Original Article

# Asprosin in Early Detection of Nephropathy in Type2 Diabetes Mellitus

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## Abstract

**Background:** Diabetic nephropathy (DN) accounts for the most prevalent cause of chronic kidney disease and end-stage renal disease (ESRD) globally, accounting for about 50% of all ESRD patients that need management with dialysis or a renal transplant. **Objectives:** The aim of this study was to the role of Asprosin as an independent and trustworthy biomarker for the quick diagnosis of DN by knowing the sensitivity and specificity, acceptable to add to the diagnostic protocol. Evaluated fasting blood glucose asprosin, lipid profile, urea, creatinine, and albumin levels in apparently healthy groups, diabetic groups, and diabetic groups with nephropathy to determine their medical significance; asprosin can be used as an independent and trustworthy biomarker for the quick diagnosis of DN by knowing the sensitivity and specificity, acceptable to add to the diagnostic protocol. **Materials and Methods:** Blood samples were obtained from the Diabetes and Endocrine Center in Hilla city, Babylon province's Marjan Medical City, from October 25, 2021 to January 31, 2022. Sixty type 2 diabetes mellitus (T2DM) patients were classified into 30 with diabetes mellitus, 30 with DN, and 30 apparently healthy patients. Serum asprosin levels were measured using a commercial enzyme-linked immunosorbent assay kit. SPSS software was used to conduct the statistical analysis. **Results:** Both T2DM patients with and without nephropathy had considerably higher levels of fasting blood glucose (FBG), asprosin, serum urea, creatinine, total cholesterol, triglyceride (TG), low-density lipoprotein cholesterol, and very low-density lipoprotein cholesterol compared to the healthy appearance group, although both groups had significantly lower levels of albumin and high-density lipoprotein cholesterol (HDL-C). Asprosin additionally showed a positively correlated with serum urea, TG, and HDL-C and shown negative correlation with serum albumin. The sensitivity and specificity of the test at the cut-off value of asprosin 17.5 ng/mL were 86.7% and 80%, respectively, and the area under the curve was 0.92, *P*-value = 0.001. **Conclusion:** Because blood asprosin levels have a sensitivity and specificity of more than 80% in T2DM patients with nephropathy, they can be used as an independent and trustworthy biomarker for the quick diagnosis of DN.

**Keywords:** Asprosin, diabetes, FBS, nephropathy

## Introduction

Diabetes mellitus (DM) (hyperglycemia): brought on characterized by anomalies in insulin secretion, insulin action, or both DM, a metabolic disorders group. Particularly susceptible to long-term harm are the blood arteries, heart, kidneys, nerves, eyes, and kidneys, dysfunction, and failure from persistent hyperglycemia in DM.[1]

Asprosin is a recently identified hormone that is mostly secreted by adipocytes from white adipose tissue during fasting situations. It acts as a glucogenic protein by binding to the OLFR734 receptor and activating the G protein-cyclic AMP (cAMP)-protein kinase A (PKA) pathway, which results in the release of glucose from hepatocytes.[2]

Asprosin acts primarily on the liver, which it stimulates to produce and release glucose.[2] According to Li *et al.*[3] in 2019, asprosin induces glucose manufacture in the liver in both abstaining and obese states by way of the Olfactory 734 (Olfr734) receptor. Activating PKA in the liver via the

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G protein and cAMP messenger system, asprosin, a fasting-induced gluconeogenic hormone, increases glucose release from hepatocytes.[4] Additionally, Romere *et al.* reported that the activity of asprosin is unrelated to the stimulation of the glucagon and catecholamine axis, which is also implicated in the proclamation of glucose.[2]

In vitro research showed that palmitate increased the release of asprosin in human primary islets cells and mouse insulinoma MIN6 cells. Recombinant asprosin was also shown to increase stimulating response and cell dysfunction in a dose-dependent way. Yet, asprosin budget reversed the impact of palmitate, reduced islet cell inflammation, and improved cell survival via increasing glucose-stimulated insulin secretion.

