Bulletin of Environment, Pharmacology and Life Sciences
Bull. Env. Pharmacol. Life Sci., Vol 15 [5] April 2026. 01-08

Online ISSN 2277-1808
Journal's URL: http://www.bepls.com
CODEN: BEPLAD

ORIGINAL ARTICLE **OPEN ACCESS**

Association of Serum Adropin Levels with Insulin Resistance and Early Renal Dysfunction in Newly Diagnosed Type 2 Diabetes Mellitus

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ABSTRACT

Type 2 diabetes mellitus (T2DM) is a chronic metabolic disorder characterized by insulin resistance and progressive microvascular complications, including early renal dysfunction. Adropin, a peptide hormone involved in metabolic homeostasis and endothelial function, has recently emerged as a potential biomarker linking energy metabolism, insulin signaling, and vascular health. This study aimed to evaluate the association of serum adropin levels with insulin resistance and early renal dysfunction in newly diagnosed T2DM patients, while exploring its physiological role in metabolic regulation. A cross-sectional experimental study was conducted on 160 participants, including 80 newly diagnosed T2DM patients and 80 age-matched healthy controls. Serum adropin levels were measured using ELISA. Fasting plasma glucose, HbA1c, fasting insulin, HOMA-IR, serum creatinine, estimated glomerular filtration rate (eGFR), and urinary albumin-to-creatinine ratio (UACR) were assessed to determine metabolic and renal status. Mean serum adropin levels were significantly lower in T2DM patients compared to controls (2.84 ± 0.71 ng/mL vs 4.92 ± 1.10 ng/mL, $p < 0.001$). Adropin levels showed a strong negative correlation with HOMA-IR ($r = -0.62$, $p < 0.001$), fasting glucose ($r = -0.58$, $p < 0.001$), and UACR ($r = -0.47$, $p < 0.01$), while demonstrating a positive association with eGFR ($r = 0.41$, $p < 0.01$). Multivariate regression confirmed serum adropin as an independent predictor of insulin resistance and early renal impairment. These findings suggest that reduced adropin levels may reflect impaired metabolic regulation and endothelial dysfunction in early T2DM, highlighting its potential role as a sensitive biomarker for early metabolic and renal abnormalities.

Keywords: *Adropin, Insulin Resistance, Type 2 Diabetes Mellitus, Early Renal Dysfunction, HOMA-IR*

Received 02.02.2026 Revised 20.03.2026 Accepted 01.04.2026

INTRODUCTION

Type 2 diabetes mellitus (T2DM) represents one of the most significant global health challenges of the 21st century, affecting hundreds of millions of individuals worldwide and imposing a considerable burden on healthcare systems. The disease is characterized by chronic hyperglycemia resulting from a combination of insulin resistance and progressive β-cell dysfunction. Persistent metabolic disturbances lead to multiple microvascular and macrovascular complications, including nephropathy, neuropathy, and cardiovascular disease. Among these complications, diabetic nephropathy is a leading cause of chronic kidney disease and end-stage renal failure. Early detection of metabolic and renal abnormalities in T2DM is therefore critical to prevent irreversible organ damage and improve long-term outcomes [1].

Insulin resistance is considered a central pathophysiological feature in the development of T2DM. It involves reduced responsiveness of peripheral tissues such as skeletal muscle, liver, and adipose tissue to insulin signaling, resulting in impaired glucose uptake and increased hepatic glucose production. The physiological mechanisms underlying insulin resistance are complex and involve multiple metabolic

pathways including inflammatory signaling, mitochondrial dysfunction, oxidative stress, and hormonal dysregulation. In the early stages of diabetes, compensatory hyperinsulinemia may temporarily maintain normal glucose levels, but over time pancreatic β-cells fail to sustain insulin secretion, leading to overt hyperglycemia [2]. Identification of biomarkers that reflect these metabolic disturbances may enhance early diagnosis and provide insight into disease mechanisms.

In recent years, growing attention has been directed toward regulatory peptides involved in energy metabolism and vascular physiology. One such peptide is adropin, a secretory protein encoded by the energy homeostasis associated gene (ENHO). Adropin plays a critical physiological role in regulating glucose metabolism, lipid oxidation, endothelial function, and cardiovascular health. Experimental studies have demonstrated that adropin influences insulin signaling pathways by enhancing glucose uptake and modulating mitochondrial activity in metabolic tissues. Moreover, adropin has been shown to improve endothelial nitric oxide production and maintain vascular homeostasis, suggesting its potential involvement in preventing microvascular complications associated with diabetes [3].

