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Role of Serum Adenosine Deaminase in Assessing Severity and Inflammatory Status in Diabetic Foot Patients

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Abstract:

Background
Diabetic Foot is a major cause of morbidity in patients with Diabetes Mellitus, characterized by chronic inflammation and impaired wound healing. Adenosine Deaminase, an enzyme involved in cellular immunity, has been proposed as a potential biomarker of inflammatory and immunological activity. This study aimed to estimate serum ADA levels and evaluate their role in risk assessment and immunomodulation in diabetic foot patients.

Methods
This hospital-based observational study included 106 participants (53 cases with diabetic foot and 53 controls with diabetes without foot complications). Clinical, biochemical, and inflammatory parameters were assessed. Serum ADA levels were measured using a standard colorimetric method. Statistical analysis included group comparisons, ANOVA for severity grading, and ROC curve analysis to determine diagnostic performance.

Results
Patients with diabetic foot had significantly higher serum ADA levels compared to controls (34.6 ± 9.2 vs 23.8 ± 6.5 U/L; $p<0.001$). ADA levels increased progressively with Wagner grade ($p<0.001$) and were significantly higher in patients with infection and longer ulcer duration. ROC analysis demonstrated good diagnostic accuracy (AUC 0.84), with a cut-off $\geqslant$32.5 U/L yielding 78.3% sensitivity and 79.2% specificity.

Conclusion
Serum ADA is a promising biomarker reflecting disease severity and immune activation in diabetic foot, with potential utility in risk stratification and clinical management.

Keywords:

Diabetic foot; Adenosine deaminase; Biomarker; Inflammation; Wagner grade

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Introduction

Diabetes Mellitus is a rapidly escalating global health concern, with an estimated 537 million adults affected worldwide in 2021, projected to rise to 643 million by 2030 [1]. One of the most debilitating complications of diabetes is Diabetic Foot, which affects approximately 15–25% of diabetic patients during their lifetime and precedes nearly 85% of non-traumatic lower limb amputations [2,3]. The pathogenesis of diabetic foot is multifactorial, involving peripheral neuropathy, peripheral arterial disease, and impaired wound healing, often compounded by infection and immune dysfunction [4].

Chronic hyperglycemia in diabetes leads to a state of persistent low-grade inflammation and altered immune response. This dysregulated immunity contributes significantly to delayed wound healing and increased susceptibility to infections in diabetic foot ulcers [5]. Several inflammatory and immunological biomarkers, including C-reactive protein, interleukins, and procalcitonin, have been investigated for their role in predicting disease severity and outcomes; however, their routine clinical utility remains limited due to cost, variability, and lack of specificity [6].

Adenosine Deaminase is a key enzyme involved in purine metabolism, catalyzing the deamination of adenosine to inosine. It plays a crucial role in lymphocyte proliferation and differentiation, particularly in T-cell mediated immunity [7]. Elevated serum ADA levels have been associated with various inflammatory and infectious conditions such as tuberculosis, rheumatoid arthritis, and chronic liver diseases, reflecting heightened cellular immune activity [8].

In the context of diabetes, emerging evidence suggests that ADA activity may be altered due to chronic inflammation, oxidative stress, and immune dysregulation. Increased ADA levels have been reported in patients with poorly controlled diabetes and its complications, indicating its potential role as a marker of metabolic and immunological imbalance [9]. Furthermore, ADA-mediated modulation of adenosine levels may influence inflammatory pathways, endothelial dysfunction, and tissue repair mechanisms, all of which are critical in the pathogenesis of diabetic foot ulcers [10].

Despite these associations, the role of serum ADA as a biomarker for risk stratification and immunomodulation in diabetic foot remains inadequately explored. Identifying a reliable, cost-effective, and easily measurable biomarker like ADA could aid in early detection of high-risk patients, guide therapeutic interventions, and improve clinical outcomes.Therefore, the present study aimed to estimate serum adenosine deaminase levels in patients with diabetic foot and evaluate its utility as a marker for risk assessment and immunomodulatory status.

Material and methods

Study Design and Setting

This hospital-based observational analytical study was conducted in the Department of Biochemistry in collaboration with the Department of General Surgery at Vyas Medical College and Associated Hospital. The study was carried out over a period of 12 months from January 2023 to December 2023. The primary objective was to estimate serum Adenosine Deaminase levels in patients with Diabetic Foot and to evaluate its role as a marker for risk assessment and immunomodulation.