As previously mentioned, the new cytokine asprosin is mostly secreted by adipose cells. Notably, asprosin can, to varying degrees, influence the metabolism of glucose and lipids and is expressed and secreted in response to the body's nutritional or metabolic condition. According to a recent report, antiasprosin can boost creatinine clearance rate while decreasing serum levels of proteinuria, creatinine, urea, and glucose. These effects are dose-dependent. Assessment of spot all patients with type 2 diabetes, individuals with type 1 diabetes during a 5-year period, and patients with type 1 diabetes had their urine albumin/creatinine ratio (UACR) and estimated glomerular filtration rate (eGFR) assessed, and in healthy individuals and in patients with hypertension are recommended by the American Diabetic Association's 2021 guidelines for screening of chronic kidney disease in DM patients on a yearly basis, at the very least.[5]

Due to the glomeruli in the kidneys being damaged by these changes, albuminuria, or the presence of albumin in the urine, develops.[6] However, when the illness worsens, glomerulosclerosis and tubulointerstitial fibrosis appear. These conditions eventually cause renal function to decline, macroalbuminuria to appear, and end-stage renal disease (ESRD) to proceed.[7,8] As a result, one of the evaluation markers of early diabetes nephropathy[9,10] is UACR.

Diabetic nephropathy (DN), which accounts for the most prevalent cause of chronic kidney disease and ESRD globally, accounting for about 50% of all ESRD patients that need management with dialysis or a renal transplant. 30% of individuals with type 1 DM and 40% of those with type 2 DM (T2DM), respectively, will ultimately develop chronic kidney disease.[11]

Amie of the study was the role of asprosin as an independent and trustworthy biomarker for the quick diagnosis of DN by knowing the sensitivity and specificity, acceptable to add to the diagnostic protocol.

## Materials and Methods

Collection of clinical and biochemical data, patient demographic information, such as age, gender, height, weight, and body mass index (BMI), was expressed as weight per square meter (kg/m$^2$). Blood was drawn from the anterior cubital vein after an overnight fast of 8 h in order to monitor various indicators or to be frozen at a temperature of 80°C. Blood urea nitrogen was then detected using the rate technique, and serum creatinine (SCr) was assessed using immunoturbidimetry using the enzymatic method. In addition, by using appropriate enzymatic tests, the blood lipid profile values were calculated for total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), and triglyceride (TG). A glucose oxidase-based test was used to measure plasma glucose at each oral glucose tolerance test time point (Beckman Coulter Inc.). Serum asprosin levels were measured using a commercial enzyme-linked immunosorbent assay kit (Jiangsu, Feiya biological technology, Jiangsu, China; catalog no. MM-1650H1). The sensitivity was 0.1 ng/mL, and the intra- and interassay variability was 10% and 15%, respectively The groups of diabetes and diabetes nephropathy were already prediagnosed by a doctor in this investigation.

### Ethical approval

The study was conducted in accordance with the ethical principles that have their origin in the Declaration of Helsinki. It was carried out with patient's verbal and analytical approval before a sample was taken. The study protocol and subject information, and consent form were reviewed and approved by a local ethics committee according to document number 702 on October 25, 2022 to get this approval.

## Results

### Demographic features of the study groups

Table 1 displays the distribution of the studied group with respect to age.

The results presented age showed that there was no significant difference (*P* value >0.05) between the study groups (T2DM, T2DM with nephropathy, and the control group).

**Table 1: The mean age according to studied groups**

| Characteristics | Study groups | No. | Mean ± SD | *P*-value |
|---|---|---|---|---|
| Age (years) | Control | 30 | 50.62 ± 5.63 | 0.264 |
| | Diabetes | 30 | 48.56 ± 6.34 | |
| | Nephropathy diabetes | 30 | 48.07 ± 4.24 | |

*P* value ≤0.05 was significant

Table 2 displays the distribution of the studied group with respect to body mass index.

The given BMI data demonstrated no statistically significant difference (*P* value >0.05) between the study groups (T2DM, T2DM with nephropathy, and the control group).

