National Board of Examinations - Journal of Medical Sciences
Volume 4, Issue 5, Pages 667–680, May 2026
DOI 10.61770/NBEJMS.2026.v04.i05.004

ORIGINAL ARTICLE

Serum Adropin Levels as a Potential Biomarker in Diabetic Nephropathy: An Analytical Cross-Sectional Analytical Study

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Accepted: 4-April-2026 / Published Online: 6-May-2026

Abstract

Background: Diabetic nephropathy is a leading cause of chronic kidney disease and end-stage renal disease worldwide. Conventional markers such as serum creatinine and urine albumin–creatinine ratio (ACR) have limitations in early detection. Adropin, a novel peptide hormone involved in metabolic regulation and endothelial function, has emerged as a potential biomarker in metabolic disorders. **Objectives:** To evaluate serum adropin levels in patients with type 2 diabetes mellitus (T2DM) with and without nephropathy and to assess its association with renal function and metabolic parameters. **Materials and Methods:** This cross-sectional study included 90 T2DM patients, divided into Group A (with nephropathy, n=45) and Group B (without nephropathy, n=45). Clinical parameters, glycaemic indices, lipid profile, renal function tests, and serum adropin levels (ELISA) were measured. Statistical analysis included independent t-test, Pearson's correlation, multiple regression, logistic regression, and ROC curve analysis. **Results:** Patients with nephropathy had significantly higher BMI, blood pressure, fasting blood glucose, HbA1C, and ACR, along with lower eGFR compared to those without nephropathy ($p<0.05$). Serum adropin levels were significantly reduced in Group A (89.9$\pm$55.2 vs 127.5$\pm$51.9 in Groip B) Adropin showed a negative correlation with BMI, glycaemic parameters, and ACR, and a positive correlation with eGFR and HDL cholesterol. Logistic regression identified adropin as an independent predictor of nephropathy. ROC analysis demonstrated moderate diagnostic accuracy (AUC=0.701), with an optimal cut-off of 80 ng/L (sensitivity 75%, specificity 72%). **Conclusion:** Serum adropin levels are significantly reduced in diabetic nephropathy and correlate with renal dysfunction. Adropin may serve as a promising adjunct biomarker for early detection and risk stratification of diabetic nephropathy.

Keywords: Adropin, Diabetic nephropathy, ACR, eGFR, Type 2 diabetes mellitus

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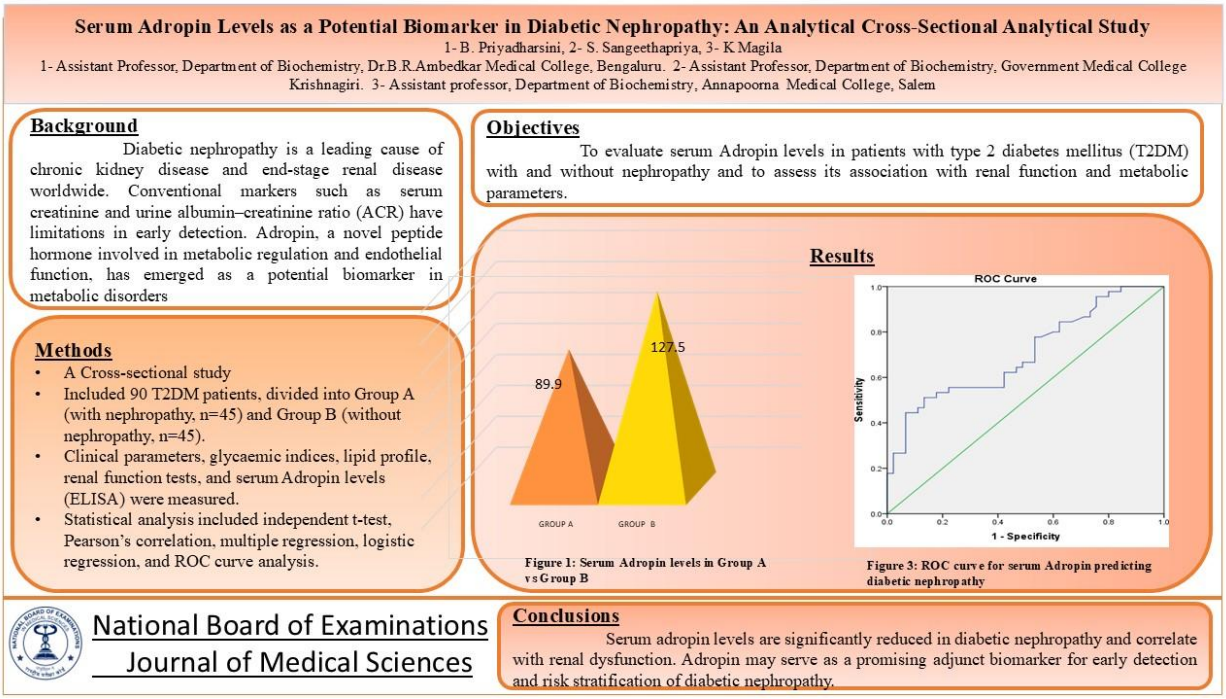
Graphical Abstract



