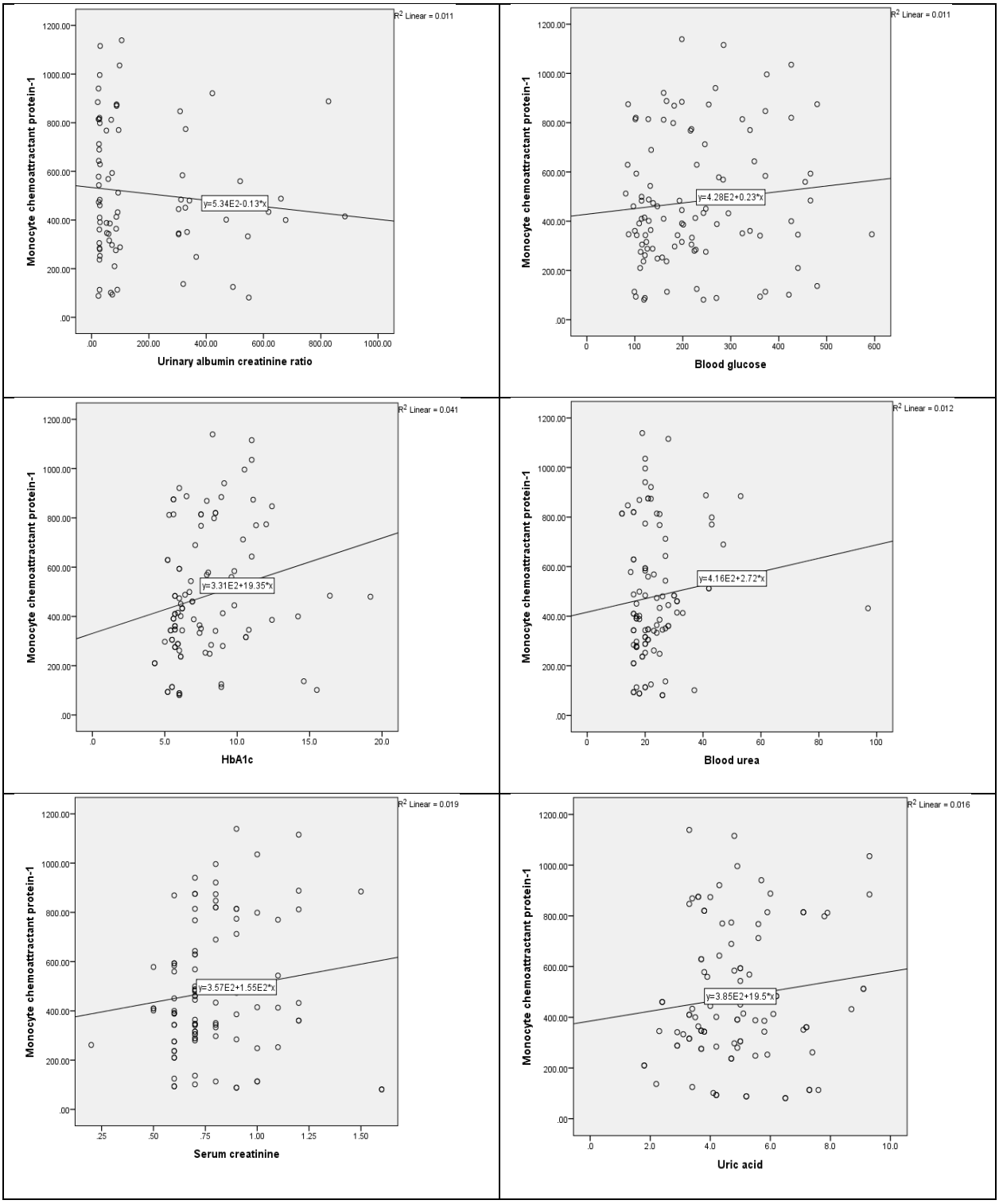

Figure 1: Pearson's correlation coefficient analysis between urinary MCP-1 levels and various clinical parameters

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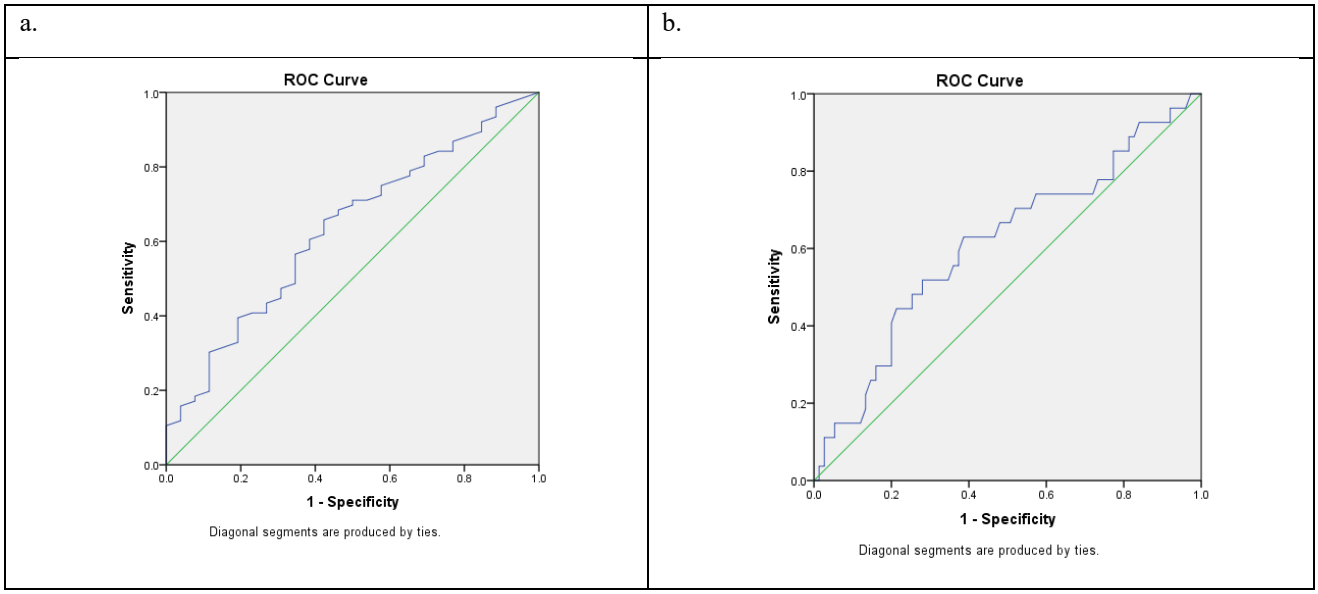

Figure 2: ROC analysis of urinary MCP-1 levels

Table 4: ROC analysis of urinary MCP-1 levels

MCP-1	AUC	95% CI	Cutoff	Sensitivity	Specificity	P value
Diabetes	0.632	0.511 to 0.753	>348.8	68.4	53.8	0.041*
DM with macroalbuminuria (UACR >300mg/g)	0.612	0.484 to 0.740	>400.7	66.7	52.0	0.086