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Original Article

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Assessment of Serum Adenosine Deaminase Levels in Diabetic Kidney Disease among Patients with Type 2 Diabetes Mellitus

Mohammad Hussain[1]*, Devendra Kumar[2], Vaibhav Shukla[3], Jalees Fatima[2], Ajay Kumar Mishra[2], Firdaus Jabeen[4], Daniyal Malik[5], Aman Aftab[6]

[1]Junior Resident, Department of Medicine, Era's Lucknow Medical College & Hospital, Lucknow, Uttar Pradesh, India
[2]Professor, Department of Medicine, Era's Lucknow Medical College & Hospital, Lucknow, Uttar Pradesh, India
[3]Professor & Head, Department of Medicine, Era's Lucknow Medical College & Hospital, Lucknow, Uttar Pradesh, India
[4]Assistant Professor, Department of Medicine, Era's Lucknow Medical College & Hospital, Lucknow, Uttar Pradesh, India
[5]Senior Resident, Department of Medicine, Era's Lucknow Medical College & Hospital, Lucknow, Uttar Pradesh, India
[6]Junior Resident, Department of Biochemistry, Era's Lucknow Medical College & Hospital, Lucknow, Uttar Pradesh, India

***Address for Correspondence:** Dr. Mohammad Hussain, Junior Resident, Department of Medicine, Era's Lucknow Medical College & Hospital, Lucknow-226003, U.P, India
E-mail: md123hussain78692@gmail.com

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ABSTRACT

Background: Diabetic kidney disease (DKD) is one of the most common microvascular complications of type 2 diabetes mellitus (T2DM) and the leading cause of chronic kidney disease worldwide. Adenosine deaminase (ADA), an enzyme involved in purine metabolism and inflammatory pathways, has been implicated in diabetes-related disorders. This study evaluated the relationship between serum ADA levels and DKD in patients with T2DM.

Methods: In this hospital-based cross-sectional study, 105 patients with T2DM (42 with DKD and 63 without DKD) were evaluated. Clinical, biochemical, and renal parameters were assessed, and serum ADA levels were determined by the enzymatic colorimetric method.

Results: Patients with DKD were older and had significantly higher systolic blood pressure, body mass index, duration of diabetes, HbA1c, serum creatinine, and dyslipidaemia. Mean serum ADA levels were significantly higher in the DKD group than in the non-DKD group (13.9 ± 3.4 vs. 9.8 ± 2.6 U/L; p<0.001). ADA >12 U/L was observed in 54.7% of DKD patients and 19.1% of non-DKD patients. Higher ADA levels were significantly associated with increased albuminuria, advanced KDIGO stages, and reduced estimated glomerular filtration rate. Elevated serum ADA remained an independent predictor of DKD after adjustment for confounding variables (adjusted OR=6.92, p=0.002). ROC analysis demonstrated good diagnostic performance (AUC=0.83), with an optimal cut-off value of 11.6 U/L showing 78.6% sensitivity and 73.0% specificity.

Conclusion: Elevated serum ADA levels are strongly associated with diabetic kidney disease and renal impairment in T2DM. ADA may serve as a simple, economical, and non-invasive biomarker for the early identification and monitoring of DKD.

Key-words: Albuminuria, Adenosine deaminase (ADA), Biomarker, Diabetic Kidney Disease (DKD), Diabetic Nephropathy, T2DM, HbA1c, Renal Dysfunction, eGFR

INTRODUCTION

Diabetes mellitus (DM) is one of the most important non-communicable diseases in the world and is a major challenge for public health systems.

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Diabetes prevalence has been increasing leading to huge morbidity, mortality and health care cost in both industrialized and developing countries [1].

