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Evaluation of Serum Asprosin Level in Development of Diabetic Nephropathy in Elderly Type 2 Diabetic Patients

Asmaa M. Gouda[1], Azza H. Mohamed [2], Marwa A. Saad[2], Sawsan S. Saad[2], Ahmed M. Mohsen[2]
[1] Department of Clinical Pathology, [2] Department of Internal Medicine (Geriatric unit),
Faculty of Medicine, Alexandria University, Alexandria, Egypt
Corresponding author: Asmaa Mustafa Gouda Elewa, **Email:** asmaa.gouda@alexmed.edu.eg,
Mobile: +201009979127, **Orchid ID**: 0009-0001-2880-126X

ABSTRACT

Background: Diabetic nephropathy (DN), a serious kidney consequence brought on by persistent hyperglycemia, hypertension, and metabolic stress, is largely caused by type 2 diabetes mellitus (T2DM). Diabetes causes a rise in asprosin, an adipose-derived hormone that raises blood sugar and has been linked to insulin resistance and inflammation. Although DN has been linked to higher asprosin, its clinical implications are still unknown.

Aim of the work: The objectives of this study are to assess blood asprosin levels in older patients with type 2 diabetes and to investigate its potential as a predictive marker for diabetic nephropathy.

Patients and Methods: T2DM patients with diabetic nephropathy (Group 2), non-diabetic controls (Group 1), and T2DM patients without diabetic nephropathy (Group 3) were the three equal groups of 105 people aged ≥65 years who participated in this cross-sectional study. The Alexandria Main University Hospital's Internal Medicine and Geriatrics clinics served as the source of participants. Levels of serum asprosin were assessed and contrasted between groups.

Results: Group 2 showed significantly higher serum asprosin levels compared to Groups 1 and 3 (90.09 ± 1.15 vs. 28.92 ± 10.12 vs. 18.58 ± 5.96 ng/mL; p<.001). A cutoff value of >41.85 ng/mL effectively differentiated DN cases, with 94.29% sensitivity, 91.43% specificity, and an AUC of 0.9.

Conclusion: Elevated serum asprosin is strongly associated with diabetic nephropathy in elderly T2DM patients. According to these results, asprosin may serve as a helpful biomarker for early detection and a possible target for treatment in the management of diabetic kidney problems.

Keywords: Diabetes, Diabetic nephropathy, Biomarker, Asprosin.

INTRODUCTION

Chronic hyperglycemia brought on by compromised insulin secretion and/or action is a hallmark of type 2 diabetes mellitus (T2DM), which can result in macrovascular and microvascular consequences, such as diabetic nephropathy (DN), a major cause of end-stage renal disease and mortality in T2DM [(1-2)]. Persistent hyperglycemia, hypertension, and osmotic diuresis contribute to glomerular injury and progression of DN [(2)].

Asprosin, a fasting-induced hormone secreted by white adipose tissue, promotes hepatic glucose release via the OLFR734–cAMP–PKA pathway [(3)]. Elevated asprosin levels in obesity, insulin resistance, and diabetes are associated with hyperglycemia, increased appetite, inflammation, and insulin resistance, which may contribute to DN. However, studies on its role in diabetic complications are limited [(4)].

So, the purpose of this study is to measure blood asprosin levels in older T2DM patients and see whether they are useful as a DN prognostic factor.

PATIENTS AND METHODS

Study Design and Setting

This cross-sectional study had 105 participants aged 65 years and above. They were divided into three equal groups: Group 1: non-diabetic persons, Group 2: elderly persons with T2DM and DN, and Group 3: elderly T2DM patients without DN. Patients were recruited from the Internal Medicine Sections and Geriatrics Clinic of Alexandria Main University Hospital.

Eligibility Criteria

Inclusion criteria include T2DM patients aged ≥65 years of both sexes diagnosed either with diabetic nephropathy confirmed by laboratory investigations and Ultrasound renal assessment, or T2DM patients without diabetic nephropathy.

***Exclusion criteria*:** Age <65 years, acute diabetic complications, severe liver diseases, malignancy, obesity (BMI>30), and coronary disease.