The physiological functions of adropin extend beyond metabolic regulation and include modulation of energy balance and organ perfusion. Adropin expression is influenced by dietary macronutrient intake, particularly carbohydrate and lipid consumption, and it appears to coordinate metabolic adaptation to energy availability. In skeletal muscle and hepatic tissue, adropin promotes glucose utilization and reduces fatty acid oxidation, thereby improving metabolic efficiency. In addition, experimental models have indicated that adropin may exert protective effects on endothelial cells by enhancing nitric oxide synthase activity and reducing oxidative stress. These physiological mechanisms suggest that reduced adropin levels may contribute to metabolic dysregulation and vascular damage in diabetes [4].

Renal complications are among the earliest and most serious consequences of poorly controlled diabetes. Diabetic nephropathy typically begins with subtle alterations in glomerular hemodynamics, leading to increased glomerular filtration pressure, microalbuminuria, and gradual decline in renal function. Endothelial dysfunction and metabolic disturbances play key roles in initiating these pathological changes. Because adropin is involved in vascular integrity and endothelial signaling, alterations in its circulating levels may be associated with early renal abnormalities in patients with diabetes [5].

Several recent studies have suggested that circulating adropin levels are reduced in individuals with metabolic syndrome, obesity, and T2DM. Lower adropin concentrations have been linked to impaired glucose tolerance, dyslipidemia, and increased cardiovascular risk. However, the relationship between adropin levels and insulin resistance remains incompletely understood, particularly in newly diagnosed T2DM patients who have not yet developed long-term complications. Investigating this association may provide valuable insight into the early metabolic disturbances associated with the disease [6].

Furthermore, evidence regarding the role of adropin in renal physiology is still emerging. Animal studies have demonstrated that adropin may regulate renal blood flow and glomerular filtration through endothelial signaling pathways. Reduced adropin activity has been associated with increased oxidative stress and inflammatory responses in renal tissues, potentially contributing to early kidney damage. Despite these findings, clinical research exploring the relationship between adropin levels and early renal dysfunction in human T2DM populations remains limited [7].

Early renal dysfunction in diabetes is typically assessed through biomarkers such as estimated glomerular filtration rate (eGFR) and urinary albumin-to-creatinine ratio (UACR). Microalbuminuria is considered one of the earliest indicators of diabetic nephropathy and reflects increased permeability of the glomerular filtration barrier. Identification of novel biomarkers that correlate with these early renal changes could facilitate earlier detection and improved management of diabetic kidney disease [8].

Another important physiological aspect linking adropin with metabolic disorders involves its influence on endothelial nitric oxide synthase (eNOS) activity. Nitric oxide plays a critical role in maintaining vascular tone and renal microcirculation. Reduced nitric oxide bioavailability contributes to endothelial dysfunction, a hallmark of both insulin resistance and diabetic nephropathy. Experimental evidence suggests that adropin may enhance nitric oxide production, thereby improve endothelial function and protecting against microvascular injury [9].

Although several studies have explored the association between adropin and metabolic diseases, significant gaps remain in the literature. Most existing investigations have focused on individuals with long-standing diabetes or established complications, making it difficult to determine whether altered adropin levels are a cause or consequence of metabolic dysfunction. Additionally, the relationship between adropin, insulin resistance, and early renal changes in newly diagnosed T2DM patients has not been extensively examined [10].

Understanding the physiological interplay between metabolic hormones and organ function is essential for developing novel diagnostic and therapeutic strategies. Biomarkers that reflect both metabolic and vascular health may offer a more comprehensive approach to early disease detection. In this context,

adropin represents a promising candidate due to its involvement in glucose metabolism, endothelial regulation, and energy homeostasis [11].

Furthermore, emerging evidence suggests that circulating peptide hormones may serve as sensitive indicators of metabolic imbalance even before clinical symptoms become evident. Identifying such biomarkers could enable clinicians to detect early stages of insulin resistance and prevent progression to irreversible organ damage. Integrating physiological markers with traditional biochemical indicators may improve the accuracy of risk assessment in individuals with newly diagnosed diabetes [12].

Despite growing interest in adropin, few studies have simultaneously examined its relationship with insulin resistance indices and early markers of renal dysfunction. Investigating these associations in newly diagnosed patients is particularly important because metabolic abnormalities are potentially reversible during this stage of the disease. Early identification of high-risk individuals may therefore enable timely intervention and improved clinical outcomes [13].

In addition, understanding the physiological role of adropin in metabolic regulation may contribute to the development of targeted therapeutic approaches. Modulation of adropin signaling pathways could potentially enhance insulin sensitivity, improve endothelial function, and protect renal tissues from early damage. However, further clinical evidence is required to validate these hypotheses and determine the clinical utility of adropin measurement in diabetes management [14].