Background

Chronic kidney disease (CKD) has emerged as a significant global public health concern, with its burden steadily increasing across both developed and developing nations. Recent global estimates indicate that CKD-related mortality has risen by nearly one-third, approximately 31.5%, over the past decade, reflecting its growing contribution to morbidity and mortality worldwide [1]. Among the various etiological factors, diabetic nephropathy remains the leading cause of CKD and end-stage renal disease (ESRD), accounting for a substantial proportion of patients requiring renal replacement therapy. It is estimated that nearly 20–40% of individuals with type 1 or type 2 diabetes mellitus eventually develop diabetic nephropathy, underscoring its critical role in the natural history of diabetes [2]. In the Indian context, the burden is particularly alarming, with approximately 72 million individuals affected by diabetes as of 2019, and nearly one-third, around 34.4%, progressing to nephropathy, thereby placing a considerable strain on healthcare resources [3].

The pathogenesis of diabetic nephropathy is multifactorial and involves complex metabolic and hemodynamic alterations triggered by chronic hyperglycaemia. Key biochemical pathways implicated include the formation of advanced glycation end products (AGE), activation of protein kinase C (PKC), and increased flux through the polyol and hexosamine pathways [4]. These mechanisms converge to induce oxidative stress and inflammatory responses, primarily mediated through activation of nuclear factor-kappa B (NF-κB), leading to increased production of pro-inflammatory cytokines and growth factors. Consequently, these processes promote mesangial expansion, extracellular matrix accumulation, fibrosis, and ultimately

glomerulosclerosis, which are hallmark features of progressive renal damage [5]. Clinically, the assessment of diabetic nephropathy relies on conventional markers such as urine albumin–creatinine ratio (ACR) and estimated glomerular filtration rate (eGFR). However, these markers have inherent limitations; serum creatinine levels typically rise only after nearly 50% of renal function has already been compromised, while ACR may not consistently detect early glomerular injury or reflect subtle structural changes [6]. Therefore, there is an increasing need to identify sensitive and specific biomarkers that can facilitate early detection and timely intervention in diabetic nephropathy [7].

Adropin, a recently identified peptide hormone consisting of 76 amino acids and encoded by the ENHO gene located on chromosome 9p13.3, has gained attention for its potential role in metabolic regulation [8]. It is widely expressed in various tissues, including the liver, brain, kidney, heart, and vascular endothelium, suggesting its systemic physiological significance. Functionally, adropin is involved in maintaining energy homeostasis by modulating glucose metabolism, enhancing insulin sensitivity, regulating lipid metabolism, and preserving endothelial integrity [9]. At the molecular level, adropin exerts vasoprotective effects by activating endothelial nitric oxide synthase (eNOS), thereby increasing nitric oxide (NO) bioavailability and improving endothelial function [10]. Additionally, it exhibits anti-inflammatory properties by downregulating pro-inflammatory cytokines such as tumor necrosis factor-alpha (TNF-α) and interleukin-6 (IL-6) at the mRNA level [11]. Adropin also influences lipid metabolism by suppressing the expression of lipogenic genes through peroxisome proliferator-activated receptor gamma (PPAR-γ) pathways, thereby contributing to metabolic balance [12].

Emerging evidence suggests that circulating adropin levels are significantly reduced in several metabolic and cardiovascular disorders, including type 2 diabetes mellitus [13], obesity [14], non-alcoholic fatty liver disease (NAFLD) [15], hypertension and coronary artery disease [10]. In the context of diabetic nephropathy, previous studies have demonstrated a significant association between decreased adropin levels and worsening renal function. Notably, Hu and Chen [16] reported that patients with diabetic nephropathy had significantly lower serum adropin concentrations, which showed a negative correlation with urine ACR and a positive correlation with eGFR, indicating its potential utility as a biomarker of renal dysfunction. However, despite these promising findings, there is a paucity of data from the Indian population. In view of this gap in literature, the present study was undertaken to evaluate the role of serum adropin in diabetic nephropathy and its association with renal function parameters.