The following data were collected from every patient after enrollment into the study:

1. **Demographic Data**: age and sex.
2. **History taking**: full medical history, duration of Diabetes Mellitus, Diabetic kidney disease, drug history, complications of DM, and other comorbidities.
3. **A thorough clinical assessment**
4. **Radiological investigations:** Ultrasound of abdomen and pelvis.
5. **Standard laboratory studies** including complete blood count (CBC), renal function tests such blood urea and serum creatinine, and measurement of serum electrolytes [sodium (Na), calcium (Ca), potassium (K), and phosphorus (P)]. Additional tests included urine albumin-to-creatinine ratio (UACR), lipid profile (total cholesterol (TC), triglycerides (TG), high-density lipoprotein cholesterol (HDL-C), and low-density lipoprotein cholesterol (LDL-C), serum uric acid, and diabetes workup (fasting blood glucose, 2-hour postprandial glucose, and glycated hemoglobin HbA1c).

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6. **Specific investigations:** using the enzyme-linked immunosorbent assay (ELISA) technique to measure the fasting serum asprosin level according to the manufacturer's instructions.

Ethical consideration: **Informed consent was obtained, and the study was approved by the Alexandria Faculty of Medicine Ethics Committee. The Declaration of Helsinki was adhered to during the research.**

Statistical Analysis of Data: SPSS v27 was used. The median (IQR) or mean±SD were used to express continuous variables, and categorical variables as frequencies. ANOVA, Chi-square, Mann-Whitney, and ROC analyses were applied. $p \leq 0.05$ was considered significant.

RESULTS

The demographic information for the groups under study is displayed in Table 1 with no gender disparities (p=.710), marital status (p=.379), or smoking (p=.50).

Table (1): Comparison between the studied groups regarding their demographic data

	Group (1) (n = 35)		Group (2) (n = 35)		Group (3) (n = 35)		Test of Sig.	P-value
	No.	%	No.	%	No.	%		
Gender								
Male	16	45.7	19	54.3	16	45.7	χ^2= 0.686	0.710
Female	19	54.3	16	45.7	19	54.3		
Age (years)								
Min. – Max.	65.0 – 80.0		65.0 – 80.0		65.0 – 75.0		F= 4.834*	0.010*
Mean ± SD.	69.09 ± 4.20		70.69 ± 5.26		67.60 ± 2.53			
Median (IQR)	68.0 (65.0 – 73.0)		68.0 (66.0 – 75.0)		67.0 (66.0 – 69.50)			
Sig. bet. Grps	p_1=0.245,p_2=0.297,p_3=0.007*							
Marital status								
Married	26	74.3	24	68.6	29	82.9	χ^2= 1.943	0.379
Widow	9	25.7	11	31.4	6	17.1		
Special habits								
Non-smoker	24	68.6	18	51.4	21	60.0	χ^2= 3.294	MCp= 0.500
Smoker	11	31.4	15	42.9	13	37.1		
Ex-smoker	0	0.0	2	5.7	1	2.9		

IQR: Inter quartile range; **SD:** Standard deviation; **F:** F for One way ANOVA test, Pairwise comparison bet. each 2 groups was done using **Post Hoc Test** (Tukey); χ^2: Chi square test ; **MC**: Monte Carlo test; **p:** p value for comparing between the three studied groups; p_1: p-value between group (1) and group (2); p_2**:** p-value between **group (1)** and **group (3);** p_3**:** p-value between **group (2)** and **group (3)**; *: Statistically significant at $p \leq 0.05$; **Sig. bet. grps.:** significance between groups.

Group (1): Non diabetic participants.
Group (2): type 2 diabetic patients complicated with DN.
Group (3): type 2 diabetic patients without DN.

Comorbidities are shown in Table 2, with group 3 having higher hypertension (60%) than group 2 (45.7%) and 1 (25.7%) (p=.015); there are no differences in COPD (p=.618), asthma (p=.365), or heart failure (p=.727).

