AMERICAN JOURNAL OF BIOMEDICINE AND PHARMACY
https://biojournals.us/index.php/AJBP
ISSN: 2997-7177

Article

Assessment of Serum Asprosin Levels in Obesity and Type 2 Diabetes Mellitus

Sawsan Salman Hosi*[1]

1. Department of Biochemistry, College of Medicine, University of Tikrit, Iraq
*Correspondence: sawsan.salman@tu.edu.iq

Citation: Hosi S S. Assessment of Serum Asprosin Levels in Obesity and Type 2 Diabetes Mellitus. American Journal of Biomedicine and Pharmacy 2026, 3(9), 71-80.

Received: 10th Jun 2026
Revised: 11th Jul 2026
Accepted: 20th Aug 2026
Published: 07th Sept 2026

Abstract: Obesity is a common metabolic disorder associated with insulin resistance, chronic low grade inflammation and elevated cardiovascular risk, and type 2 diabetes mellitus (T2DM) is another metabolic disorder of insulin resistance. Recently Asporin has been identified as an adipokine which may play a role as a regulator of glucose metabolism and energy balance, but whose clinical relevance is not yet established. The purpose of this study was to assess the association of circulating asprosin concentration with anthropometric, metabolic and inflammatory parameters in obese people with T2DM. This case-control study was conducted in Tikrit Teaching Hospital, Tikrit, Iraq from May 2025 to April 2026. Participants ages 20 to 70 years were divided into four groups: healthy controls, obese non-diabetic, non-obese patients with T2DM, and obese patients with T2DM. Anthropometric data were recorded and blood samples were taken after an overnight fast for biochemical parameters, insulin and asprosin by ELISA. There are significantly higher concentrations of asprosin in all groups of patients than in healthy controls, with the highest concentration found in obese patients with T2DM ($P < 0.001$). Other measures of obesity, such as fasting glucose, HbA1c, insulin, HOMA-IR, inflammatory markers, triglycerides, TyG index, and lipid accumulation product were also significantly elevated in the diseased groups. The correlation analysis results revealed positive correlations between asprosin and obesity indices, glycemic variables, insulin resistance, triglycerides, leptin, hs-CRP, TyG index, and lipid accumulation product, and negative correlation between asprosin and adiponectin. The multiple regression model revealed that BMI, HbA1c, HOMA-IR and TyG index were independent factors for serum asprosin. The results indicate that the high level of asprosin is a good indicator of metabolic dysfunction and could be a potential target for therapy and biomarker in obesity-related T2DM. It's clinical usefulness needs further prospective studies to confirm.

Keywords: Asprosin; Obesity; Type 2 Diabetes Mellitus; Insulin Resistance; Adipokines.

Introduction

Obesity and type 2 diabetes mellitus (T2DM) are among the most prevalent chronic metabolic diseases in the world and are a significant public health problem due to the rising prevalence and the complications that are associated with them. They are driven by genetic, environmental, hormonal, and

lifestyle factors, and result in insulin resistance, chronic low-grade inflammation, and metabolic dysfunction [1]. T2DM, cardiovascular disease and healthcare costs are now a major factor in the global burden of obesity, both in developed and developing countries [2].

Today, adipose tissue is known to be an active endocrine gland and not just a place of energy storage. It produces several biologically active molecules called adipokines that affect appetite, insulin sensitivity, glucose levels, fat metabolism and inflammation [3]. The imbalance in adipokine secretion is strongly linked to obesity, insulin resistance, metabolic syndrome and T2DM [4]. In addition to well-known adipokines like adiponectin and leptin, new adipokines have been discovered and are driving further advances of knowledge on metabolic diseases.

Asprosin was first discovered in 2016 and is a fasting-induced adipokine primarily released from white adipose tissue after proteolysis of profibrillin [5]. It activates the cyclic adenosine monophosphate (cAMP) pathway, which activates the hepatic release of glucose, and helps to maintain blood glucose during fasting [5]. Moreover, it has been shown that asprosin enters the blood-brain barrier and stimulates appetite-controlling neurons in the hypothalamus, suggesting a crucial function in the regulation of energy balance and body weight [6].