Study Population and Sample Size

The study included a total of 106 participants, comprising 53 patients diagnosed with diabetic foot (cases) and 53 age- and sex-matched individuals with Diabetes Mellitus without foot complications (controls). The sample size was calculated using the mean difference in serum Adenosine Deaminase levels reported in study by Wang et al., assuming a

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mean difference of approximately 8 U/L and a pooled standard deviation of 10 U/L [11]. Using a confidence level of 95% and a statistical power of 80%, the minimum required sample size was estimated to be 25 participants per group. After accounting for potential variability and to enhance statistical robustness, the final sample size was increased to 53 participants in each group.

Inclusion and Exclusion Criteria

Patients aged ⩾18 years with a confirmed diagnosis of diabetes mellitus and clinical evidence of diabetic foot, including ulceration, infection, or gangrene, were included in the study. Diagnosis of diabetic foot was based on clinical examination and relevant investigations. Patients with acute or chronic infections unrelated to diabetic foot (such as tuberculosis), autoimmune disorders, malignancies, chronic liver disease, renal failure, or those on immunosuppressive therapy were excluded, as these conditions could independently alter serum ADA levels. Pregnant women and patients unwilling to provide consent were also excluded.

Clinical Evaluation and Data Collection

All participants underwent a detailed clinical evaluation, including history taking with emphasis on duration of diabetes, glycemic control, comorbidities, and history of previous foot lesions or amputations. A thorough physical examination was performed, and diabetic foot lesions were assessed using standard classification systems such as Wagner's grading system for ulcer severity. Relevant anthropometric parameters and vital signs were recorded. Laboratory investigations including fasting blood glucose, postprandial blood glucose, and glycated hemoglobin (HbA1c) were performed to assess glycemic status.

Sample Collection and Biochemical Analysis

Venous blood samples (5 mL) were collected from all participants under aseptic conditions after an overnight fast. The samples were allowed to clot and centrifuged at 3000 rpm for 10 minutes to separate serum, which was then used for biochemical analysis. Serum ADA levels were estimated using a standard colorimetric method based on the enzymatic conversion of adenosine to inosine, with subsequent formation of ammonia measured spectrophotometrically. The assay was performed using commercially available kits as per the manufacturer's instructions, and results were expressed in units per liter (U/L). All samples were processed in the central laboratory under standardized conditions to ensure reliability and reproducibility.

Outcome Measures

The primary outcome measure was the level of serum ADA in patients with diabetic foot compared to controls. Secondary outcomes included correlation of serum ADA levels with severity of diabetic foot lesions, duration of diabetes, glycemic control (HbA1c), and presence of infection or complications, to assess its role in risk stratification and immunomodulation.

Statistical Analysis

Data were entered into Microsoft Excel and analyzed using SPSS version 23.0. Continuous variables were expressed as mean ± standard deviation (SD), while categorical variables were presented as frequencies and percentages. Normality of data distribution was assessed using appropriate tests (Shapiro–Wilk test). For comparison between two groups, the independent Student's t-test was applied for normally distributed variables, while the Mann–Whitney U test was used for non-normally distributed data. Categorical variables were compared using the Chi-square test or Fisher's exact test as appropriate. For comparison of serum Adenosine Deaminase levels across multiple severity categories (Wagner grades), one-way analysis of variance (ANOVA) was used for normally distributed data, and the Kruskal–Wallis test was applied when normality assumptions were not met. Receiver operating characteristic (ROC)

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curve analysis was performed to evaluate the diagnostic performance of serum ADA in predicting severe diabetic foot (Wagner grade ⩾3), and the area under the curve (AUC), optimal cut-off value, sensitivity, specificity, positive predictive value (PPV), negative predictive value (NPV), and diagnostic accuracy were calculated. A p-value of <0.05 was considered statistically significant.

Ethical Considerations

The study was conducted in accordance with the ethical principles outlined in the Declaration of Helsinki. Ethical clearance was obtained from the Institutional Ethics Committee prior to the commencement of the study. Written informed consent was obtained from all participants after explaining the purpose and procedures of the study in their local language. Confidentiality of patient data was strictly maintained throughout the study.

Results

The mean age of patients with Diabetic Foot was comparable to controls (58.6 ± 9.8 vs 56.9 ± 8.7 years; p=0.341), with a male predominance in both groups. No significant differences were observed in residence, socioeconomic status, BMI, or lifestyle factors such as smoking and alcohol use (p>0.05). However, the duration of Diabetes Mellitus was significantly longer in cases compared to controls (11.8 ± 5.6 vs 7.9 ± 4.3 years; p<0.001). Notably, peripheral neuropathy (71.7% vs 32.1%; p<0.001), peripheral arterial disease (39.6% vs 17.0%; p=0.011), and previous history of ulcer or amputation (34.0% vs 11.3%; p=0.006) were significantly more prevalent among cases, indicating a higher burden of microvascular and macrovascular complications (Table 1).