Table 3 displays the distribution of the studied group with respect to gender.

The results presented gender showed that there was no significant difference (*P* value > 0.05) between the study groups (T2DM, T2DM with nephropathy, and the control group).

This matching is crucial to avoiding any influence from these factors on the final outcome.

According to the current investigation, both T2DM patients with and without nephropathy had significantly higher mean FBG levels compared to the control group, as shown in Table 2.

Comparison of FBG level in patients (T2DM, Type 2 Diabetes Mellitus Nephropathy (T2DMNP)) with control groups.

The mean ± SD for FBG level in patients (T2DM, T2DMNP) with control groups are shown in Table 4.

The mean ± SD for serum (urea, creatinine) Renal function parameters in studied groups are shown in Table 5.

The mean ± SD for serum albumin level in patients (T2DM, T2DMNP) with control groups are shown in Table 6.

Comparison of lipid profile level in patients (T2DM, T2DMNP) with control groups.

The mean ± SD for lipid profile level in patients (T2DM, T2DMNP) with control groups are shown in Table 7.

Comparison of asprosin level in patients (T2DM, T2DMNP) with control groups.

The mean ± SD for asprosin level in patients (T2DM, T2DMNP) with control groups are shown in Table 8.

**Table 2: The mean BMI according to studied groups**

| Characteristics | Study groups | No. | Mean ± SD | *P*-value |
|---|---|---|---|---|
| BMI (kg/m$^2$) | Control | 30 | 26.3±3.08 | 0.221 |
| | Diabetes | 30 | 27.83±5.16 | |
| | Nephropathy diabetes | 30 | 27.9±3.43 | |

*P* value ≤0.05 was significant

**Table 3: Association between studied groups and gender**

| Gender | Study group | | | Total | *P* value |
|---|---|---|---|---|---|
| | Control | Diabetes | Nephropathy diabetes | | |
| 50 | 16 | 16 | 18 | Male | 0.835 |
| 40 | 14 | 14 | 12 | Female | |
| 90 | 30 | 30 | 30 | Total | |

*P* value ≤ 0.05 was significant

**Table 4: Fasting blood glucose in studied groups**

| Variables | Study groups | No. | Mean ± SD | *P*-value |
|---|---|---|---|---|
| FBG (mmol/L) | Control | 30 | 5.47±0.766 | 0.0001* |
| | Diabetes | 30 | 12.65±4.21 | |
| | Nephropathy diabetes | 30 | 14.56±4.63 | |

**P* value ≤0.05 was significant, FBG

**Table 5: Renal function parameters in studied groups**

| Variables | Study group | No. | Mean ± SD | *P*-value |
|---|---|---|---|---|
| Blood urea (mmol/L) | Control | 30 | 5.05±1.04 | <0.05* |
| | Diabetes | 30 | 5.7±0.95 | |
| | Nephropathy with DM | 30 | 12.51±3.27 | |
| Serum creatinine (mmol/L) | Control | 30 | 0.35±0.12 | <0.05* |
| | Diabetes | 30 | 0.41±0.21 | |
| | Nephropathy with DM | 30 | 1.21±0.56 | |

**P* value ≤0.05 was significant

**Table 6: Serum albumin parameter in studied groups**

| Parameter | Study groups | No. | Mean ± SD | *P*-value |
|---|---|---|---|---|
| Serum albumin (g/L) | Control | 30 | 43.13 ± 4.74 | **0.0001** |
| | Diabetes | 30 | 42.62 ± 3.63 | |
| | Nephropathy diabetes | 30 | 26.34 ± 3.77 | |