According to the International Diabetes Federation (IDF), in 2021, diabetes was present in some 537 million people aged 20 to 79 years and this number is estimated to reach 783 million by 2045 [2]. Type 2 diabetes mellitus (T2DM) accounts for approximately 90% of all cases of diabetes and is mainly attributed to urbanization, sedentary lifestyle, unhealthy diet, obesity, and aging population [3]. The impact of the disease is felt most

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strongly in low and middle-income countries with limited access to healthcare services and preventative measures. India has a major share of the global diabetes burden. Recent estimates indicate that more than 100 million Indians are afflicted with diabetes and an even larger population is at risk due to pre-diabetes. The increase in T2DM has been accompanied by a rise in both microvascular and macrovascular sequelae, which are major causes of disability, reduced quality of life and premature death. Chronic hyperglycemia can cause progressive damage of various organs such as kidney, retina, peripheral nerves and cardiovascular system [4,5]. Thus, early identification of patients at risk of developing diabetes complications is essential for improving long term clinical outcomes.

T2DM is a multifactorial metabolic disorder characterized by insulin resistance, reduced insulin production and chronic hyperglycemia [6]. Recent years have witnessed increased interest in the role of persistent low-grade inflammation and immunological dysregulation in the etiology of T2DM [7]. Elevated inflammatory cytokines, elevated oxidative stress and changes in immune cell function have been associated with pathogenesis of insulin resistance and diabetes complications [8]. These data demonstrate such close links between metabolic abnormalities and inflammatory pathways in the development of T2DM.

ADA is a key enzyme of purine metabolism and an important marker of cell mediated immunity. It catalyses the deamination of adenosine to inosine and is highly expressed in lymphoid tissues, in particular T cells [9]. Adenosine itself has an important role in the regulation of glucose and lipid metabolism and possesses anti-inflammatory effects in many tissues [10]. ADA activity increases extracellular adenosine concentrations, and therefore reduces its beneficial metabolic and immunomodulatory activities. Higher blood ADA levels were observed in T2DM patients, particularly those with poor glycemic control, in several studies, which suggests a possible correlation between ADA activity, insulin resistance and systemic inflammation.

DKD is one of the most frequent and clinically important microvascular complications of T2DM. It is a major cause of chronic kidney disease and end-stage renal disease worldwide and is associated with increased cardiovascular morbidity and mortality. While DKD is expected to develop in nearly one-third of T2DM patients during the disease course [11]. Urinary albumin excretion and estimated glomerular filtration rate (eGFR) are often used to assess renal involvement, but these markers do not always represent early kidney damage. In some individuals, renal function may deteriorate prior to detection of clinically significant albuminuria, highlighting the limitations of existing diagnostic modalities. There is increasing evidence that inflammation is important in the initiation and progression of diabetic kidney disease. Chronic hyperglycemia results in increased oxidative stress, activation of pro-inflammatory signaling pathways, and infiltration of immune cells into renal tissues leading to structural and functional renal impairment [12]. ADA may be involved in the pathogenesis of diabetic kidney disease by modulating inflammation through adenosine metabolism. However, few studies have examined the association between blood ADA level and DKD, particularly in Indian patients.

Therefore, the present study was carried out to determine the blood adenosine deaminase levels in patients with type 2 diabetes mellitus and to assess its relationship with diabetic kidney disease and indices of renal function. This association may provide insight into the therapeutic relevance of ADA as a potential biomarker for early diagnosis and assessment of renal involvement in T2DM.

MATERIALS AND METHODS

Study Design and Setting- This hospital-based observational cross-sectional study was conducted in the Department of Medicine in collaboration with the Department of Biochemistry, Era's Lucknow Medical College and Hospital, a tertiary care teaching hospital, over a period of two years.

Study Participants- Adult patients with T2DM attending the outpatient and inpatient departments were screened for eligibility. A total of 105 eligible patients were recruited after obtaining written informed consent and were categorized into Group A (T2DM without diabetic kidney disease; n = 63) and Group B (T2DM with diabetic kidney disease; n = 42).

Inclusion and Exclusion Criteria- Patients aged >18 years with established T2DM were included. Patients with type 1 diabetes mellitus, steroid use within the preceding 90

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days, solid organ or hematological malignancies, chronic inflammatory diseases, acute infections, autoimmune disorders, diabetic ketoacidosis, and hyperosmolar hyperglycemic state were excluded.