Table 1. Baseline Demographic and Clinical Characteristics of Study Participants.

Variable	Cases (n=53)	Controls (n=53)	p-value
	Frequency (%)/mean ± SD		
Age (years)	58.6 ± 9.8	56.9 ± 8.7	0.341
Gender			
Female	17 (32.1%)	19 (35.8%)	0.608
Male	36 (67.9%)	34 (64.2%)	
Residence			
Urban	24 (45.3%)	29 (54.7%)	0.332
Rural	29 (54.7%)	24 (45.3%)	
Socioeconomic status			
Upper	6 (11.3%)	8 (15.1%)	0.343
Middle	16 (30.2%)	19 (35.8%)	
Lower	31 (58.5%)	26 (49.1%)	
Duration of diabetes (years)	11.8 ± 5.6	7.9 ± 4.3	<0.001
BMI (kg/m²)	26.4 ± 3.2	25.9 ± 3.0	0.451
Hypertension	31 (58.5%)	27 (50.9%)	0.483
Dyslipidemia	28 (52.8%)	22 (41.5%)	0.284
Smoking	19 (35.8%)	14 (26.4%)	0.299
Alcohol use	15 (28.3%)	11 (20.8%)	0.316
Peripheral neuropathy	38 (71.7%)	17 (32.1%)	<0.001
Peripheral arterial disease	21 (39.6%)	9 (17.0%)	0.011

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Previous ulcer/amputation	**18 (34.0%)**	**6 (11.3%)**	**0.006**

Patients with diabetic foot demonstrated significantly poorer glycemic control, with higher fasting blood glucose (168.4 ± 42.6 vs 142.7 ± 36.5 mg/dL; p=0.001), postprandial glucose (242.3 ± 58.1 vs 198.6 ± 49.2 mg/dL; p<0.001), and HbA1c levels (9.1 ± 1.6 vs 7.8 ± 1.3%; p<0.001). Markers of inflammation and infection, including total leukocyte count, ESR, and CRP, were significantly elevated in cases (all p<0.001). Additionally, cases had significantly lower hemoglobin and serum albumin levels, along with higher serum creatinine (p ⩽ 0.002), suggesting anemia, malnutrition, and renal involvement. Although total cholesterol and triglyceride levels were higher in cases, these differences did not reach statistical significance (p>0.05). Importantly, serum ADA levels were markedly elevated in cases compared to controls (34.6 ± 9.2 vs 23.8 ± 6.5 U/L; p<0.001) (Table 2).

Table 2. Comparison of Glycemic, Hematological, and Biochemical Parameters.

Parameter	Cases (n=53)	Controls (n=53)	p-value
	mean ± SD		
Fasting blood glucose (mg/dL)	**168.4 ± 42.6**	**142.7 ± 36.5**	**0.001**
Postprandial glucose (mg/dL)	**242.3 ± 58.1**	**198.6 ± 49.2**	**<0.001**
HbA1c (%)	**9.1 ± 1.6**	**7.8 ± 1.3**	**<0.001**
Total leukocyte count (/mm³)	**10,980 ± 2,850**	**8,740 ± 2,120**	**<0.001**
Hemoglobin (g/dL)	**11.2 ± 1.8**	**12.6 ± 1.5**	**<0.001**
ESR (mm/hr)	**48.3 ± 18.5**	**26.7 ± 12.4**	**<0.001**
CRP (mg/L)	**14.8 ± 6.2**	**6.1 ± 3.5**	**<0.001**
Serum creatinine (mg/dL)	**1.3 ± 0.5**	**1.0 ± 0.3**	**0.002**
Serum albumin (g/dL)	**3.2 ± 0.6**	**3.8 ± 0.5**	**<0.001**
Total cholesterol (mg/dL)	**196.5 ± 38.2**	**182.4 ± 34.7**	**0.086**
Triglycerides (mg/dL)	**178.2 ± 52.6**	**154.3 ± 47.8**	**0.082**
Serum ADA (U/L)	**34.6 ± 9.2**	**23.8 ± 6.5**	**<0.001**

Serum ADA levels demonstrated a significant progressive increase with advancing severity of diabetic foot as per Wagner grading (p<0.001), rising from 28.1 ± 6.3 U/L in Grade 1 to 40.7 ± 9.1 U/L in Grades 4–5. Patients with infection had significantly higher ADA levels compared to those without infection (36.9 ± 8.8 vs 29.7 ± 7.2 U/L; p=0.002). Similarly, longer ulcer duration (≥1 month) was associated with significantly elevated ADA levels (36.8 ± 9.1 vs 30.2 ± 7.5 U/L; p=0.011). These findings indicate a strong association between ADA levels and disease severity, infection status, and chronicity (Table 3).