Others have found high levels of asprosin in obese, T2DM, metabolic syndrome, and insulin resistant patients [7]. While the specific mechanism by which it is involved in obesity still remains to be established, its effects on the decrement of insulin signaling, increase in hepatic glucose production, appetite stimulation, and inflammatory response via oxidative stress and activation of pro-inflammatory pathways are increasingly recognized [9]. Thus, asprosin is gaining considerable interest as a biomarker and a therapeutic target for metabolic diseases.

Insulin resistance is regarded as the main mechanism through which obesity is associated with T2DM. As insulin action diminishes, there is hyperglycemia, compensatory hyperinsulinemia, and ultimate pancreatic β-cell dysfunction [10]. Asprosin has also been shown to be associated with elevated levels of HbA1c, fasting insulin, and HOMA-IR, which are markers for glucose dysregulation and insulin resistance [11].

Dyslipidemia is defined as disorders associated with abnormal lipids and is often found in obese individuals, in whom T2DM is accompanied by increased cardiovascular risk and elevated inflammatory markers, including high-sensitivity C-reactive protein (hs-CRP) [12]. The recent study also revealed very strong correlations between serum asprosin and unfavorable lipid levels, inflammatory markers, and cardiometabolic risk factors [13]. Therefore, this study was designed to assess the serum level of asprosin in the obese population and patients with T2DM and to explore the correlation of asprosin with anthropometric parameters, glycemic status, IR, lipid profile, inflammatory markers, adipokines, and CMRIs.

Materials and Methods

Study Design and Participants

This is a prospective case-control study conducted from 1st of May 2025 to 1st of April 2026 at Tikrit Teaching Hospital, Salah Al Din, Iraq. This study aimed to assess the relationship between circulating serum asprosin and anthropometric, biochemical and metabolic parameters in obese and type 2 diabetes mellitus (T2DM) subjects. A total of 200 subjects (20–70 years old) of both sexes were enrolled and gave informed written consent after obtaining institutional approval. The participants were randomly divided into four groups of equal size (50 per group): healthy non-obese controls, obese non-diabetic controls, non-obese patients with T2DM, and obese patients with T2DM. All laboratory findings were compared to the healthy controls which acted as the control population.

Eligibility Criteria

Patients with type 1 diabetes mellitus, gestational diabetes, pregnancy, lactation, malignancy, autoimmune disorders, chronic inflammatory diseases, acute infections, severe liver disease, chronic kidney disease or recent cardiovascular events were excluded. The study excluded participants taking drugs that are known to have a significant effect on glucose metabolism or adipokine production.

Clinical and Anthropometric Assessment

Structured questionnaires and review of medical records were used for demographic and clinical data. Data consisted of age, sex, smoking status, diabetes duration, family history, current antidiabetic treatment and medical comorbidities. Body weight, height, waist circumference and hip circumference were measured using standard procedures in anthropometric evaluation. These were then followed by calculating the BMI, WHR and WHtR to evaluate overall and central obesity.

Sample Collection and Laboratory Measurements

Venous blood (approx. 10-12 mL) was collected following an overnight fast under aseptic conditions. All serum samples were then centrifuged at 3000 RPM for 10 minutes and then kept frozen at −80°C for analysis. Asprosin concentration was measured in the serum using a commercially available enzyme-linked immunosorbent assay (ELISA) following the manufacturer's instructions, and all samples were duplicated to ensure analytical reliability.

The biochemical parameters were measured routinely, such as fasting blood glucose (FBG), glycated hemoglobin (HbA1c), fasting insulin, total cholesterol, triglycerides, HDL-C, LDL-C, VLDL-C, blood urea, serum creatinine and aspartate aminotransferase (AST). Homeostatic Model Assessment indices (HOMA-IR and HOMA-β) were calculated to estimate insulin resistance and pancreatic β-cell function, respectively.

Assessment of Inflammatory and Cardiometabolic Biomarkers

Other metabolic markers were also measured to get a complete picture of the metabolic status. The levels of serum leptin and adiponectin were used as markers of adipose tissue function, and high-sensitivity C-reactive protein (hs-crp) was used as a marker of systemic inflammation. Serum uric acid was added due to its known association with obesity and metabolic syndrome. To further assess the cardiometabolic risk, the triglyceride-glucose (TyG) index, atherogenic index of plasma (AIP), lipid accumulation product (LAP) and estimated glomerular filtration rate (eGFR) were calculated.