Clinical and Anthropometric Assessment- Clinical evaluation included demographic characteristics, duration of diabetes, history of hypertension and dyslipidaemia, and antidiabetic treatment profile. Anthropometric measurements were performed with slight modifications according to Misra et al. Body weight and height were measured using standard instruments, and body mass index (BMI) was calculated as kg/m² according to Indian BMI classification criteria. Blood pressure was measured after five minutes of rest using a standard mercury sphygmomanometer. Two readings were obtained five minutes apart, and the average value was used for analysis.

Laboratory Investigations- After overnight fasting (8–10 hours), venous blood samples were collected under aseptic precautions. Glycaemic parameters included fasting plasma glucose, post-prandial plasma glucose, and glycated haemoglobin (HbA1c). Renal function assessment included serum creatinine, eGFR, and urinary albumin-to-creatinine ratio (UACR). eGFR was calculated using the CKD-EPI equation. Lipid profile and liver function tests were performed using standard laboratory methods.

Estimation of Serum Adenosine Deaminase- Serum ADA was estimated by the enzymatic colorimetric method with slight modification (Lisiê et al., 2019). Serum ADA concentrations were expressed as U/L and categorized into three groups: <10 U/L, 10–12 U/L, and >12 U/L.

Definition and Staging of Diabetic Kidney Disease- Diabetic kidney disease was defined according to Kidney Disease: Improving Global Outcomes (KDIGO) criteria as chronic kidney disease in patients with diabetes characterized by persistent albuminuria (UACR ≥30 mg/g), reduced eGFR (<60 mL/min/1.73 m²), or both for at least three months. Patients with DKD were further classified according to KDIGO glomerular filtration rate categories (G1–G5).

Statistical Analysis- Data were entered into Microsoft Excel and analysed using SPSS version 24.0. Continuous variables were expressed as mean ± standard deviation, whereas categorical variables were presented as frequencies and percentages. Student's t-test and chi-square test were used for group comparisons. Receiver operating characteristic (ROC) curve analysis was performed to evaluate the diagnostic performance of serum ADA for diabetic kidney disease. Multivariate logistic regression analysis was used to identify independent predictors of DKD. A p-value <0.05 was considered statistically significant.

Ethical Considerations- Ethical approval was obtained from the Institutional Ethics Committee of Era's Lucknow Medical College and Hospital before commencement of the study. Written informed consent was obtained from all participants. Patient confidentiality was maintained throughout the study by anonymizing all data, and the information was used exclusively for research purposes in accordance with institutional ethical guidelines.

RESULTS

The present study included 105 patients with T2DM, comprising 63 patients without DKD and 42 patients with DKD. Comparative analysis was performed to evaluate demographic characteristics, blood pressure profile, glycaemic status, biochemical parameters, renal function, and serum ADA levels.

Age distribution demonstrated that DKD was more prevalent among older individuals (Fig. 1). In the non-DKD group, the highest proportion of participants belonged to the 50–59 years age group (31.7%), whereas in the DKD group, the majority of patients were aged ≥60 years (42.9%). Male participants predominated in both groups, accounting for 60.3% of the non-DKD group and 64.3% of the DKD group, while females constituted 39.7% and 35.7%, respectively. These findings indicate that advancing age was more commonly associated with DKD, whereas gender distribution remained comparable between the groups.