*P* value ≤ 0.05 was significant

**Table 7: Comparison of lipid profile level in patients (T2DM, T2DMNP) with control groups**

| Parameters | Study group | No. | Mean ± SD | *P*-value |
|---|---|---|---|---|
| Cho (mmol/L) | Control | 30 | 2.98 ± 0.46 | 0.025 |
| | Diabetes | 30 | 3.45 ± 0.58 | |
| | Nephropathy | 30 | 3.99 ± 0.68 | |
| TG (mmol/L) | Control | 30 | 1.81 ± 0.48 | <0.05 |
| | Diabetes | 30 | 3.02 ± 0.56 | |
| | Nephropathy | 30 | 3.69 ± 0.84 | |
| HDL (mmol/L) | Control | 30 | 0.84 ± 0.14 | >0.05 |
| | Diabetes | 30 | **0**.38 ± 0.13 | |
| | Nephropathy | 30 | 0.27 ± 0.11 | |
| LDL (mmol/L) | Control | 30 | 2.7 ± 0.44 | 0.008 |
| | Diabetes | 30 | 3.8 ± 0.58 | |
| | Nephropathy | 30 | 4.2 ± 0.49 | |
| VLDL (mmol/L) | Control | 30 | 0.4 ± 0.08 | 0.000 |
| | Diabetes | 30 | 0.6 ± 0.11 | |
| | Nephropathy | 30 | 0.72 ± 0.16 | |

*P* value ≤0.05 was significant

**Table 8: Comparison of asprosin level in patients (T2DM, T2DMNP) with control groups**

| Parameter | Study groups | No. | Mean ± SD | *P*-value |
|---|---|---|---|---|
| Serum asprosin (ng/mL) | Control | 30 | 12.12 ± 1.24 | **0.002** |
| | Diabetes | 30 | 15.95 ± 1.23 | |
| | Nephropathy diabetes | 30 | 18.20 ± 0.97 | |

*P* value ≤ 0.05 was significant

BMI, TG, TC, and LDL-C all had a positive correlation with serum asprosin concentrations ($r$ = 0.44, $P$ = 0.739), ($r$ = 0.33, $P$ = 0.01), ($r$ = 0.131, $P$ = 0.317), and ($r$ = 0.22, $P$ = 0.081) respectively, as shown in Table 9. LDL-C was a separate healthy risk that affected asprosin concentration.[12]

The area under the curve for our study's findings at the asprosin, as determined by the receiver operating characteristic curve analysis, was 0.92, with a *P*-value of 0.001. At the asprosin cut-off value of ≥ 17.5 ng/mL, the test's sensitivity and specificity were 86.7% and 80%, as shown in Figure 1.

According to the results of the current study, asprosin is positively correlated with urea and creatinine and negatively correlated with albumin in T2DM patients, which suggests that circulating asprosin may play a role in the pathogenesis of DN by influencing blood glucose levels, insulin resistance, and inflammatory reactions in the kidney. Figures 2-5 illustrate correlation of asprosin with serum urea, serum creatinine, serum albumin, and TG, respectively..

**Table 9: Correlation between asprosin with other study variables in T2DM patients with nephropathy**

| Study variables | Asprosin (ng/mL) | |
|---|---|---|
| | *r* | *P*-value |
| BMI | 0.44 | 0.739 |
| Sugar | 0.51 | 0.700 |
| Blood urea | 0.562 | 0.000* |
| Creatinine | 0.468 | 0.000* |
| TG | 0.33 | 0.01* |
| TC | 0.131 | 0.317 |
| HDL-C | -0.265 | 0.041* |
| LDL-C | 0.22 | 0.081 |
| VLDL-C | -0.327 | 0.11 |
| Albumin | -0.656 | 0.000* |