Objectives

To evaluate serum adropin levels in patients with type 2 diabetes mellitus (T2DM) with and without nephropathy and to assess its association with renal function and metabolic parameters.

Materials and Methods

This cross-sectional analytical study was conducted at the Institute of Diabetology and Nephrology, Rajiv Gandhi Government General Hospital, a tertiary care teaching institution affiliated with Madras Medical College over a period of one year from December 2018 to December

2019. Participants were recruited from outpatient and inpatient services of the study centre. Participants diagnosed with type 2 diabetes mellitus as per American Diabetes Association (ADA) criteria with age between 35 and 70 years who provided informed written consent were included in the study. Patients with severe cardiovascular disease, malignancy, acute infections, chronic inflammatory conditions and other endocrine disorders were excluded. A total of 90 participants diagnosed with type 2 diabetes mellitus (T2DM) were included in the study and categorized into two groups:

- Group A: 45 patients with diabetic nephropathy
- Group B: 45 patients without diabetic nephropathy

Diabetic nephropathy was defined based on the presence of persistent albuminuria (ACR ≥30 mg/g) and/or reduced estimated glomerular filtration rate (eGFR <60 mL/min/1.73 m^2), in accordance with established clinical guidelines [6].

Venous blood samples were collected from all participants after an overnight fasting period of 8–12 hours under aseptic conditions. Serum was separated by centrifugation at 3000 revolutions per minute for 15 minutes. EDTA-anticoagulated samples were used for glycated hemoglobin (HbA1C) estimation. Serum aliquots of 1 mL were stored at –20°C for subsequent adropin analysis. Additionally, spot urine samples were collected for estimation of urine albumin–creatinine ratio (ACR).

Biochemical parameters were measured using standardized laboratory methods. Serum adropin levels were quantified using enzyme-linked immunosorbent assay (ELISA). Fasting blood glucose was estimated by the hexokinase end-point method. Blood urea levels were measured using the kinetic urease–glutamate dehydrogenase (GLDH) method, while serum creatinine was assessed using Jaffé's kinetic method traceable to isotope dilution mass spectrometry (IDMS). Estimated glomerular filtration rate (eGFR) was calculated using the CKD-EPI equation. Glycated hemoglobin (HbA1C) was determined using capillary electrophoresis (Sebia Capillarys 2 system). Lipid profile parameters, including total cholesterol, triglycerides, and high-density lipoprotein, were estimated by enzymatic spectrophotometric methods, and low-density lipoprotein was calculated using the Friedewald formula. Urine albumin–creatinine ratio was measured using immunoturbidimetric methods for albumin and Jaffé method for creatinine.

Data were analysed using SPSS. Continuous variables were expressed as mean ± standard deviation, and categorical variables as frequencies and percentages. The Chi-square test was used to compare categorical variables between groups. Student's independent t-test was applied to compare continuous variables between the two groups. Pearson's correlation analysis was performed to evaluate associations between serum adropin levels and clinical as well as biochemical parameters. Multiple linear regression analysis was used to identify independent determinants of eGFR. Logistic regression analysis was performed to assess serum adropin as an independent predictor of diabetic nephropathy. Receiver operating characteristic (ROC) curve analysis was conducted to determine the optimal cut-off

value of serum adropin for predicting nephropathy. A p-value of less than 0.05 was considered statistically significant, while p-values less than 0.001 were considered highly significant.

Results

Table 1 presents a comparative analysis of demographic, clinical, and biochemical parameters between Group A (type 2 diabetes mellitus with nephropathy) and Group B (type 2 diabetes mellitus without nephropathy).

The two groups were comparable with respect to age and gender distribution, showing no statistically significant difference. However, BMI was marginally higher in Group A and reached statistical significance. The duration of diabetes was significantly longer in Group A, indicating a strong association between prolonged disease duration and development of nephropathy. Both systolic and diastolic blood pressures were significantly higher in Group A, reflecting poorer cardiovascular risk profiles in patients with nephropathy. Glycaemic parameters demonstrated significantly elevated fasting blood glucose and HbA1C levels in Group A, indicating poorer glycaemic control among patients with nephropathy. Regarding renal parameters, serum urea did not differ significantly between groups, whereas serum creatinine was higher and eGFR was significantly lower in Group A, confirming impaired renal function. Urine albumin–creatinine ratio (ACR) was markedly elevated in Group A, consistent with established nephropathy.