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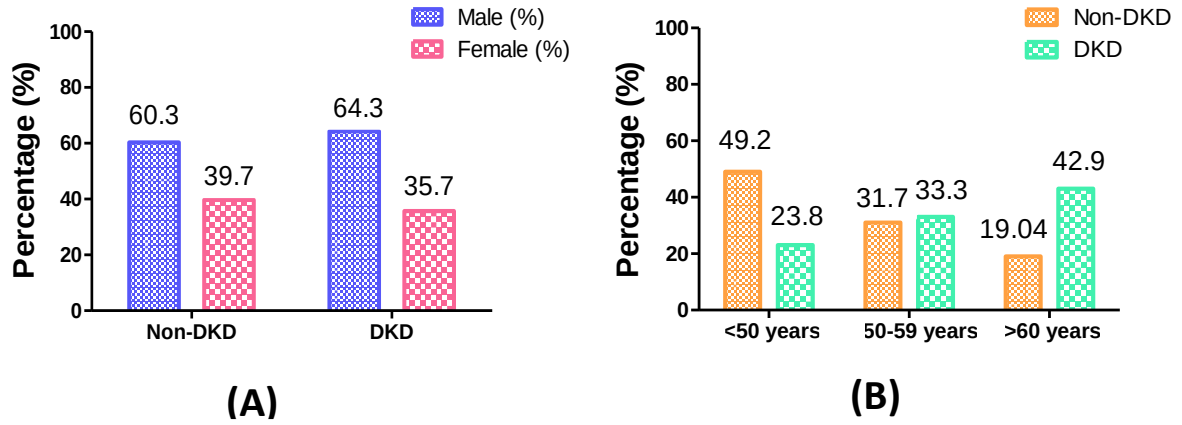

Fig. 1: Age and gender distribution in non-DKD and DKD cohorts.

Elevated systolic blood pressure was more frequently observed among patients with DKD (Fig. 2). Among non-DKD participants, 69.8% had systolic blood pressure <140 mmHg, whereas 59.5% of DKD patients had systolic blood pressure ≥140 mmHg. Diastolic blood pressure ≥90 mmHg was also slightly more prevalent among DKD patients (33.3%) than non-DKD patients (27%). Similarly, poor glycaemic control was significantly more common in the DKD group, where 83.3% of patients had HbA1c ≥7% compared with 58.7% in the non-DKD group. These findings demonstrate that hypertension and inadequate glycaemic control were strongly associated with diabetic kidney disease.

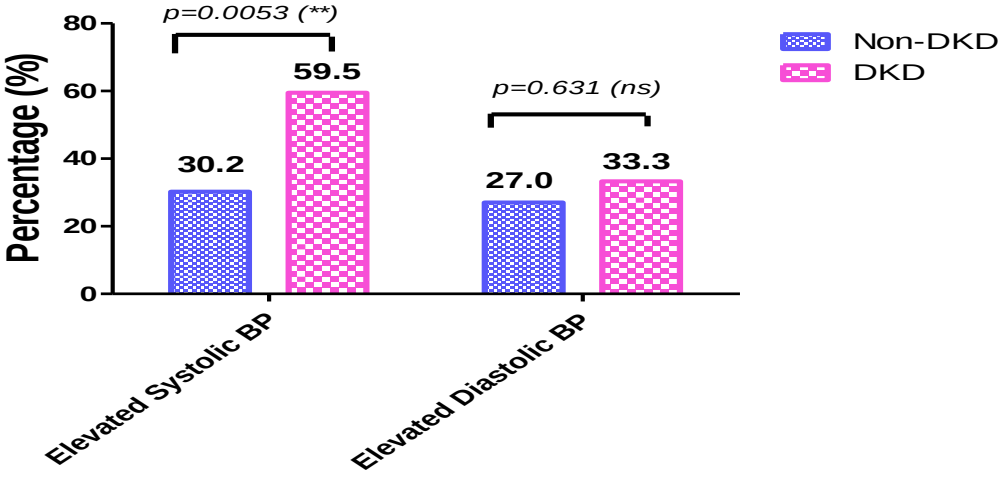

Fig. 2: Blood pressure profile in DKD and Non-DKD groups.

Poor glycaemic control is illustrated in Fig. 3. Controlled glycaemic status (HbA1c <7%) was more frequently observed in the non-DKD group, whereas the majority of DKD patients had HbA1c ≥7%. These findings indicate that inadequate glycaemic control remains an important factor associated with the development and progression of diabetic kidney disease.