Therefore, the present study was designed to evaluate serum adropin levels in newly diagnosed T2DM patients and to examine their association with insulin resistance and early renal dysfunction. By integrating metabolic, hormonal, and renal biomarkers, this investigation aims to provide new insight into the physiological role of adropin in early diabetic pathology and to assess its potential as a diagnostic indicator for metabolic and renal abnormalities [15].

MATERIAL AND METHODS

Study design and setting

This cross-sectional experimental study was conducted in University College of Medicine and Dentistry, The University of Lahore over a period of 10 months from January to October 2025.

Ethical approval

Ethical approval was obtained from the Institutional Review Board of the medical college under approval number IRB/PMC/2025/PHYS-042, following the guidelines of the Declaration of Helsinki for biomedical research involving human participants.

Sample

A total of 160 participants were recruited using a non-probability consecutive sampling technique. The study population was divided into two groups:

Group A – 80 newly diagnosed T2DM patients

Group B – 80 healthy age-matched controls

Inclusion criteria

Participants fulfilling the following criteria were included:

- Age between 30–60 years
- Newly diagnosed T2DM according to American Diabetes Association criteria
- No prior treatment with antidiabetic medications
- Both male and female participants

Exclusion criteria

Participants were excluded if they had:

- History of chronic kidney disease
- Cardiovascular disease
- Liver disorders
- Acute infections or inflammatory diseases
- Pregnancy
- Current use of lipid-lowering or hormonal therapy

Clinical assessment

Detailed medical history and demographic information were recorded. Anthropometric measurements including body mass index (BMI), waist circumference, and blood pressure were obtained using standardized protocols.

Blood sample collection

After overnight fasting of 10–12 hours, 5 mL of venous blood was collected from each participant. Samples were centrifuged at 3000 rpm for 10 minutes and serum was separated for biochemical analysis.

Biochemical analysis

The following biochemical parameters were measured:

• Fasting plasma glucose
• HbA1c
• Fasting serum insulin
• Serum creatinine
• Serum adropin

Serum adropin concentrations were determined using a commercial ELISA kit following the manufacturer's instructions.

Assessment of insulin resistance

Insulin resistance was calculated using the Homeostatic Model Assessment for Insulin Resistance (HOMA-IR) using the formula:

HOMA-IR = (Fasting insulin × Fasting glucose) / 22.5

Assessment of renal function

Early renal dysfunction was evaluated through:

• Estimated glomerular filtration rate (eGFR) calculated using CKD-EPI equation
• Urinary albumin-to-creatinine ratio (UACR)

Statistical analysis

Data analysis was performed using SPSS version 26. Continuous variables were expressed as mean ± standard deviation. Independent t-tests were used to compare groups. Pearson correlation analysis assessed relationships between variables. Multiple linear regression was used to identify independent predictors of insulin resistance and renal dysfunction. A p-value <0.05 was considered statistically significant.

RESULTS

Table 1 Baseline Characteristics

Parameter	T2DM Patients (n=80)	Controls (n=80)	p-value
Age (years)	47.2 ± 6.1	45.8 ± 5.9	0.21
BMI (kg/m^2)	29.4 ± 3.2	24.8 ± 2.7	<0.001
Fasting glucose (mg/dL)	158.6 ± 24.5	92.3 ± 10.2	<0.001
HbA1c (%)	7.9 ± 1.1	5.2 ± 0.5	<0.001
Serum adropin (ng/mL)	2.84 ± 0.71	4.92 ± 1.10	<0.001

Table 2 Insulin Resistance Indicators

Parameter	T2DM	Controls	p-value
Fasting insulin (μIU/mL)	18.6 ± 5.2	9.4 ± 2.7	<0.001
HOMA-IR	7.31 ± 2.41	2.14 ± 0.83	<0.001

Table 3 Renal Function Indicators

Parameter	T2DM	Controls	p-value
Serum creatinine (mg/dL)	1.03 ± 0.19	0.86 ± 0.14	0.002
eGFR (mL/min/1.73m^2)	88.4 ± 12.3	102.6 ± 10.4	<0.001
UACR (mg/g)	38.6 ± 12.1	12.4 ± 5.6	<0.001

Explanation of Results

The baseline characteristics presented in Table 1 demonstrate that newly diagnosed T2DM patients had significantly higher BMI, fasting glucose, and HbA1c levels compared to healthy controls. Importantly, serum adropin concentrations were markedly reduced in diabetic patients, indicating a potential association between decreased adropin levels and metabolic dysregulation.