Table (2): Comparison between the studied groups according to comorbidities

	Group (1) (n = 35)		Group (2) (n = 35)		Group (3) (n = 35)		χ^2	P-value
	No.	%	No.	%	No.	%		
HTN	9	25.7	16	45.7	21	60.0	8.434*	0.015*
Sig. bet. grps.	p_1=0.081,p_2=0.004*,p_3=0.231							
Hypothyroidism	4	11.4	0	0.0	0	0.0	5.953	MCp=0.031*
Sig. bet. grps.	p_1=0.039*,p_2=0.039*,p_3–							
COPD	3	8.6	1	2.9	1	2.9	1.476	MCp=0.618
Asthma	3	8.6	2	5.7	0	0.0	2.920	MCp=0.365
Anemia	24	68.6	23	65.7	15	42.9	5.750	0.056
Heart Failure	2	5.7	4	11.4	2	5.7	1.041	0.727

χ^2**:** Chi square test; **MC:** Monte Carlo test; **p:** p-value for comparing between the three studied groups; p_1**:** p-value between group (1) and group (2); p_2: p-value between group (1) and group (3); p_3: p-value between group (2) and group (3); *: Statistically significant at $p \leq 0.05$ **Sig. bet. grps.:** significance between groups; **DM**: diabetes mellitus; **HTN**: hypertension; **COPD:** chronic obstructive pulmonary disease.

Group (1): Non diabetic participants.
Group (2): type 2 diabetic patients complicated with DN.
Group (3): type 2 diabetic patients without DN.

Table 3 shows that Group 2 had longer diabetes duration and more insulin use; group 3 used more oral agents; neuropathy was similar.

Table (3): Comparison between the two studied groups according to medical history

	Group (2) (n = 35)		**Group (3) (n = 35)**		**Test of Sig.**	**P-value**
	No.	**%**	**No.**	**%**		
Antidiabetic medications						
OADs	10	28.6	19	54.3	χ^2=4.769*	0.029*
Insulin	25	71.4	17	48.6	χ^2=3.810	0.051
Duration of diabetes (years)						
Min – Max.	10.0 – 30.0		2.0 – 10.0		U= 7.500*	<0.001*
Mean ± SD.	16.37 ± 4.85		5.26 ± 2.21			
Median (IQR)	15.0 (12.50 – 20.0)		5.0 (3.50 – 7.0)			
Complications of diabetes	35	100.0	12	34.3	χ^2=34.255*	<0.001*
Diabetic Retinopathy Cerebrovascular diseases (strokes)	35 2	100.0 5.7	0 1	0.0 2.9	χ^2=70.000* χ^2=0.348	<0.001* 0.555
Diabetic Neuropathy	8	22.9	12	34.3	χ^2=1.120	0.290
Diabetic foot	2	5.7	1	2.9	χ^2=0.348	FEp=1.000

IQR: Inter quartile range; **SD**: Standard deviation; **U**: Mann Whitney test; **χ^2:** Chi square test; **FE**: Fisher Exact; **p:** p-value between the three studied groups; *****: Statistically significant at $p \leq 0.05$;
OADs: oral antidiabetic drugs;
Group (2): type 2 diabetic patients complicated with DN
Group (3): type 2 diabetic patients without DN

As illustrated in Table 4, group 2 had higher height, weight, and BMI vs. group 3, and higher height/weight vs. group 1; groups 1 and 3 were similar.

Table (4): Comparison between the studied groups according to anthropometric measurement

	Group (1) (n = 35)	**Group (2) (n = 35)**	**Group (3) (n = 35)**	**F**	**P-value**
Height (m)					
Mean ± SD.	1.67 ± 0.06	1.71 ± 0.05	1.67 ± 0.04	4.594*	0.012*
Sig. bet. grps.	p_1=0.027*,p_2=1.000,p_3=0.027*				
Weight (kg)					
Mean ± SD.	79.51 ± 6.36	83.66 ± 4.39	78.11 ± 4.44	10.984*	<0.001*
Sig. bet. grps.	p_1=0.003*,p_2=0.493,p_3<0.001*				
BMI (kg/m^2)					
Mean ± SD.	28.35 ± 1.0	28.73 ± 0.96	27.90 ± 1.46	4.471*	0.014*
Sig. bet. grps.	p_1=0.362, p_2=0.242, p_3=0.010*				

IQR: Inter quartile range; **SD:** Standard deviation; **F:** F for One way ANOVA test, Pairwise comparison bet. each 2 groups was done using **Post Hoc Test** (Tukey); **p:** p-value between the three studied groups; **p_1**: p-value between group (1) and group (2); **p_2:** p-value for between **group (1)** and **group (3); p_3:** p-value between **group(2)** and **group (3)**; *****: Statistically significant at $p \leq 0.05$; **M:** meter; **Kg**: Kilogram; **BMI**: body mass index; **Sig. bet. grps.:** significance between groups.