Serum ADA levels were significantly higher among patients with DKD (Fig. 4). In the non-DKD group, 46.0% of participants had ADA levels <10 U/L, whereas only 14.3% of DKD patients belonged to this category. Conversely, ADA levels >12 U/L were observed in 54.7% of DKD patients compared with only 19.1% of non-DKD participants. The difference in ADA distribution was statistically significant ($\chi^2 = 17.38$, $p < 0.001$), indicating a strong association between elevated serum ADA levels and diabetic kidney disease.

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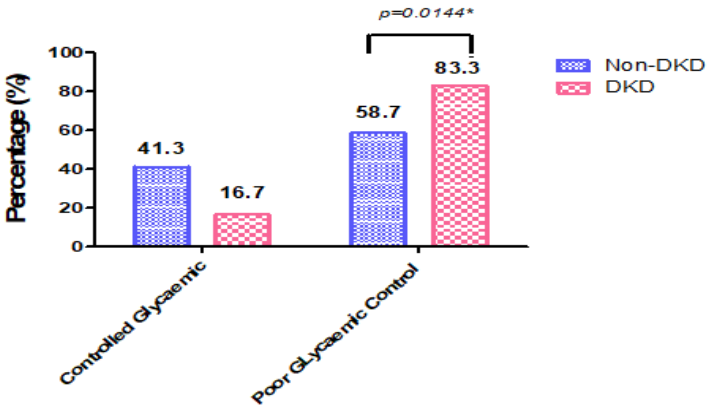

Fig. 3: Comparison of glycaemic control between DKD and non-DKD cohorts.

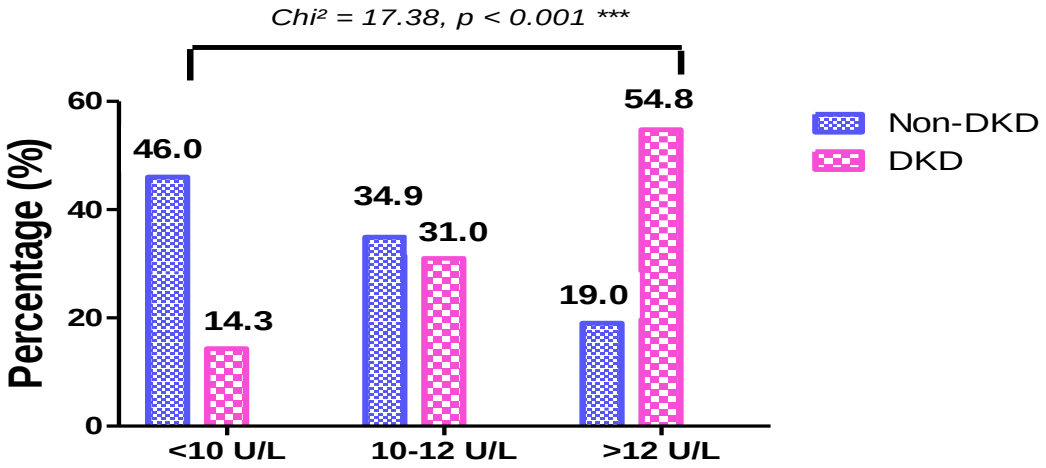

Fig. 4: Distribution of serum ADA levels among DKD and non-DKD patients.

Patients with DKD also demonstrated a higher prevalence of obesity, dyslipidaemia, abnormal liver function parameters, and greater dependence on insulin-based therapy than patients without DKD. Mean age, duration of diabetes, body mass index, systolic blood pressure, fasting and post-prandial blood glucose, HbA1c, serum creatinine, and serum ADA levels were significantly higher in the DKD group, whereas eGFR was significantly lower, reflecting more advanced metabolic and renal impairment (Fig. 5).