The primary outcome, serum adropin levels, was significantly lower in Group A compared to Group B. Lipid profile parameters showed atherogenic dyslipidaemia in Group A, with higher total cholesterol, triglycerides, LDL cholesterol, and lower HDL cholesterol, all statistically significant.

Figure 1 illustrates the comparison of serum adropin levels between the two groups. Group A (with nephropathy) demonstrates markedly lower mean serum adropin levels (89.9 ng/L), whereas Group B (without nephropathy) shows significantly higher levels (127.5 ng/L), with a highly significant difference.

Table 1. Comparison of all clinical and biochemical parameters between Groups

Parameter	Group A (n=45) Mean ± SD	Group B (n=45) Mean ± SD	p Value
DEMOGRAPHIC & ANTHROPOMETRIC			
Age (years)	58.8 ± 5.1	58.1 ± 7.0	0.72 NS
Gender (M/F)	24 / 21	24 / 21	1.00 NS
BMI (Kg/m²)	24.11 ± 1.04	23.51 ± 1.07	0.05 *
Duration of DM (years)	8.2 ± 1.2	7.6 ± 1.2	0.003 **
Systolic BP (mmHg)	126 ± 18.6	116.4 ± 10	0.005 **
Diastolic BP (mmHg)	81.7 ± 11.1	76 ± 7.5	0.001 **
GLYCAEMIC PARAMETERS			
FBG (mg/dL)	**190.1 ± 69**	**118.7 ± 19.5**	**0.001 ** **
HbA1C (%)	**8.44 ± 1.3**	**6.9 ± 0.65**	**0.001 ** **
RENAL PARAMETERS			

Urea (mg/dL)	27 ± 6.06	25.07 ± 6.42	0.24 NS
Creatinine (mg/dL)	0.87 ± 0.25	0.61 ± 0.07	0.05 *
eGFR (mL/min/1.73m²)	**84.82 ± 20.6**	**106.6 ± 5.4**	**0.001 ** **
ACR (mg/g)	**124.8 ± 66.9**	**16.6 ± 7.14**	**0.001 ** **
PRIMARY OUTCOME: SERUM ADROPIN			
Serum Adropin (ng/L)	**89.9 ± 55.2**	**127.5 ± 51.9**	**0.001 ** **
LIPID PROFILE			
Total Cholesterol (mg/dL)	209.5 ± 40.8	140 ± 26.5	0.001 **
Triglycerides (mg/dL)	180 ± 59.6	103.5 ± 25.7	0.001 **
HDL Cholesterol (mg/dL)	23.4 ± 4.7	49.8 ± 4.0	0.001 **
LDL Cholesterol (mg/dL)	150.7 ± 39.7	69.6 ± 24.8	0.001 **

*Group A = Type 2 DM with nephropathy; Group B = Type 2 DM without nephropathy. NS = Not Significant; *Significant (p<0.05); **Highly Significant (p<0.001).*