Table 3. Association of Serum ADA Levels with Clinical Severity Parameters in Diabetic Foot.

Variable	Category	Serum ADA (U/L)	p-value
		mean ± SD	
Wagner Grade	**Grade 1 (n=12)**	**28.1 ± 6.3**	**<0.001**
	Grade 2 (n=15)	**31.9 ± 7.4**	
	Grade 3 (n=13)	**35.8 ± 8.2**	
	Grade 4–5 (n=13)	**40.7 ± 9.1**	

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Infection status	**Present (n=37)**	**36.9 ± 8.8**	**0.002**
	Absent (n=16)	**29.7 ± 7.2**	
Ulcer duration	**<1 month (n=18)**	**30.2 ± 7.5**	**0.011**
	≥1 month (n=35)	**36.8 ± 9.1**	

Receiver operating characteristic (ROC) curve analysis demonstrated that serum ADA has good diagnostic accuracy in predicting severe diabetic foot. The AUC was 0.84 (95% CI: 0.75–0.92), indicating strong discriminative ability. An optimal cut-off value of ≥32.5 U/L yielded a sensitivity of 78.3% and specificity of 79.2%. The corresponding positive and negative predictive values were 75.0% and 81.8%, respectively, with an overall diagnostic accuracy of 78.8%. These findings support the potential utility of serum ADA as a reliable biomarker for severity assessment in diabetic foot (Table 4 and Figure 1).

Table 4. Diagnostic Performance of Serum ADA for Predicting Severe Diabetic Foot (ROC Analysis).

Parameter	**Value (95% CI)**
AUC (Area Under Curve)	**0.84 (0.75–0.92)**
Optimal Cut-off (U/L)	**≥ 32.5**
Sensitivity (%)	**78.30%**
Specificity (%)	**79.20%**
Positive Predictive Value (PPV)	**75.00%**
Negative Predictive Value (NPV)	**81.80%**
Diagnostic Accuracy (%)	**78.80%**

HOMA-IR showed a significant positive correlation with age (r=0.21, p=0.007), BMI (r=0.34, p<0.001), and fasting plasma glucose (r=0.42, p<0.001), indicating that insulin resistance increases with advancing age, higher adiposity, and glycemic levels. Additionally, moderate positive correlations were observed with liver enzymes ALT (r=0.29, p<0.001) and AST (r=0.25, p=0.002), suggesting a relationship between hepatic inflammation and insulin resistance (Table 5).

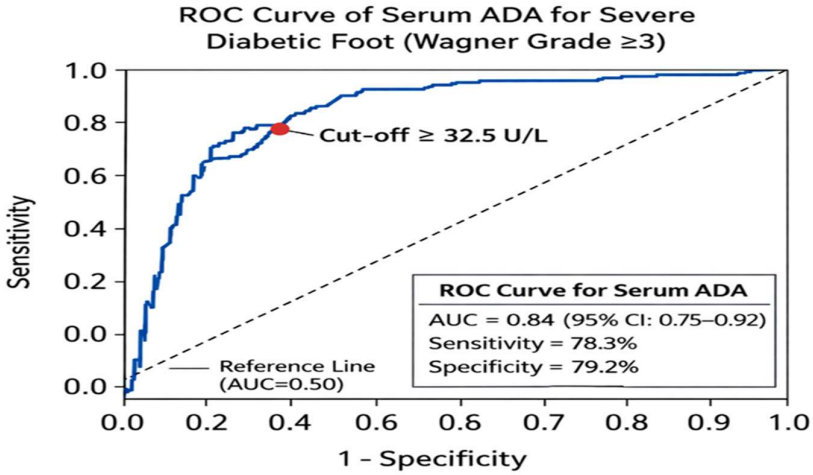

Figure 1. Receiver Operating Characteristic (ROC) Curve of Serum Adenosine Deaminase for Predicting Severe Diabetic Foot (Wagner Grade ≥3).