*P*-value <0.05* was considered to be statically significant

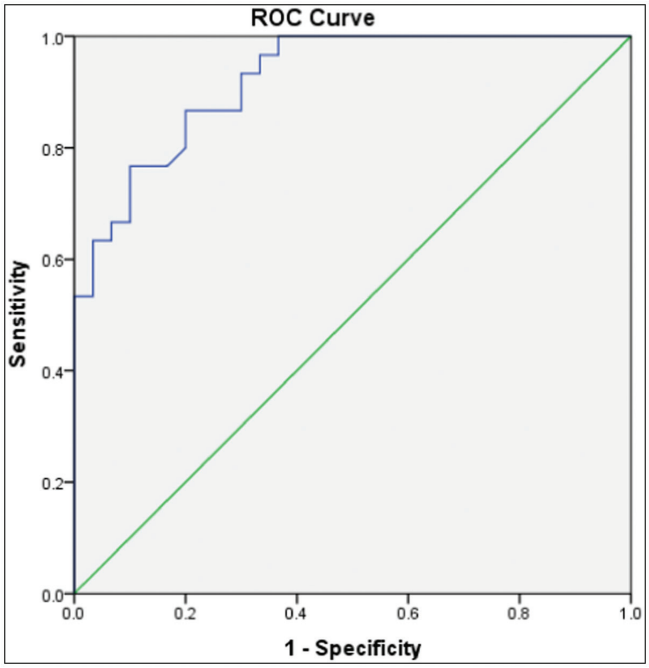


**Figure 1:** Receiver operating characteristic curve for sensitivity and specificity of asprosion to predict diabetes nephropathy

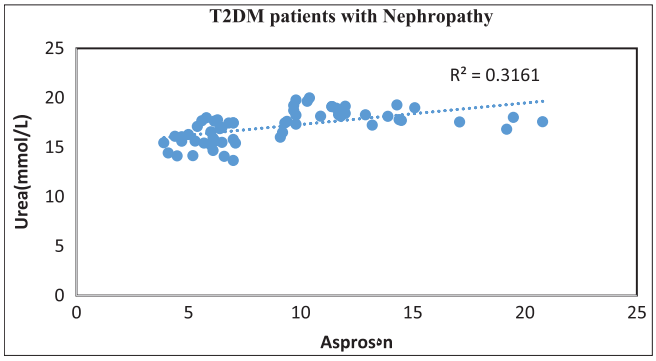


**Figure 2:** Correlation between asprosin (ng/mL) and urea (mmol/L) ($r$ = 0.468, $P$ = 0.000). T2DM patients with nephropathy

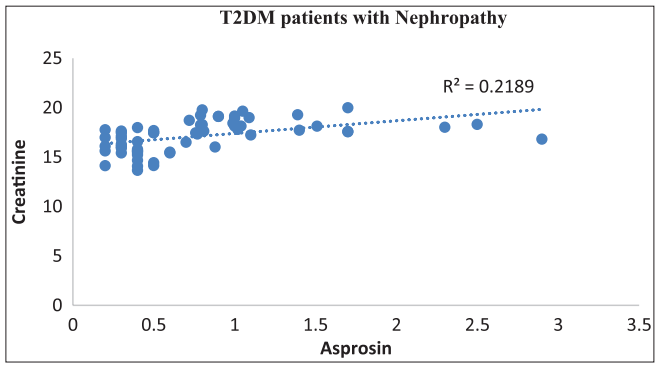


**Figure 3:** Correlation between asprosin (ng/mL) and creatinine (mmol/L). T2DM patients with nephropathy. ($r$ = 0.468, $P$ =0.000)

## Discussion

According to the findings of the current investigation, FBG levels were considerably higher in T2DM patients with and without nephropathy when associated with the ostensibly healthy group ($P$ > 0.000). These findings corroborate earlier studies that contend hyperglycemia is the primary factor contributing to the development of diabetes nephropathy.[10]

One of the accelerated generations of advanced glycation end products and activation of their receptor (RAGE), which elevates protein kinase C, nuclear factor-kappa B, transforming growth factor, and connective tissue growth factors, are the biological processes that lead to hyperglycemia-mediated kidney injury. A prolonged subacute inflammatory process and the resulting production of reactive oxygen species are crucial in the development of DN.[13]

According to this study, patients with T2DM who had nephropathy had greater values of the parameters measured during renal function tests (urea and creatinine) than patients without nephropathy or the control group ($P$-value 0.001). According to Table 5, the simplest method of keeping track of renal function is through blood tests for urea and creatinine. These substances are common metabolic wastes that the kidneys eliminate. The breakdown of proteins produces urea. Due to abnormal excretion in kidney disease, these substances build up in the body and raise urea levels in the blood. The main metabolite of creatine, which is mostly found in skeletal muscle, is SCr.