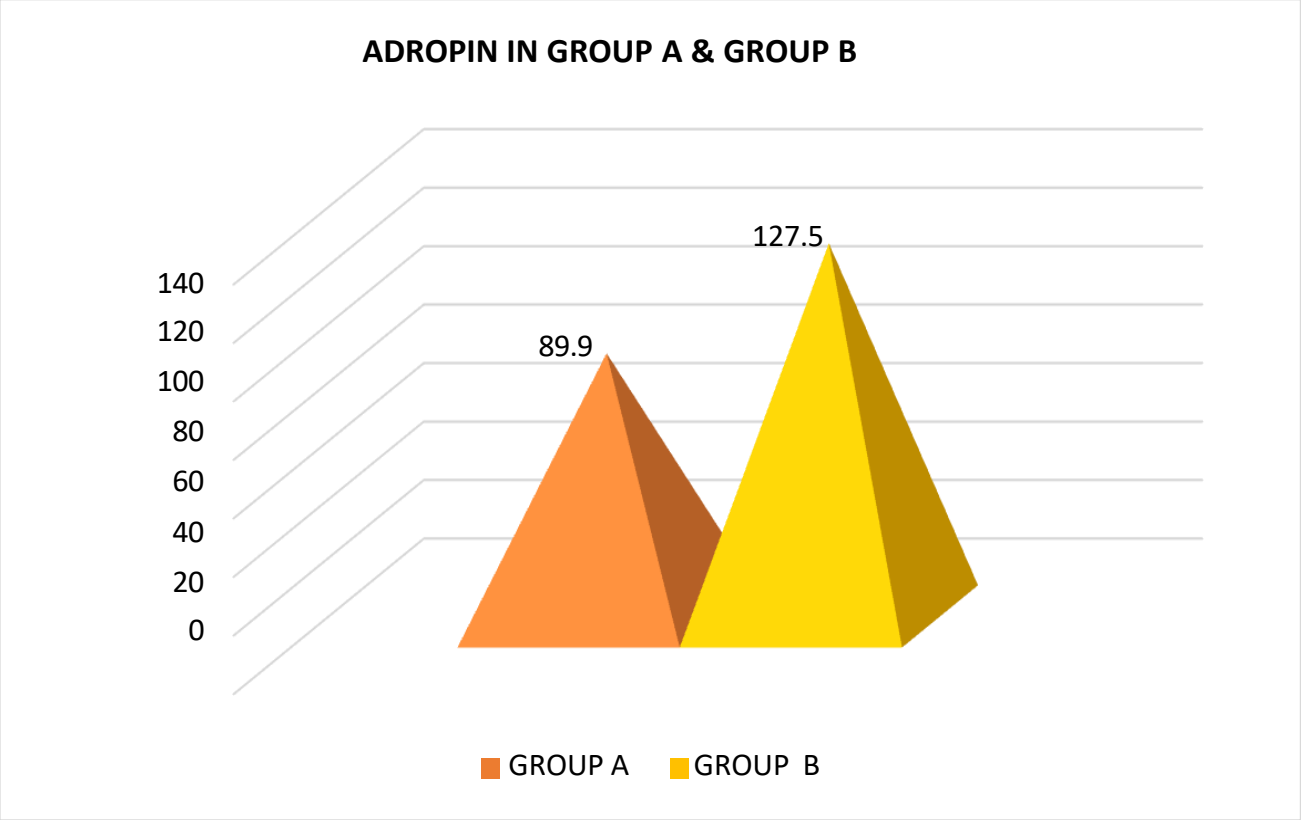


Figure 1. Serum Adropin levels in Group A vs Group B

Table 2 depicts Pearson's correlation analysis between serum adropin levels and various clinical and biochemical parameters in both Group A (type 2 diabetes mellitus with nephropathy) and Group B (without nephropathy).

In Group A, serum adropin demonstrated significant negative correlations with BMI, fasting blood glucose (FBG), HbA1C and urine albumin–creatinine ratio (ACR). Among these, the strongest negative correlation (r=-0.779, p<0.001) was observed with ACR,

indicating that lower adropin levels are associated with higher albuminuria. Adropin also showed a significant positive correlation with eGFR (r=0.594, p<0.001), suggesting better renal function with higher adropin levels. Additionally, a mild but significant positive correlation was noted with HDL cholesterol (r=0.32, p<0.05). Other lipid parameters, urea, creatinine, and total cholesterol did not show statistically significant correlations.

In Group B, no significant correlations were observed between serum adropin and most clinical or biochemical parameters. The relationships were weak and statistically non-significant, indicating a relatively stable metabolic and renal profile in patients without nephropathy.

Table 2. Pearson's correlation coefficients of serum adropin with clinical parameters

Parameter	Group A r value	Group A p value	Group B r value	Group B p value
ANTHROPOMETRIC				
BMI (Kg/m²)	**−0.60**	**<0.0001***	−0.12	0.434
GLYCAEMIC PARAMETERS				
FBG (mg/dL)	**−0.36**	**0.013***	−0.02	0.896
HbA1C (%)	**−0.51**	**<0.0001***	−0.04	0.751
RENAL PARAMETERS				
Urea (mg/dL)	−0.10	0.493 NS	−0.01	0.928
Creatinine (mg/dL)	−0.20	0.241 NS	−0.04	0.751
ACR (mg/g)	**−0.779**	**<0.0001***	−0.07	0.613
eGFR (mL/min/1.73m²)	**+0.594**	**<0.0001***	+0.11	0.471
LIPID PROFILE				
Total Cholesterol (mg/dL)	−0.25	0.09 NS	−0.03	0.831
Triglycerides (mg/dL)	−0.16	0.281 NS	−0.16	0.273
HDL Cholesterol (mg/dL)	**+0.32**	**0.031***	+0.08	0.574
LDL Cholesterol (mg/dL)	−0.24	0.117 NS	+0.01	0.925

*Pearson's correlation. *Significant at 0.05 level, 2-tailed. NS=Not significant. Highlighted cells = strongest correlations.*