Outcome Measures

The main findings of the study were the levels of serum Asprosin in the study groups. Secondary outcomes involved assessing the associations between serum asprosin and anthropometric parameters, glycemic control, lipid profile, insulin resistance, inflammatory markers, adipokines, and cardiometabolic risk markers.

Statistical Analysis

The statistical package for the social sciences (SPSS) version 27.0 was used for data analysis. Data for continuous variables were reported as mean ± SD, while the categorical variables were reported by frequency and percentage. Data distribution was tested using Shapiro–Wilk test. One-way analysis of variance (ANOVA) with subsequent Tukey's post hoc test or Kruskal-Wallis test was used for comparison among the groups depending on the normality of the data. Pearson's or Spearman's correlation coefficient was used as appropriate to examine the relationships. Independent factors associated with asprosin after adjustment for potential confounding factors were determined using multiple linear regression analysis. $P < 0.05$ was regarded as statistically significant.

Results

200 individuals, equally divided into four groups—healthy controls, obese non-diabetics, non-obese T2DM patients, and obese T2DM patients—were involved in this research. Table 1 displays the demographic information for the groups under study.

Table 1. Demographic Characteristics of the Study Population.

Parameter	Control (n=50)	Obese (n=50)	T2DM (n=50)	Obese + T2DM (n=50)	P-value
Age (years)	46.2 ± 8.1	47.5 ± 7.6	49.3 ± 8.8	50.1 ± 9.2	0.082
Male/Female	26/24	25/25	27/23	28/22	0.921
Smoking (%)	18%	20%	24%	28%	0.351
Duration of Diabetes (years)	—	—	7.4 ± 3.2	8.1 ± 3.7	0.441

Table 2 shows that the anthropometric measurements had significant differences between obese groups and controls; with higher indices related to obesity in obese groups.

Table 2. Anthropometric Parameters of the Studied Groups.

Parameter	Control	Obese	T2DM	Obese + T2DM	P-value
Weight (kg)	71.5 ± 9.2	98.4 ± 11.5	74.2 ± 10.1	102.8 ± 12.6	<0.001
BMI (kg/m²)	24.3 ± 2.1	34.8 ± 3.7	25.6 ± 2.5	36.4 ± 4.2	<0.001
Waist Circumference (cm)	85.7 ± 7.5	112.3 ± 10.4	92.6 ± 8.1	118.5 ± 11.2	<0.001
Hip Circumference (cm)	98.5 ± 6.2	120.7 ± 8.8	102.8 ± 7.1	126.4 ± 9.6	<0.001
WHR	0.87 ± 0.05	0.93 ± 0.06	0.90 ± 0.04	0.94 ± 0.05	<0.001
WHtR	0.49 ± 0.04	0.66 ± 0.06	0.54 ± 0.05	0.70 ± 0.07	<0.001

Glycemic measures revealed anomalies in glucose metabolism in diabetic obese individuals as well as substantial differences across diabetes groups (Table 3).

Table 3. Glycemic Profile of the Study Groups.

Parameter	Control	Obese	T2DM	Obese + T2DM	P-value
FBG (mg/dL)	89.2 ± 8.4	96.3 ± 10.7	176.4 ± 38.1	194.8 ± 42.5	<0.001
HbA1c (%)	5.2 ± 0.4	5.7 ± 0.5	8.3 ± 1.4	9.1 ± 1.7	<0.001
Fasting Insulin (μIU/mL)	8.1 ± 2.4	17.5 ± 4.2	15.8 ± 4.5	24.3 ± 5.8	<0.001
HOMA-IR	1.8 ± 0.5	4.2 ± 1.1	6.8 ± 1.9	11.7 ± 3.2	<0.001
HOMA-β	102.5 ± 15.6	118.3 ± 18.1	73.4 ± 14.2	58.2 ± 11.8	<0.001

The significant dyslipidemia was observed in the obese and diabetic patients in the lipid profile parameters (Table 4).

Table 4. Lipid Profile Parameters.