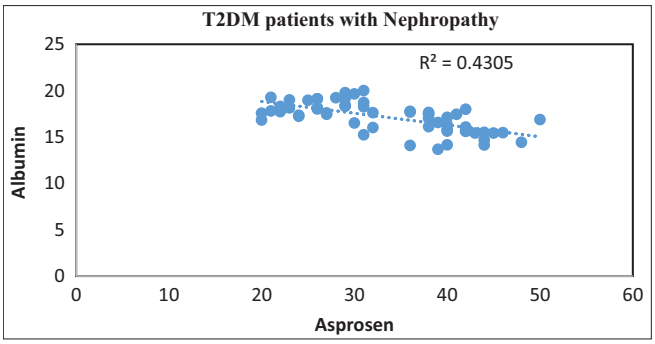


**Figure 4:** Correlation between asprosin (ng/mL) and albumin (mmol/L) ($r$ = -0.656, $P$ = 0.000). T2DM patients with nephropathy

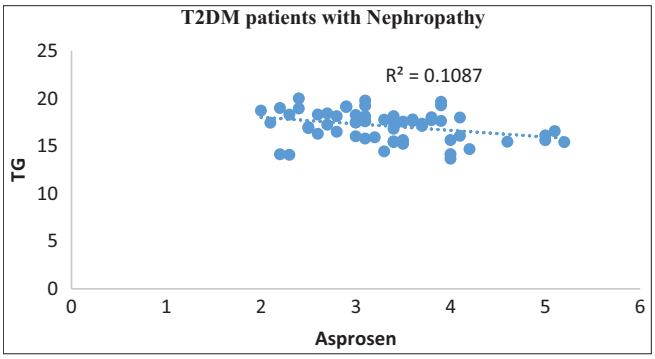


**Figure 5:** Correlation between asprosin (ng/mL) and TG (mmol/L) ($r$ = -0.33, $P$ = 0.010). T2DM patients with nephropathy

Creatine levels per skeletal mass and the rate at which it is degraded are both constant. Due to the remarkable stability of plasma creatinine content, it serves as a direct indication of skeletal muscle mass.[14] This investigation reveals a considerable variation in blood urea and creatinine levels between T2DM patients with nephropathy and the control group and those without nephropathy. As the disease reached its advanced stages, patients' levels of urea and creatinine increased.[15] Because the anhydrous form of creatine that is found in muscles is called creatinine. Since creatinine is filtered by the glomerulus, measuring SCr is considered an indirect measure of glomerular filtration.

The SCr and urea levels increase as GFR declines. It's interesting to note that Harita *et al.*[7] proposed may that type 2 diabetes risk is raised by decreased SCr, which be due to a decrease in skeletal muscle volume. Less target sites for insulin in skeletal muscle result in increased insulin resistance. T2DM is brought on as a result.[16] As indicated in Table 4, there was a highly significant difference in serum albumin value (mean ± SD) between the T2DM with nephropathy, T2DM without nephropathy, and control groups in this investigation [Table 6] ($P = 0.0001$).

However, there was no discernible difference between the healthy group and the T2DM without nephropathy ($P = 0.09$).

The most prevalent plasma protein, human serum albumin, is produced in the liver and secreted into the vascular space, where it is distributed throughout all bodily tissues.[17] Human serum albumin is a monomeric macromolecule having a molecular mass of about 66.5 kDa.[18]

It is crucial for maintaining homeostasis and maintaining the proper balance of hydrostatic and colloid osmotic pressure inside the vessels growing proof indicates that hypoalbuminemia was brought on by insufficient protein or energy despite the disorders implicated, consumption is a substantial risk factor and predictor of increased morbidity and death and may result from reduced hepatic synthesis, decreased intestinal absorption, increased tissue catabolism, or increased loss.[8]