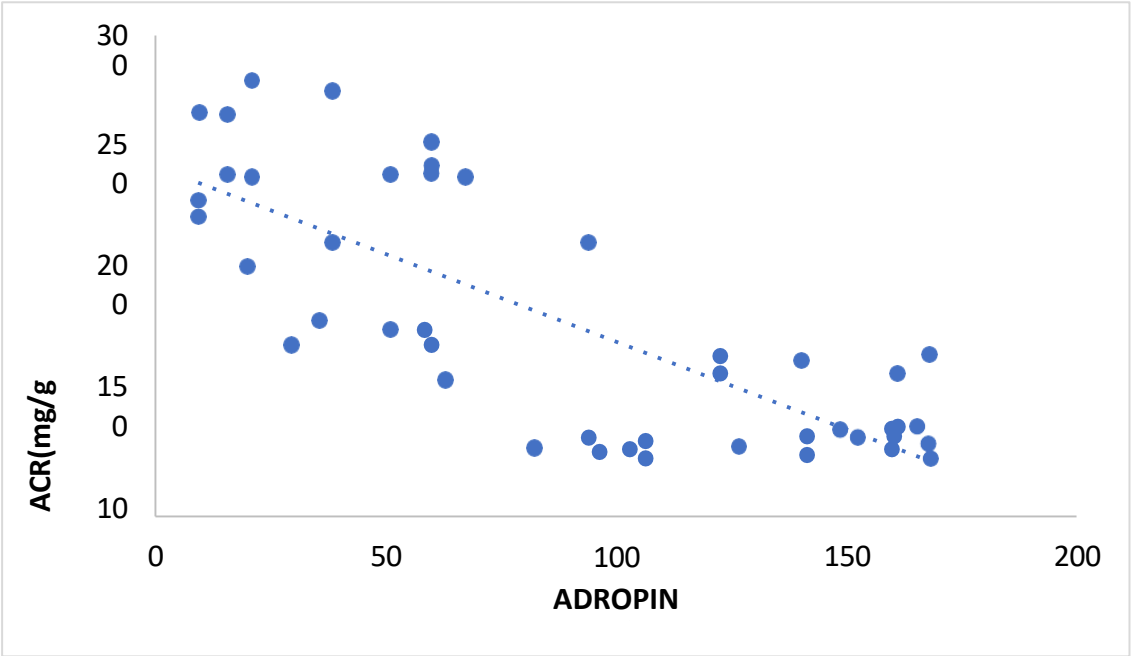


Figure 2. Scatter plot —Correlation between serum Adropin and urine ACR in Group A.

Figure 2 presents a scatter plot illustrating the correlation between serum adropin levels and urine ACR in Group A. The plot shows a clear downward trend, indicating a negative correlation between adropin and ACR. As serum adropin levels increase, urine ACR values decrease.

Table 3 presents the results of linear regression analysis with eGFR as the dependent variable to identify independent determinants of renal function.

Among the variables analysed, urine albumin–creatinine ratio (ACR) showed a strong and statistically significant negative association with eGFR, indicating that higher albuminuria is independently associated with reduced renal function. Serum adropin demonstrated a positive association with eGFR, suggesting a potential protective role; however, this association was borderline significant. Fasting blood glucose (FBG) and HbA1C did not show statistically significant associations with eGFR in the multivariate model, indicating that their effects may be mediated through other variables or are less influential when adjusted for confounders.

Table 3. Linear regression (eGFR as dependent variable)

Variable	B	Std. Error	Beta	p Value
ACR	−0.131	0.030	−0.530	**0.000 ***
Adropin	0.061	0.032	0.184	0.051
FBG	0.043	0.034	0.142	0.219 NS
HbA1C	0.642	1.679	0.045	0.703 NS

**Significant at 0.05 level, 2-tailed. NS=Not significant.*

Table 4 shows the results of logistic regression analysis with diabetic nephropathy as the dependent variable, identifying independent predictors of nephropathy.

Serum adropin demonstrated a statistically significant inverse association with diabetic nephropathy, with lower levels associated with higher odds of nephropathy. Duration of diabetes, systolic blood pressure, fasting blood glucose, and HbA1C were all significantly associated with increased risk of nephropathy, indicating their role as established risk factors. Conversely, eGFR showed a protective association, with higher eGFR linked to reduced odds of nephropathy.