Group (1): Non diabetic participants
Group (2): type 2 diabetic patients complicated with DN
Group (3): type 2 diabetic patients without DN

No meaningful differences were demonstrated in WBC (*p*=.053) or platelets (*p*=.741), but hemoglobin was lower in group 2 in comparison with group 3 (*p*=.036) as illustrated in Table 5.

Table (5): Comparison between the studied groups according to CBC

	Group (1) (n = 35)	**Group (2) (n = 35)**	**Group (3) (n = 35)**	**F**	**P-value**
Hb(g/dl)					
Mean ± SD.	9.78 ± 1.11	9.73 ± 1.44	10.58 ± 1.65	3.959*	0.022*
Sig. bet. grps.	p_1=0.986, p_2=0.053, p_3=0.036*				
PLT (10^{11}/unit)					
Mean ± SD.	256.6 ± 52.13	256.2 ± 58.03	242.6 ± 52.14	0.300	0.741
WBCs (10^9/L)					
Mean ± SD.	7.58 ± 1.72	8.97 ± 2.14	7.44 ± 1.72	3.034	0.053

IQR: Inter quartile range; **SD:** Standard deviation; **F:** F for One way ANOVA test, Pairwise comparison bet. each 2 groups was done using **Post Hoc Test** (Tukey); **p:** p-value between the three studied groups; **p_1**: p-value between group (1) and group (2); **p_2:** p-value between **group (1)** and **group (3); p_3:** p-value between **group(2)** and **group (3)**; *: Statistically significant at $p \leq 0.05$; CBC**: complete blood count; Hb:** hemoglobin; PLT: platelet; WBCs: white blood cell; Sig. **bet. grps**.: significance between groups.

Group (1): Non diabetic participants
Group (2): type 2 diabetic patients complicated with DN
Group (3): type 2 diabetic patients without DN

Table 6 showed higher FBG, 2HPP, and HbA1C in group 2 in comparison with groups 1 and 3 (p<.001), with FBG also higher in group 3 in comparison with group 1 (p<.001).

Table (6): The glycemic profile of the three studied groups

	Group (1) (n = 35)	**Group (2) (n = 35)**	**Group (3) (n = 35)**	**F**	**p-value**
FBG ((mg/dl)					
Mean ± SD.	87.74 ± 7.06	154.5 ± 30.90	112.6 ± 20.10	84.747*	<0.001*
Sig. bet. grps.	p_1<0.001*,p_2<0.001*,p_3<0.001*				
2HPP (mg/dl)					
Mean ± SD.	108.3 ± 11.61	213.9 ± 24.39	213.9 ± 44.39	107.540*	<0.001*
Sig. bet. grps.	p_1<0.001*,p_2<0.001*,p_3<0.001*				
HbA1C					
Mean ± SD.	5.16 ± 0.31	8.50 ± 0.76	7.25 ± 0.46	337.324*	<0.001*
Sig. bet. grps.	p_1<0.001*,p_2<0.001*,p_3<0.001*				

IQR: Inter quartile range; **SD:** Standard deviation; **F:** F for One way ANOVA test, Pairwise comparison bet. each 2 groups were done using **Post Hoc Test** (Tukey); **p:** p-value between the three studied groups; **p_1**: p-value between group (1) and group (2); **p_2:** p-value between **group (1)** and **group (3); p_3:** p-value between **group(2)** and **group (3)**; *: Statistically significant at $p \leq 0.05$; **FBG**: Fasting blood glucose; **2HPP**: two-hours post prandial; **HbA1c:** glycated hemoglobin.

Group (1): Non diabetic participants
Group (2): type 2 diabetic patients complicated with DN
Group (3): type 2 diabetic patients without DN

As shown in Table 7, group 2 had greater levels of BUN, urea, creatinine, and uric acid than groups 1 and 3 (p<.001).