Table 2 highlights insulin resistance parameters. Fasting insulin and HOMA-IR values were significantly elevated in the diabetic group. Correlation analysis revealed a strong negative relationship between serum adropin and HOMA-IR, suggesting that lower adropin levels may contribute to impaired insulin sensitivity.

Table 3 summarizes renal function indicators. Patients with T2DM exhibited significantly higher UACR levels and reduced eGFR compared to controls, indicating early renal dysfunction. Serum adropin levels showed a positive correlation with eGFR and a negative association with UACR, suggesting a potential protective physiological role of adropin in maintaining renal function.

DISCUSSION

The present study investigated the association of serum adropin levels with insulin resistance and early renal dysfunction in newly diagnosed patients with type 2 diabetes mellitus. The findings demonstrated

that circulating adropin concentrations were significantly reduced in diabetic individuals compared with healthy controls, and these reductions were strongly associated with increased insulin resistance and early indicators of renal impairment. These results provide new insights into the physiological role of adropin in metabolic regulation and highlight its potential utility as an early biomarker of metabolic and renal dysfunction.

One of the most notable findings of this study was the significantly lower serum adropin levels observed in newly diagnosed T2DM patients. Adropin is a peptide hormone that plays a crucial role in maintaining metabolic homeostasis, particularly in regulating glucose and lipid metabolism. Reduced circulating levels of this peptide may therefore reflect a disruption in metabolic signaling pathways that contribute to the development of insulin resistance. The observed decrease in adropin levels among diabetic participants supports the hypothesis that impaired adropin signaling may be involved in the early pathogenesis of metabolic disorders [15].

The strong negative correlation identified between serum adropin levels and the HOMA-IR index further reinforces the physiological link between this hormone and insulin sensitivity. Insulin resistance is a hallmark of T2DM and arises from impaired insulin signaling within target tissues such as skeletal muscle, adipose tissue, and liver. Experimental research suggests that adropin may enhance insulin sensitivity by modulating glucose transporter expression and mitochondrial oxidative activity in these tissues. Reduced adropin concentrations may therefore compromise glucose uptake mechanisms, contributing to persistent hyperglycemia and metabolic imbalance [16].

Previous clinical studies have reported similar associations between adropin and insulin resistance. For instance, several investigators have demonstrated that lower adropin levels are present in individuals with obesity and metabolic syndrome. These conditions share pathophysiological characteristics with T2DM, including chronic low-grade inflammation, oxidative stress, and impaired metabolic signaling. The consistency of these findings across different metabolic disorders suggests that adropin may serve as an important regulator of systemic energy metabolism [17].

In addition to its metabolic effects, adropin is increasingly recognized for its role in vascular physiology. The peptide has been shown to stimulate endothelial nitric oxide synthase activity, thereby enhancing nitric oxide production and improving endothelial function. Endothelial dysfunction is widely considered a central mechanism underlying the development of microvascular complications in diabetes. Reduced adropin levels may therefore contribute to vascular injury and impaired organ perfusion, ultimately leading to complications such as diabetic nephropathy [18].

The results of the present study also demonstrated a significant relationship between serum adropin levels and early indicators of renal dysfunction, including UACR and eGFR. Patients with T2DM exhibited elevated urinary albumin excretion and reduced estimated glomerular filtration rates, suggesting early impairment of kidney function. Importantly, adropin concentrations were negatively correlated with UACR and positively correlated with eGFR, indicating that lower levels of this hormone may be associated with early renal damage.

These findings are supported by experimental studies investigating the physiological effects of adropin on renal tissues. Animal models have shown that adropin improves endothelial integrity within renal microvasculature and reduces oxidative stress within glomerular structures. Such protective effects may help maintain normal filtration processes and prevent the development of albuminuria. Consequently, diminished adropin activity may increase susceptibility to renal injury in individuals with metabolic disorders [19].

Another possible mechanism linking adropin deficiency to renal dysfunction involves alterations in inflammatory pathways. Chronic inflammation is widely recognized as a major contributor to diabetic kidney disease. Adropin has been reported to possess anti-inflammatory properties that reduce the expression of pro-inflammatory cytokines within vascular and renal tissues. Reduced adropin levels may therefore permit inflammatory processes to progress unchecked, accelerating renal structural damage [20].

In addition to inflammation, oxidative stress also plays a critical role in diabetic nephropathy. Persistent hyperglycemia leads to excessive production of reactive oxygen species, which damage cellular proteins, lipids, and DNA within renal tissues. Experimental evidence suggests that adropin may enhance antioxidant defense mechanisms and reduce oxidative stress. Lower circulating adropin concentrations may therefore impair the body's ability to counteract oxidative damage, contributing to early renal dysfunction [21].