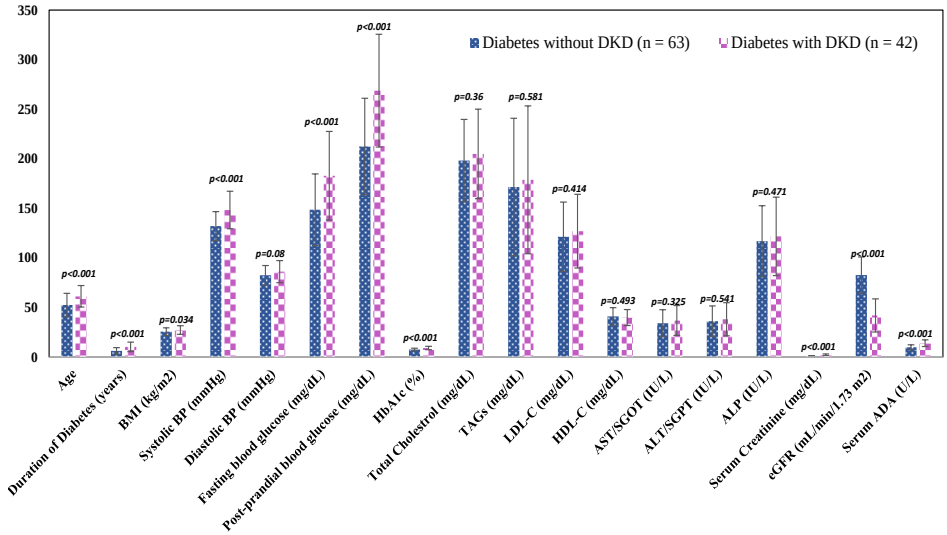

Fig. 5: Comparison of demographic, clinical, metabolic and renal characteristics between T2DM patients with and without DKD

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Multivariate logistic regression analysis demonstrated that increasing serum ADA levels were independently associated with diabetic kidney disease (Fig. 6). Patients with ADA levels >12 U/L had 9.26-fold higher odds of DKD in the unadjusted model. After adjustment for age, BMI, systolic blood pressure, HbA1c, renal function, and lipid parameters, elevated ADA remained a significant independent predictor of DKD (adjusted OR = 6.92, p = 0.002), with a significant positive trend across increasing ADA categories.

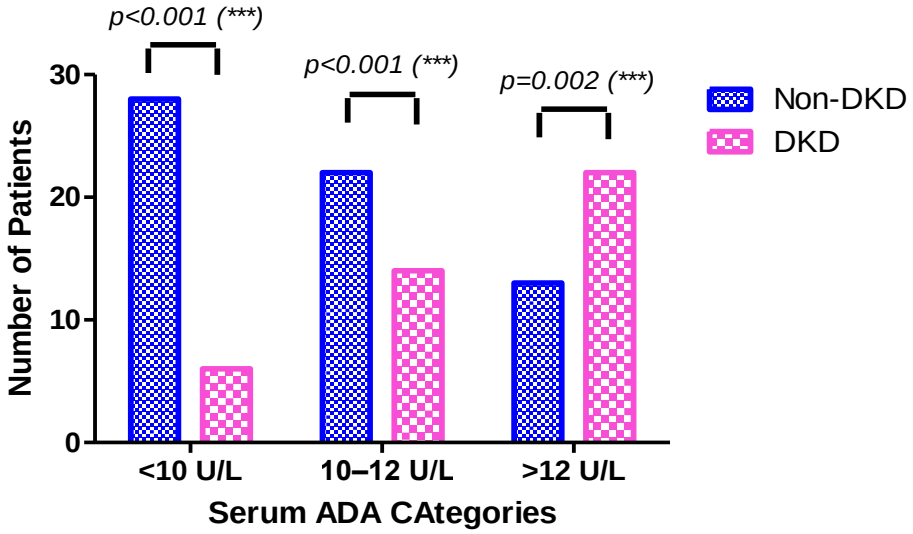

Fig. 6: Comparison of serum ADA categories between T2DM patients with and without DKD.