Table (7): Comparison between the studied groups according to Renal functions

	Group (1) (n = 35)	**Group (2) (n = 35)**	**Group (3) (n = 35)**	**F**	**P-value**
BUN (mg/dl).					
Mean ± SD.	15.51 ± 4.99	52.27 ± 10.99	15.64 ± 5.82	262.430*	<0.001*
Sig. bet. grps.	p_1<0.001*,p_2=0.997,p_3<0.001*				
Blood Urea (mg/dl).					
Mean ± SD.	33.23 ± 1.69	112.0 ± 23.55	33.51 ± 2.48	262.430*	<0.001*
Sig. bet. grps.	p_1<0.001*,p_2=0.997,p_3<0.001*				
Serum Creatinine (mg/dl).					
Mean ± SD.	0.63 ± 0.12	2.19 ± 0.58	0.67 ± 0.10	232.764*	<0.001*
Sig. bet. grps.	p_1<0.001*,p_2=0.888,p_3<0.001*				
Serum Uric acid (mg/dl).					
Mean ± SD.	4.92 ± 0.93	7.11 ± 1.39	5.04 ± 0.95	42.991*	<0.001*
Sig. bet. grps.	p_1<0.001*,p_2=0.884,p_3<0.001*				

IQR: Inter quartile range; **SD:** Standard deviation; **F:** F for One way ANOVA test, Pairwise comparison bet. each 2 groups was done using **Post Hoc Test** (Tukey); **p:** p-value between three studied groups; **p_1**: p-value between group (1) and group (2); **p_2:** p-value between **group (1)** and **group (3); p_3:** p- value between **group(2)** and **group (3)**; *: Statistically significant at $p \leq 0.05$; **BUN:** blood urea nitrogen.

Group (1): Non diabetic participants
Group (2): type 2 diabetic patients complicated with DN
Group (3): type 2 diabetic patients without DN

Compared to groups 1 and 3, group 2 had worse renal function, as indicated by a higher ACR and a lower eGFR (Table 8).

Table (8): Comparison between the studied groups according to Urinary ACR and eGFR using MDRD

	Group (1) (n = 35)	**Group (2) (n = 35)**	**Group (3) (n = 35)**	**H**	**P-value**
Urinary ACR (mg/g)					
Mean ± SD.	19.34 ± 4.05	846.2 ± 53.6	25.52 ± 3.35	83.023*	<0.001*
Sig. bet. grps.	p_1<0.001*,p_2<0.001*,p_3<0.001*				
eGFR using MDRD (ml/min/1.73 $m^{2)}$					
Mean ± SD.	118.1 ± 22.92	29.63 ± 5.34	110.1 ± 22.84	69.678*	<0.001*
Sig. bet. grps.	p_1<0.001*,p_2=0.596,p_3<0.001*				

IQR: Inter quartile range, **SD:** Standard deviation; **H:** H for Kruskal Wallis test, Pairwise comparison bet. each 2 groups was done using **Post Hoc Test** (Dunn's for multiple comparisons test); **p**: p-value for comparing between the three studied groups; **p_1:** p-value between group(1) and group (2); **p_2**: p- value between group (1) and group (3); **p_3:** p-value between group (2) and group (3); ***:** Statistically significant at $p \leq 0.05$; **ACR**: Albumin-creatinine ratio; **eGFR**: estimated glomerular filtration rate; **MDRD:** Modification of Diet in Renal Disease. **Sig. bet. grps**.: significance between groups.

Group (1): Non diabetic participants
Group (2): type 2 diabetic patients complicated with DN
Group (3): type 2 diabetic patients without DN

Table 9: showed that group 2 had lower serum sodium and calcium levels, and higher serum potassium and phosphorus levels, while groups 1 and 3 were similar.

Table (9): Comparison between the studied groups according to serum electrolytes

Serum electrolyte	Group (1) (n = 35)	Group (2) (n = 35)	Group (3) (n = 35)	F	P-value
Na (mmol/L)					
Mean ± SD.	138.5 ± 3.38	136.1 ± 4.83	137.3 ± 3.73	3.183*	0.046*
Sig. bet. grps.	p_1=0.035*,p_2=0.429,p_3=0.412				
K (mmol/L)					
Mean ± SD.	4.31 ± 0.48	4.47 ± 0.67	4.0 ± 0.47	6.553*	0.002*
Sig. bet. grps.	p_1=0.456, p_2=0.053, p_3=0.002*				
Calcium (mg/dL)					
Mean ± SD.	8.33 ± 0.38	7.82 ± 0.40	8.30 ± 0.42	18.370*	<0.001*
Sig. bet. grps.	p_1<0.001*,p_2=0.952,p_3<0.001*				
Phosphorus (mg/dL)					
Mean ± SD.	3.30 ± 0.53	4.70 ± 1.18	3.58 ± 0.72	26.401*	<0.001*
Sig. bet. grps.	p_1<0.001*,p_2=0.352,p_3<0.001*				