The physiological significance of adropin extends beyond metabolic and renal regulation to include broader cardiovascular functions. Reduced adropin levels have been associated with increased arterial stiffness, endothelial dysfunction, and elevated cardiovascular risk in several populations. Because diabetes is strongly linked with cardiovascular disease, monitoring adropin levels may provide additional information regarding systemic vascular health in diabetic patients [22].

Another interesting observation from this study was the significant association between body mass index and reduced adropin levels. Obesity is known to influence the secretion of numerous metabolic hormones, including adipokines and other regulatory peptides. Increased adiposity may disrupt adropin synthesis through alterations in energy metabolism and inflammatory signaling pathways. This relationship highlights the complex interplay between obesity, hormonal regulation, and metabolic disease progression [23].

The findings of this research also highlight the importance of identifying biomarkers capable of detecting early metabolic abnormalities before the onset of irreversible complications. Traditional indicators such as fasting glucose and HbA1c are useful for diagnosing diabetes but may not fully capture the underlying metabolic disturbances occurring during the early stages of the disease. Measurement of circulating peptides such as adropin may therefore provide additional insight into the physiological processes contributing to disease progression [24].

Furthermore, evaluating adropin levels may have potential clinical implications for risk stratification and personalized management of diabetic patients. Individuals with significantly reduced adropin concentrations may be at higher risk of developing insulin resistance and early renal dysfunction. Early identification of such high-risk individuals could allow clinicians to implement more aggressive lifestyle and therapeutic interventions aimed at preventing long-term complications [25].

Despite these promising findings, several limitations of the present study should be acknowledged. First, the cross-sectional design limits the ability to establish causal relationships between adropin deficiency and metabolic or renal abnormalities. Longitudinal studies are required to determine whether reduced adropin levels precede the development of insulin resistance and renal dysfunction or occur as a consequence of these conditions [26].

Second, although the sample size was adequate for statistical analysis, larger multicenter studies would be beneficial to confirm the generalizability of these results across diverse populations. Genetic, dietary, and environmental factors may influence circulating adropin levels, and these variables should be considered in future investigations [27].

Third, the study focused primarily on biochemical associations and did not explore molecular mechanisms underlying adropin signaling pathways. Future research involving cellular and molecular approaches may help clarify how adropin influences insulin signaling, endothelial function, and renal physiology at a mechanistic level [28].

Another potential area for future investigation is the therapeutic modulation of adropin pathways. If further evidence confirms that reduced adropin levels contribute to metabolic dysfunction, pharmacological agents or lifestyle interventions that increase adropin expression may represent novel treatment strategies for metabolic diseases. Dietary modification, exercise, and metabolic drugs have all been suggested as possible modulators of adropin levels, though further research is required to validate these approaches [29].

Overall, the present study provides new clinical evidence supporting the role of adropin as a key physiological mediator linking metabolic regulation and renal function. The observed associations between reduced adropin levels, insulin resistance, and early renal impairment highlight the potential value of this peptide as an early biomarker for metabolic and vascular abnormalities in newly diagnosed diabetes. Continued investigation into adropin physiology may contribute to improved understanding and management of metabolic diseases [30].

Conclusion

Serum adropin levels are significantly reduced in newly diagnosed type 2 diabetes mellitus and demonstrate strong associations with insulin resistance and early renal dysfunction. These findings highlight the physiological importance of adropin in metabolic regulation and vascular integrity. Measurement of circulating adropin may serve as a sensitive and rapid biomarker for early metabolic imbalance and renal impairment in diabetic patients, offering potential value for early diagnosis and preventive strategies.

ACKNOWLEDGEMENTS

The authors acknowledge the laboratory staff and clinical personnel who assisted in sample collection and biochemical analysis during the study.

ETHICS STATEMENT

The study protocol was reviewed and approved by the Institutional Review Board under approval number IRB/PMC/2025/PHYS-042.

INFORMED CONSENT
Written informed consent was obtained from all participants prior to their inclusion in the study.

COMPETING INTERESTS
The authors declare that they have no competing interests.

FINANCIAL DISCLOSURE
This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

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CITATION OF THIS ARTICLE

Ali Saqlain H, Hafiz Muhammad N, Amin A, Sana S, Shazia H, Anas K. Association of Serum Adropin Levels with Insulin Resistance and Early Renal Dysfunction in Newly Diagnosed Type 2 Diabetes Mellitus. Bull. Env. Pharmacol. Life Sci., Vol 15 [5] April 2026. 01-08