The findings of this investigation strongly revealed a connection between albumin levels in the serum and the prognosis of the kidneys. However, the reduction of eGFR is substantially and independently correlated with serum albumin levels. On the other hand, the finding that patients with lower serum albumin levels were high likely to develop ESRD (may prompt nephrologists to closely monitor these patients and maybe administer a more aggressive course of treatment. Additionally, proteinuria and glomerular lesions, two recognized risk factors for the advancement of diabetes nephropathy, had a substantial inverse connection with serum albumin levels, which may help to explain the link between hypoalbuminemia and renal prognosis. The findings of this study revealed that low serum albumin levels might be useful for DN prognosis. Our findings revealed patients with lower serum albumin levels were more likely to develop ESRD.[9]

The current investigation discovered that patient groups with T2DM and T2DMNP had substantially higher mean blood levels of (CHO, TG, LDL, and very low-density lipoprotein [VLDL]) than controls ($P$ value 0.005), at mean ± SD as shown in Table 7.

A major clinical condition identified in people with DM is dyslipidemia. Increased plasma TG levels in diabetic dyslipidemia are characterized by low levels of HDL-C and an increase in the concentration of small, dense LDL (sdLDL) particles. The migration of free fatty acids from adipose tissue to the liver causes an increase in TG production as a result of insulin resistance.

Long-term diabetes would cause lipoprotein lipase to malfunction, causing the accrual of big, TG-rich VLDL particles that, in turn, produce sdLDL particles.[10] Therefore, even when T2DM patients' levels of LDL-C are not increased, they nevertheless have a majority of atherogenic sdLDL particles.[19]

One of the most serious side effects of diabetes is DN, which is characterized by proteinuria and renal insufficiency. It is a recognized risk factor for both mortality and cardiovascular events.[20] Patients with DN are known to have abnormalities in their lipoproteins, and the majority of them pass away from cardiovascular causes before their renal dysfunction develops into ESRD.[21,22] Both quantitative and compositional alterations in lipids are brought on by DN. Atherogenic sdLDL particle presence is elevated even in DN patients with normal TC and TG values.[21] Dyslipidemia has also been demonstrated to hasten the rate of kidney injury.

Clinical and experimental studies have revealed that dyslipidemia among diabetes patients causes a progressive deterioration of renal function.[23] Lipids have been suggested to harm kidney's vascular, mesangial, and tubular cells, albeit the exact mechanism of action is still unclear.[24] The kidney damage brought on by dyslipidemia in diabetic patients is accelerated further by underlying dysglycemia.[25] Thus, dyslipidemia and nephropathy exacerbate the clinical condition and raise the risk of renal or cardiovascular complications in diabetes patients by working in concert. Studies from India have examined lipid deviations in individuals with chronic renal failure.[26,27]

Hyperlipidemia is currently regarded as an independent and important driver of the course of renal disease in diabetes, despite the fact that a number of variables may influence the genesis and advancement of DN. According to the findings of the current investigation, both T2DM patients with and without nephropathy had considerably higher levels of lipid profile as compared to the ostensibly healthy control group. These findings

confirm findings from other studies that dyslipidemia in diabetes. Moorhead *et al.*[28] published their findings more than 20 years ago and found a link between hyperlipidemia and damage to glomerular capillaries. The authors hypothesized that chronic renal damage is promoted by the ongoing filtration of lipids and lipoproteins.[28]

Numerous observational studies that followed provided evidence for the connection between glomerulosclerosis development and albuminuria and high serum lipid levels.[29,30] According to Ravid *et al.*,[30] there was a strong correlation between the degree of albuminuria and the blood cholesterol levels at the beginning and end of a 5-year cohort group of patients with T2DM. Similarly to this, Gall *et al.*[31] showed a substantial association between increased urine albumin excretion and total blood cholesterol.

VLDL cholesterol (VLDL-C), LDL-C, intermediate-density lipoprotein cholesterol, and TGs are all plasma concentrations that are higher in people with DN but lower in people without DN[32].The aforementioned lipid profile is known as "diabetic dyslipidemia," and those with type 2 diabetes are most likely to have it. It's interesting to note that when renal function and urine albumin excretion deteriorate, the aforementioned lipid abnormalities in DN become more obvious.[12,28,33] Insulin resistance and faulty insulin action in the metabolism of lipoproteins are the causes of lipid changes.