Table 4. Logistic Regression Analysis (Diabetic Nephropathy as dependent variable)

Variable	Adjusted Odds Ratio (95% CI)	p Value
Serum Adropin	**0.987 (0.97–0.99)**	**0.002 ***
Duration of DM	1.5 (1.06–2.16)	0.021
Systolic BP	1.04 (1.01–1.07)	0.005
FBG	1.08 (1.05–1.12)	0.001
HbA1C	5.1 (2.57–10.2)	0.001
eGFR	0.79 (0.70–0.90)	0.001

**Significant at 0.05 level, 2-tailed. NS=Not significant.*

Figure 3 illustrates the Receiver Operating Characteristic (ROC) curve for serum adropin in predicting diabetic nephropathy. The ROC curve demonstrates the trade-off between sensitivity and specificity across various cut-off values of serum adropin. The curve lies above the diagonal reference line, indicating that serum adropin has discriminatory ability in differentiating patients with and without nephropathy. The area under the curve (AUC) is 0.701 with a standard error of 0.055 and is statistically significant ($p = 0.001$). The 95% confidence interval ranges from 0.594 to 0.809, indicating moderate diagnostic accuracy. A cut-off value of 80 ng/L for serum adropin was identified as optimal, yielding a sensitivity of 75% and specificity of 72%.

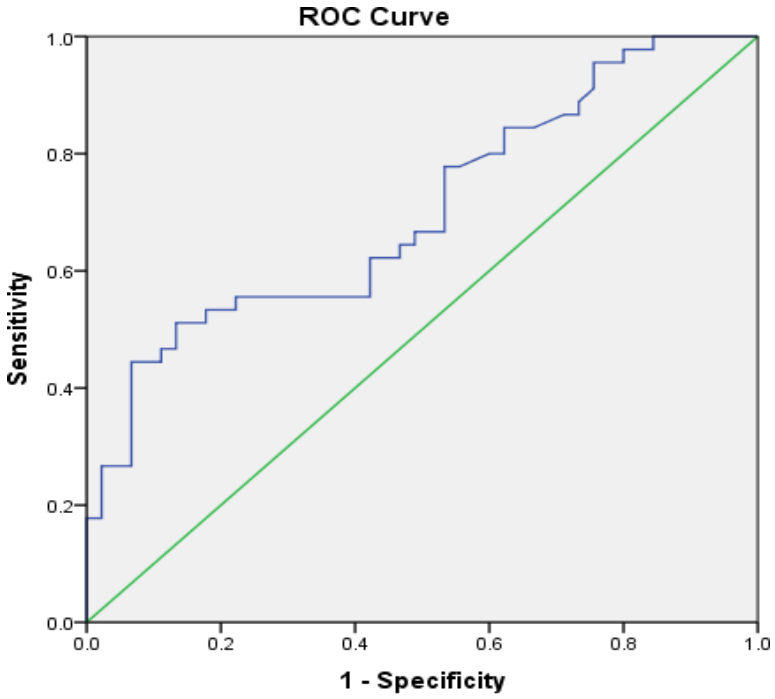


Figure 3. ROC curve for serum Adropin predicting diabetic nephropathy.

Discussion

The present study evaluated the role of serum adropin in type 2 diabetes mellitus (T2DM) patients with and without nephropathy and explored its association with clinical, metabolic, and renal parameters. The findings demonstrate that patients with diabetic nephropathy exhibit significantly poorer glycaemic control, higher cardiovascular risk factors, and marked renal dysfunction, along with significantly reduced serum adropin levels. These observations are consistent with the established pathophysiological understanding of diabetic kidney disease (DKD), where chronic hyperglycaemia and metabolic dysregulation contribute to progressive renal damage [1,3].

In the current study, patients with nephropathy had a significantly longer duration of diabetes, higher systolic and diastolic blood pressure, and increased BMI compared to those without nephropathy. These findings align with previous reports identifying duration of diabetes, hypertension, and obesity as major risk

factors for DKD progression [17-19]. The strong association between elevated blood pressure and nephropathy observed in our study supports the concept of hemodynamic stress contributing to glomerular injury and sclerosis [20]. Similarly, the higher BMI observed in Group A reflects the contributory role of obesity in accelerating renal damage through inflammatory and metabolic pathways [19].

Glycaemic parameters in our study revealed significantly higher fasting blood glucose and HbA1C levels in patients with nephropathy, indicating poor glycaemic control. This is in agreement with earlier studies which have demonstrated that chronic hyperglycaemia drives renal injury through mechanisms such as advanced glycation end product formation and activation of protein kinase C pathways. [4,5] Hussain et al. [3] and Tziomalos et al. [7] also emphasized that poor glycaemic control is a key determinant in the onset and progression of diabetic nephropathy. However, in multivariate analysis, glycaemic parameters did not independently predict eGFR decline in our study, suggesting that their effect may be mediated through downstream renal damage markers such as albuminuria.