IQR: Inter quartile range; **SD:** Standard deviation; **F:** F for One way ANOVA test, Pairwise comparison bet. each 2 groups was done using **Post Hoc Test** (Tukey); **p:** p-value between the three studied groups; **p_1**: p-value between group (1) and group (2); **p_2:** p-value between **group (1)** and **group (3); p_3:** p-value between **group(2)** and **group (3)**; *: Statistically significant at $p \leq 0.05$; **Na:** Sodium; **K:** Potassium; **Sig. bet. grps.:** significance between groups.

Group (1): Non diabetic participants
Group (2): type 2 diabetic patients complicated with DN
Group (3): type 2 diabetic patients without DN

A comparison of the lipid profiles of the three groups under study is shown in Table 10.

- Group 2 had higher TG than Group 3 (*p*=.038), but there were no discernible differences in cholesterol (*p*=130), LDL-C (*p*=.072), or HDL-C (*p*=.322).

Table (10): Comparison between the studied groups according to their lipid profile

	Group (1) (n = 35)	Group (2) (n = 35)	Group (3) (n = 35)	F	P-value
Serum Cholesterol (mg/dl)					
Mean ± SD.	161.6 ± 24.06	174.5 ± 29.29	169.5 ± 26.24	2.085	0.130
Serum Triglycerides (mg/dl)					
Mean ± SD.	123.9 ± 23.57	135.8 ± 26.80	121.2 ± 23.27	3.511*	0.034*
Sig. bet. grps.	p_1=0.109, p_2=0.894, p_3=0.038*				
LDL-C (mg/dl)					
Mean ± SD.	87.31 ± 21.21	99.98 ± 22.69	98.45 ± 23.03	2.694	0.072
HDL-C (mgdl)					
Mean ± SD.	49.49 ± 9.13	47.31 ± 8.93	46.77 ± 5.11	1.145	0.322

IQR: Inter quartile range; **SD:** Standard deviation; **F:** F for One way ANOVA test, Pairwise comparison bet. each 2 groups was done using **Post Hoc Test** (Tukey); **p:** p-value between three studied groups; **p_1**: p-value between group (1) and group (2); **p_2:** p-value between **group (1)** and **group (3); p_3:** p- value between **group (2)** and **group (3)**; *: Statistically significant at $p \leq 0.05$; **LDL-C**: low density lipoprotein cholesterol; **HDL-C:** high density lipoprotein cholesterol.

Group (1): Non diabetic participants
Group (2): type 2 diabetic patients complicated with DN
Group (3): type 2 diabetic patients without DN

As shown in Table 11, the mean serum asprosin levels were 18.58 ± 5.96 ng/mL in group 1, 90.09 ± 1.15 ng/mL among group 2, and 28.92 ± 10.12 ng/mL in group 3. Patients in groups 1 and 3 had markedly reduced levels in comparison with those in group 2 (*p*1 < .001* and *p*3 < .001*).

Table (11): Comparison between the studied groups according to Serum asprosin Level

	Group 1 (n = 35)	Group 2 (n = 35)	Group 3 (n = 35)	F	P-value
Serum Asprosin Level (ng/mL)					
Mean ± SD.	18.58 ± 3.96	90.09 ± 1.15	28.92 ± 1.12	141.536	<0.001*
Sig. bet. grps.	p_1<0.001*,p_2=0.068,p_3<0.001*				

IQR: Inter quartile range; **SD:** Standard deviation; **F:** F for One way ANOVA test, Pairwise comparison bet. each 2 groups was done using **Post Hoc Test** (Tukey); **p:** p value between the three studied groups; **p_1**: p value for comparing between group (1) and group (2); **p_2:** p-value between **group (1)** and **group (3); p_3:** p-value between **group (2)** and **group (3)**; *: Statistically significant at $p \leq 0.05$;

Group (1): Non diabetic participants
Group (2): type 2 diabetic patients complicated with DN
Group (3): type 2 diabetic patients without DN

In order to distinguish diabetic patients with DN (group 2) from diabetic patients without DN (group 3), Table 12 shows the cutoff value of serum asprosin. To distinguish between the two groups, serum asprosin at concentrations more than 41.85 ng/mL has a sensitivity of 94.29% and a specificity of 91.43%.