Further assessment revealed that most DKD patients belonged to advanced KDIGO GFR stages and had moderate-to-severe albuminuria. Elevated serum ADA levels were significantly associated with reduced eGFR and progressively increased with worsening KDIGO stage, indicating a close relationship between ADA activity and severity of renal dysfunction. ROC curve analysis demonstrated good diagnostic performance of serum ADA for predicting diabetic kidney disease, with an AUC of 0.83 (95% CI: 0.74–0.91), sensitivity of 78.6%, and specificity of 73.0% at an optimal cut-off value of 11.6 U/L (Fig. 7).

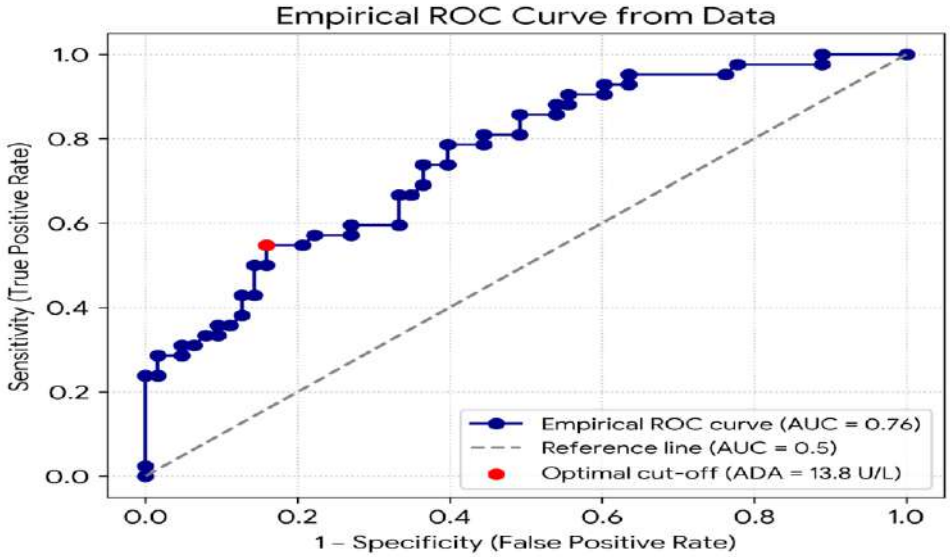

Fig. 7: Receiver operating characteristic (ROC) curve analysis of serum ADA levels for predicting diabetic kidney disease.

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DISCUSSION

The present study evaluated the association between serum ADA levels and DKD in patients with T2DM. The findings demonstrated that serum ADA levels were significantly elevated in patients with DKD compared with those without renal involvement. In addition, higher ADA levels were associated with poor glycemic control, increased albuminuria, reduced eGFR, and advanced KDIGO stages, suggesting a close relationship between ADA activity and the severity of renal impairment in diabetes [13].

Patients with DKD were older and had a longer duration of diabetes than those without DKD. These observations are consistent with previous reports identifying advancing age and prolonged exposure to hyperglycemia as major determinants of diabetic nephropathy progression [14]. Chronic hyperglycemia contributes to renal injury through glomerular hyperfiltration, oxidative stress, activation of inflammatory pathways, and structural damage to the renal microvasculature.

A greater proportion of DKD patients exhibited poor glycemic control, as reflected by higher HbA1c (≥7%) levels. This finding supports evidence from the UK Prospective Diabetes Study (UKPDS), which established that inadequate glycemic control significantly increases the risk of microvascular complications, including nephropathy [15]. Persistent hyperglycemia promotes the formation of advanced glycation end-products (AGEs), activation of protein kinase C, and generation of reactive oxygen species, all of which accelerate glomerular and tubular damage [16].

Patients with DKD were also more likely to have hypertension and dyslipidaemia. The increased systolic blood pressure in the present study is consistent with the findings of the ADVANCE study which identified hypertension as a major determinant of progression of renal disease in diabetes [17]. The increased prevalence of elevated triglycerides, LDL-cholesterol and low HDL-cholesterol in DKD patients also supports the role of lipid abnormalities in accelerating glomerular injury through endothelial dysfunction and inflammatory mechanisms.