The end result is increased lipolysis, which leads to an increase in free fatty acid synthesis and VLDL-C, a defect in LPL activity that prolongs the lifespan of chylomicrons and VLDL-C in circulation, an increase in cholesterol esters transferred into LDL-C, which is rich in TGs, and finally an increase in plasma TGs and a decreased ratio of LPL-C to hepatic lipase that accelerates HDL-C breakdown.[34] Due to the fact that excessive high blood lipid levels increase blood viscosity, which reduces albumin leakage and may be exacerbated by decreased blood supply to the kidneys, which results in renal ischemia, hypoxia, destruction to glomerular capillaries, and increased permeability.

Additionally, this study discovered that there was a significant difference in the serum asprosin levels (mean ± SD) between the T2DM with nephropathy, T2DM without nephropathy, and control groups, as shown in Table 8.

A recently identified hormone called asprosin is largely produced and released by white adipose tissues before being transferred to the liver. Asprosin increases hepatic gluconeogenesis, which ultimately results in higher amounts of glucose and insulin. Additionally, asprosin crosses the blood–brain barrier and directly activates orexigenic neurons through cAMP-dependent signals, stimulating appetite and causing weight gain.[2]

Asprosin is known to contribute to insulin resistance and diabetes, according to recent studies. Compared to controls, T2DM patients had higher levels of serum asprosin.[2,35] Additionally, compared to healthy controls, serum asprosin levels were higher in people with poor glucose regulation.[36] However, the goal of this study was to look at asprosin's potential use in T2DM patients with early-stage renal illnesses.

The findings of this study showed that T2DM patients with nephropathy had considerably higher serum asprosin levels than T2DM patients without nephropathy and the normal control groups. These findings imply that asprosin may contribute to the onset of DN. The latest findings are consistent with earlier research.[37]

Asprosin plays a significant function in controlling blood glucose levels (it induces hepatic glucose release) and insulin sensitivity, which may be the possible mechanism underlying higher asprosin levels in DN. So, the two main causes of DN are hyperglycemia and insulin resistance. The treatment of diabetes and diabetic complications may therefore benefit from the therapeutic targeting of asprosin. Microvascular problems in people with diabetes may be linked to asprosin. Elevated serum asprosin concentrations. BMI, TG, TC, and LDL-C all had a positive correlation with serum asprosin concentrations ($r$ = 0.44, $P$ = 0.739), ($r$ = 0.33, $P$ = 0.01), ($r$ = 0.131, $P$ = 0.317), and ($r$ = 0.22, $P$ = 0.081), respectively. LDL-C was a separate healthy risk that affected asprosin concentration.[12] In accordance with our findings, other investigations also showed that asprosin levels in obese and diabetic people were favorably connected inversely linked with HDL-C and positively with TG and LDL-C.[12,33] As a result, dyslipidemia may play a role in asprosin's ability to treat diabetes. According to the results of the current study, asprosin is positively correlated with urea and creatinine and negatively correlated with albumin in T2DM patients, which suggests that circulating asprosin may play a role in the pathogenesis of DN by influencing blood glucose levels, insulin resistance, and inflammatory reactions in the kidney. [Figures 2-5] for blood urea, creatinine, albumin, and TG, respectively, illustrate this.

## Conclusion

According to our research, T2DM and T2DM with DN. Their serum contains more asprosin than normal. The results of this study suggest that aspirosine may contribute to the development and progression of DN in people with T2DM. It also suggests that aspirosine may be a significant contributor to the onset of T2DM and a suitable biomarker for the prediction of both T2DM and T2DM with DN. The findings of the current study offer novel and crucial clinical understandings of the role that asprosin plays in the etiology of T2DM and T2DM with DN.

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## Conflicts of interest
There are no conflicts of interest.



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