Table (12): Diagnostic performance for Serum asprosin Level to discriminate Group (2) from Group (3)

	AUC	p-value	95% C.I	Cut off#	Sensitivity	Specificity	PPV	NPV
Serum Asprosin Level (ng/mL)	0.900	<0.001*	0.844 – 0.956	>41.85	94.29	91.43	91.7	94.1

AUC: Area Under a Curve p-value: Probability value CI: Confidence Intervals
NPV: Negative predictive value PPV: Positive predictive value
*: Statistically significant at $p \leq 0.05$ #Cut off was choosing according to Youden index.

A comparison of the three groups under study with respect to ultrasonographic renal assessment is shown in Table 13. Ultrasonographic signs of CKD (small size of kidneys, increased echogenicity, and poor corticomedullary differentiation) were detected in 37.1% of the group (2), while no patients in Groups (1) and (3) showed abnormal features.

Table (13): Comparison between the studied groups according to US renal assessment

	Group (1) (n = 35)		Group (2) (n = 35)		Group (3) (n = 35)		χ^2	mcp-value
	No.	%	No.	%	No.	%		
US renal assessment								
Normal	35	100.0	22	62.9	35	100.0	26.879	0.001*
Signs of CKD	0	0.0	13	37.1	0	0.0		
Sig. bet. grps.	p_1<0.001*,p_2–,p_3<0.001*							

χ^2: Chi square test; **MC**: Monte Carlo test; **p:** p-value between the three studied groups; **p_1**: p-value between group (1) and group (2); **p_2:** p-value between **group (1)** and **group (3); p_3:** p-value between **group (2)** and **group (3)**; *: Statistically significant at $p \leq 0.05$.

Group (1): Non diabetic participants
Group (2): type 2 diabetic patients complicated with DN
Group (3): type 2 diabetic patients without DN

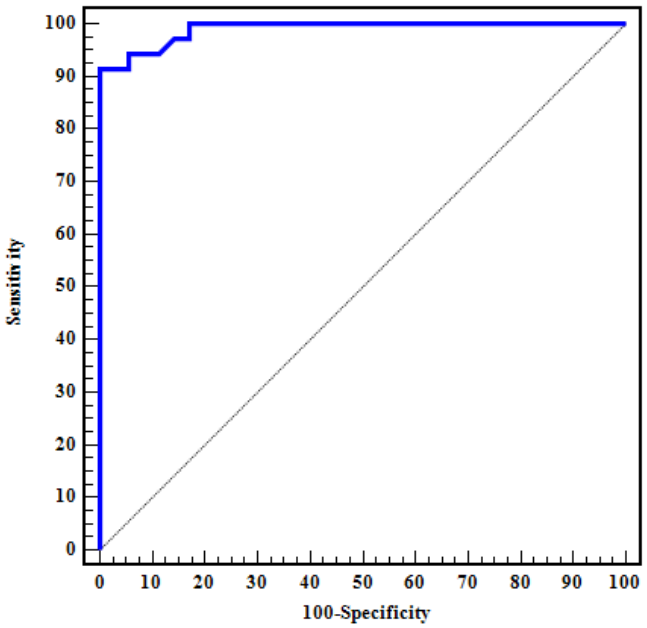

Fig. (1): ROC curve for serum asprosin level to discriminate Group (2) from Group (3)

DISCUSSION

Our study showed that diabetes duration was longer in group 2 vs. group 3 (16.37 ± 4.85 vs. 5.26 ± 2.21 years). Similarly, **Rabea *et al.*** [5] reported longer duration in DN vs. non-nephropathy diabetics (8.61 ± 2.63 vs. 4.88 ± 0.97 years).

Group 2 showed higher BUN (52.27 ± 10.99 vs. 15.64 ± 5.82 vs. 15.51 ± 4.99), creatinine, uric acid, and UACR (846.2 ± 503.6 vs. 25.52 ± 3.35 vs. 19.34 ± 4.05 mg/g), with lower eGFR (29.63 ± 8.34 vs. 110.1 ± 22.84 vs. 118.1 ± 22.92 ml/min/1.73 m²) vs. groups 1 and 3 (p<.001). These findings align with **El Badawy *et al.*** [6], **El-Soudany *et al.*** [7], and **Rabea *et al.*** [5], who reported higher BUN/creatinine and UACR and reduced eGFR in DN compared to non-DN diabetics and controls.