The most important finding of this study was the significant elevation of serum ADA levels in DKD patients. Mean ADA levels were significantly higher in the DKD group (13.9 ± 3.4 U/L) than in the non-DKD group (9.8 ± 2.6 U/L). Similar results have been reported by [18,19], who observed increased ADA activity in diabetic patients with poor metabolic control and microvascular complications. ADA plays a central role in purine metabolism by converting adenosine into inosine. Since adenosine possesses anti-inflammatory, antioxidant, and renoprotective properties, increased ADA activity reduces adenosine availability and may enhance inflammatory responses within renal tissues. This mechanism may partly explain the strong association between elevated ADA levels and DKD observed in the present study.

The association between ADA and renal dysfunction was further supported by the significant relationship between ADA levels, albuminuria, and eGFR [20]. Patients with ADA levels >12 U/L exhibited a markedly higher prevalence of reduced eGFR and advanced KDIGO stages [21]. Furthermore, ADA levels progressively increased with worsening renal function, indicating a potential role of ADA in disease severity assessment. These findings agree with reports suggesting that chronic inflammation contributes significantly to DKD progression and that ADA may reflect the degree of immune activation occurring within the kidney [22,23].

Multivariate logistic regression analysis demonstrated that elevated ADA remained an independent predictor of DKD even after adjustment for age, BMI, blood pressure, HbA1c, lipid profile, and renal parameters. Patients with ADA >12 U/L had nearly seven-fold higher odds of DKD compared with those having ADA <10 U/L. These findings indicate that ADA provides additional prognostic information beyond traditional risk factors. Moreover, ROC analysis revealed good diagnostic performance of ADA with an AUC of 0.83, sensitivity of 78.6%, and specificity of 73.0% at a cut-off value of 11.6 U/L. This diagnostic accuracy is clinically relevant because ADA estimation is inexpensive, widely available, and easily performed in routine laboratory settings.

Overall, the present findings suggest that serum ADA is closely associated with metabolic dysregulation, inflammation, and renal impairment in T2DM. Elevated ADA levels may therefore serve as a valuable adjunct biomarker for identifying diabetic patients at increased risk of kidney disease and monitoring disease progression. However, the cross-sectional design limits causal inference, and larger prospective studies are required to establish the predictive value of ADA and explore its mechanistic role in DKD pathogenesis.

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CONCLUSIONS

The present study demonstrated that serum ADA levels were significantly higher in patients with DKD than in those without renal involvement. Elevated ADA levels were associated with lower eGFR, higher urinary albumin excretion, poor glycaemic control, and advanced stages of renal disease, indicating a significant relationship between serum ADA activities and the presence and severity of renal impairment in patients with T2DM. After adjustment for established clinical and biochemical risk factors, serum ADA remained an independent predictor of DKD, suggesting that it provides additional diagnostic information beyond conventional renal markers. Owing to its low cost, wide availability, and ease of estimation, serum ADA may serve as a useful adjunct biomarker for early identification of T2DM patients at increased risk of diabetic kidney disease and for monitoring disease progression. Further large-scale prospective studies are required to validate these findings.

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CONTRIBUTION OF AUTHORS

Research concept- Devendra Kumar, Vaibhav Shukla
Research design- Mohammad Hussain, Devendra Kumar, Vaibhav Shukla
Supervision- Devendra Kumar, Vaibhav Shukla
Materials- Mohammad Hussain, Firdaus Jabeen
Data collection- Mohammad Hussain, Daniyal Malik, Aman Aftab
Data analysis and Interpretation- Mohammad Hussain, Devendra Kumar, Aman Aftab
Literature search- Mohammad Hussain, Firdaus Jabeen
Writing article- Mohammad Hussain
Critical review- Devendra Kumar, Vaibhav Shukla, Jalees Fatima, Ajay Kumar Mishra
Article editing- Devendra Kumar, Vaibhav Shukla
Final approval- Devendra Kumar, Vaibhav Shukla, Jalees Fatima, Ajay Kumar Mishra

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