Renal parameters in our study showed significantly higher serum creatinine and ACR levels along with reduced eGFR in patients with nephropathy. The markedly elevated ACR in Group A confirms its role as a sensitive marker of glomerular damage. However, as highlighted by Persson and Rossing,[6] traditional markers such as creatinine and ACR may not reliably detect early renal injury, underscoring the need for novel biomarkers. In our regression analysis, ACR emerged as the strongest independent predictor of reduced eGFR, reinforcing its importance in assessing renal disease severity.

The primary finding in this study is the significantly lower serum adropin in Group A (89.9±55.2 ng/L) vs Group B (127.5±51.9 ng/L, $p<0.001$) that supports the hypothesis that adropin deficiency plays a role in nephropathy, which is consistent with Hu and Chen who observed lower adropin in diabetic patients with nephropathy and negative correlation with ACR. [16] Similarly, Maciorkowska et al. [21] reported reduced adropin in CKD patients.

Correlation analysis in our study further demonstrated that serum adropin levels were negatively associated with BMI, FBG, HbA1C, and ACR, and positively associated with eGFR and HDL cholesterol in patients with nephropathy. The strong inverse correlation between adropin and ACR suggests that adropin may reflect the severity of albuminuria and glomerular damage. These findings are biologically plausible, as adropin is known to regulate endothelial function, reduce inflammatory cytokine expression, and improve nitric oxide bioavailability [10,11]. Its anti-inflammatory and vasoprotective effects may help attenuate renal injury, thereby explaining its positive association with preserved renal function.

The lipid profile findings in our study showed significant dyslipidaemia in patients with nephropathy, characterized by elevated triglycerides, total cholesterol, and LDL levels, along with reduced HDL cholesterol. This is consistent with previous literature indicating that dyslipidaemia contributes to renal injury through lipid accumulation, oxidative stress, and inflammatory pathways [20]. The observed positive correlation between adropin and

HDL cholesterol supports earlier reports suggesting that adropin plays a role in lipid metabolism and may exert protective cardiovascular effects [9,22].

In multiple regression analysis, although serum adropin showed a positive association with eGFR, it did not reach strong statistical significance, suggesting that while adropin may influence renal function, its independent predictive value requires further validation. However, in logistic regression analysis, serum adropin emerged as a significant independent predictor of diabetic nephropathy, even after adjusting for conventional risk factors such as duration of diabetes, BMI, blood pressure, and glycaemic parameters. This finding highlights the potential of adropin as a clinically relevant biomarker for nephropathy risk stratification.

The ROC curve analysis in our study demonstrated that serum adropin has moderate diagnostic accuracy for predicting diabetic nephropathy, with an AUC of 0.701. This indicates that while adropin alone may not be sufficient as a standalone diagnostic tool, it can serve as a valuable adjunct marker in combination with traditional parameters such as ACR and eGFR. Similar studies evaluating emerging biomarkers in DKD have also reported moderate predictive performance, emphasizing the need for multi-marker approaches for early detection [18].

Limitations

The study population of 90 needs to be expanded for better generalizability. The absence of a healthy control group limits comparisons across the disease spectrum. The cross-sectional design of the study and unaddressed potential confounders like Diet, Medications etc., prevents causal inferences, and a prospective study is recommended. Additionally, patients with end-stage renal disease (ESRD) on renal replacement therapy were excluded, which limits conclusions in advanced stages of the disease.

Conclusion

The findings from this study demonstrates that reduced serum adropin levels are significantly associated with diabetic nephropathy and correlate with key markers of renal dysfunction. These results suggest that serum adropin may serve as a potential adjunct biomarker and an independent predictor for risk stratification in diabetic nephropathy. However, its role in early detection warrants further validation through larger, prospective studies.

Data availability statement

The datasets generated and analysed in this study are available from the corresponding author on reasonable request. They are not publicly shared because they contain sensitive information that could indirectly identify participants.

Ethical Approval

This study has been approved by the Institution Ethics Committee of Madras Medical College carrying certificate number 25082018.

Informed Consent

Written informed consent was obtained from all participants after explaining the study procedures, potential risks and benefits. Consent covered both participation and publication of anonymised findings, with assurance of confidentiality and data privacy.

Conflicts of interest

The authors declare that they do not have conflict of interest.

Funding

No funding was received for conducting this study.

Acknowledgements

Use of AI: Authors declare the use of Claude (Claude.ai) to assist with manuscript preparation and improving overall language clarity. After using this tool, the authors reviewed and edited the content and took full responsibility for the contents of this article.

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