Serum asprosin was significantly higher in group 2 vs. groups 1 and 3 (90.09 ± 1.15 vs. 28.92 ± 10.12 vs. 18.58 ± 5.96 ng/mL, P<0.001), suggesting a role in T2DM and DN pathogenesis.

Abdulsada and Albadr[8] studied 129 T2DM patients and 51 controls, classifying patients by UACR (DN0 <30, DN1 30–299, DN2 ≥300 mg/g). Asprosin was higher in T2DM vs. controls, with DN2 > DN1 > DN0.

Wang *et al.* [9] examined 212 T2DM patients grouped by ACR (DN0, DN1, DN2) and found higher asprosin vs. healthy controls, with DN2 > DN1 > DN0. **Goodarzi *et al.*** [10] reported elevated asprosin in T2DM (6.73 ± 1.67 nmol/L) and T2DM with DN (7.11 ± 1.54 nmol/L) vs. controls (4.81 ± 1.09 nmol/L).

Zhang *et al.* [11] found higher asprosin in T2DM without DKD and early DKD (n=42, n=33) vs. normal glucose tolerance (n=30), with DKD showing the highest levels. **Deng *et al.*** [12] reported elevated asprosin in T2DM with macroalbuminuria [2.37 (1.63–3.57)] and microalbuminuria [2.10 (1.60–2.90)] vs. normoalbuminuria [1.59 (1.18–2.09)], P<0.001.

XU *et al.* [13] studied 82 participants (DM, pre-DKD, DKD) and found higher asprosin levels in pre-DKD and DKD vs. DM. Similarly, **Liu *et al.*** [14] reported elevated asprosin in T2DM and T2DM with DN vs. controls, with higher levels in T2DM with DN than T2DM (P<0.05). exceeding those in the T2DM category (P < 0.05).

With a sensitivity of 94.29%, specificity of 91.43%, and AUC of 0.9, this current study showed that a blood asprosin level at a cutoff value of more than 41.85 ng/mL may distinguish diabetic patients with DN from diabetic patients with no DN.

In comparison to healthy individuals, **Goodarzi *et al.*** [10] showed that asprosin effectively distinguishes between T2DM with nephropathy (AUC [CI] 0.890 [0.831, 0.949], p<.001, cutoff: 5.89, sensitivity: 80%, specificity: 82%) and T2DM (AUC [CI] 0.828 [0.751, 0.904], p<.001, cutoff: 5.46, sensitivity: 72%, specificity: 71%). **Alwahid *et al.*** [15] indicated that the area under the curve (AUC) for asprosin, as assessed using receiver operating characteristic curve analysis, was 0.92, with a P-value of 0.001. At the asprosin threshold of ≥ 17.5 ng/mL, the test exhibited sensitivity of 86.7% and specificity of 80%. **Liu *et al.*** [14] reported that the area under the curve for diagnosing DN using asprosin in conjunction with UAER was 0.879 (95% CI: 0.813-0.974, P < 0.05), with a diagnostic specificity of 84.19% and a sensitivity of 94.57%.

In the present study, 13 patients (37.1%) in group 2 showed abnormal ultrasonographic findings (small kidney size, increased echogenicity, poor corticomedullary differentiation), while none in groups 1 or 3 did (P<0.001).

Similarly, in their analysis of 252 patients, **Ham *et al.*** [16] defined the progression of kidney disease as a reduction in eGFR of ≥10% per year (ΔeGFR/year) or the start of renal replacement therapy. The renal scoring system included parenchymal echogenicity, cortical margin morphology, right kidney length-to-height ratio, cortical thickness-to-kidney length/height ratio (CKH-0/1), and cortical-to-parenchymal thickness ratio (CK/PK-0/1).

In conclusion, serum asprosin levels are markedly elevated in diabetic patients with DN, indicating a potential link to renal disease progression in T2DM. Asprosin may serve as a biomarker and therapeutic target, though longitudinal studies are needed to clarify causality and mechanisms.

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