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Discussion

The present study demonstrates that serum Adenosine Deaminase levels are significantly elevated in patients with Diabetic Foot and are strongly associated with disease severity, inflammatory burden, and chronicity, thereby supporting its potential role as a biomarker for risk assessment and immunomodulation. The baseline characteristics revealed that cases and controls were comparable in terms of age, gender distribution, BMI, and lifestyle factors ($p>0.05$), indicating adequate matching and minimizing confounding. However, the significantly longer duration of Diabetes Mellitus among cases ($p<0.001$) underscores the cumulative effect of chronic hyperglycemia in the development of foot complications, a finding consistent with previous epidemiological study and metanalysis by Andarge et al., and Guo et al., [12,13]. Additionally, the markedly higher prevalence of peripheral neuropathy, peripheral arterial disease, and prior ulceration among cases aligns with established pathogenic pathways involving neurovascular compromise and impaired tissue perfusion [13].

The biochemical profile further highlights the interplay between poor glycemic control and inflammatory activation in diabetic foot. Significantly elevated fasting and postprandial glucose levels and HbA1c in cases (all $p\leqslant0.001$) corroborate findings from studies by Akyüz et al., and Mohamed et al., and Farooque et al., which demonstrated that uncontrolled glycemia predisposes to infection and delayed wound healing [14,15,16]. The observed elevation in inflammatory markers such as total leukocyte count, ESR, and CRP, along with reduced hemoglobin and serum albumin levels, reflects a state of systemic inflammation, infection, and nutritional compromise. Hypoalbuminemia, in particular, may impair wound healing through reduced oncotic pressure and diminished protein reserves, as previously reported by Edakkepuram et al., [17].

Importantly, serum ADA levels were significantly higher in cases compared to controls (34.6 ± 9.2 vs 23.8 ± 6.5 U/L; $p<0.001$), which is in agreement with recent studies by Niraula et al., Ray et al., and Yu et al., evaluating ADA in diabetic complications, suggesting that ADA activity increases in response to heightened cellular immunity and oxidative stress [18,19,20].

A key finding of this study is the progressive increase in serum ADA levels with advancing Wagner grade ($p<0.001$), indicating a strong association with disease severity. This gradient—from 28.1 U/L in Grade 1 to 40.7 U/L in Grades 4–5—suggests that ADA may reflect the extent of tissue damage and inflammatory activation. Similar trends have been reported in studies assessing ADA in diabetes by Antonioli et al., Sapkota et al., and Khan et al., where elevated ADA corresponds to increased T-lymphocyte activation and macrophage response [21,22,23]. The significantly higher ADA levels in patients with infection ($p=0.002$) and longer ulcer duration ($p=0.011$) further reinforce its role as a marker of ongoing immune activation and chronic inflammation. Mechanistically, ADA regulates extracellular adenosine levels, which modulate inflammatory pathways, endothelial function, and tissue repair. Increased ADA activity reduces adenosine availability, thereby promoting pro-inflammatory cytokine release and impairing wound healing, which may explain its association with more severe and chronic lesions [24,25].

The ROC curve analysis adds significant clinical value by demonstrating that serum ADA has good diagnostic performance in predicting severe diabetic foot, with an AUC of 0.84. A cut-off value of $\geqslant32.5$ U/L yielded balanced sensitivity (78.3%) and specificity (79.2%), indicating its potential utility as a practical and cost-effective biomarker in clinical settings. Comparable diagnostic performance has been reported in studies by Raut et al., and Kundu et al., evaluating ADA in inflammatory and diabetic foot, where AUC values ranging from 0.75 to 0.88 have been observed [26,27]. The relatively high

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negative predictive value (81.8%) suggests that lower ADA levels may help exclude severe disease, which could aid in early risk stratification and management decisions.

Limitations

This study has certain limitations. Being a single-center study with a relatively modest sample size, the findings may have limited generalizability. The cross-sectional design precludes causal inference between serum Adenosine Deaminase levels and disease progression. Potential confounders such as concomitant infections or subclinical inflammatory conditions could not be entirely excluded. Additionally, longitudinal assessment of ADA trends and outcomes was not performed.

Conclusion

The present study demonstrates that serum Adenosine Deaminase levels are significantly elevated in patients with Diabetic Foot and show a strong positive association with disease severity, infection status, and duration of ulcer. The progressive rise in ADA levels across Wagner grades and its good diagnostic performance (AUC 0.84) highlight its potential as a reliable biomarker for risk stratification. Given its affordability, accessibility, and correlation with inflammatory and immunological activity, serum ADA may serve as a useful adjunct in clinical assessment. Further multicentric and longitudinal studies are warranted to validate its prognostic utility and role in guiding therapeutic interventions.

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