Parameter	Control	Obese	T2DM	Obese + T2DM	P-value
TC (mg/dL)	168.5 ± 24.6	213.4 ± 32.1	221.7 ± 35.2	245.6 ± 39.4	<0.001
TG (mg/dL)	112.8 ± 28.5	182.3 ± 44.6	196.4 ± 48.1	245.2 ± 61.7	<0.001
HDL-C (mg/dL)	52.4 ± 8.1	41.6 ± 7.2	39.8 ± 6.5	34.2 ± 5.9	<0.001
LDL-C (mg/dL)	93.6 ± 18.2	136.5 ± 25.7	143.4 ± 28.3	162.8 ± 31.5	<0.001
VLDL-C (mg/dL)	22.6 ± 5.7	36.5 ± 8.9	39.3 ± 9.6	49.0 ± 12.4	<0.001

All obese and diabetic groups had significantly higher serum asprosin levels, especially among the obese diabetic group (Table 5).

Table 5. Serum Asprosin Levels in the Studied Groups.

Group	Serum Asprosin (ng/mL)
Control	5.1 ± 1.2
Obese	8.6 ± 1.9
T2DM	10.2 ± 2.1
Obese + T2DM	13.8 ± 2.7

P-value <0.001

Patients with obesity and diabetes had significant changes in inflammatory and adipokine biomarkers (Table 6).

Table 6. Novel Metabolic Biomarkers.

Parameter	Control	Obese	T2DM	Obese + T2DM	P-value
Leptin (ng/mL)	8.4 ± 2.6	24.7 ± 6.1	18.2 ± 5.4	31.5 ± 7.8	<0.001
Adiponectin (μg/mL)	12.8 ± 2.4	7.5 ± 1.8	6.8 ± 1.6	4.9 ± 1.3	<0.001
hs-CRP (mg/L)	1.2 ± 0.5	4.1 ± 1.3	4.8 ± 1.6	7.2 ± 2.1	<0.001
Uric Acid (mg/dL)	4.8 ± 1.0	6.7 ± 1.2	6.9 ± 1.4	7.8 ± 1.6	<0.001

Cardiometabolic risk indices showed a gradual rise from controls to obese diabetic patients (Table 7).

Table 7. Cardiometabolic Risk Indices.

Parameter	Control	Obese	T2DM	Obese + T2DM	P-value
TyG Index	8.1 ± 0.3	8.9 ± 0.4	9.3 ± 0.5	9.9 ± 0.6	<0.001
AIP	0.11 ± 0.05	0.38 ± 0.08	0.45 ± 0.09	0.62 ± 0.11	<0.001
LAP	21.4 ± 5.6	59.2 ± 12.4	63.8 ± 14.7	89.6 ± 18.5	<0.001

Biochemical indicators related to the kidney and liver are summarized in Table 8.

Table 8. Liver and Kidney Function Parameters.

Parameter	Control	Obese	T2DM	Obese + T2DM	P-value
Creatinine (mg/dL)	0.82 ± 0.14	0.89 ± 0.16	0.95 ± 0.18	1.02 ± 0.21	0.012
Urea (mg/dL)	29.1 ± 6.4	31.7 ± 7.5	35.6 ± 8.1	38.8 ± 9.4	0.004
eGFR (mL/min/1.73m²)	102.5 ± 11.4	98.3 ± 12.1	92.7 ± 13.8	88.6 ± 14.5	0.009
ALT (U/L)	24.3 ± 7.1	38.5 ± 10.4	40.2 ± 11.8	49.6 ± 13.2	<0.001
AST (U/L)	21.8 ± 5.4	31.2 ± 7.6	33.4 ± 8.5	40.8 ± 9.7	<0.001

Table 9 shows Significant positive correlations were found between serum asprosin levels and BMI, fasting glucose, HbA1c (Fig. 3), fasting insulin, HOMA-IR (Fig. 4), triglycerides, leptin (Fig. 1), hs-CRP, TyG index and LAP, while a significant negative correlation was found with adiponectin levels (Fig. 2).

Table 9. Correlation of Serum Asprosin with Metabolic Parameters.

Variable	r-value	P-value
BMI	0.642	<0.001
Waist Circumference	0.618	<0.001
FBG	0.701	<0.001
HbA1c	0.683	<0.001
HOMA-IR	0.755	<0.001
Triglycerides	0.597	<0.001
Leptin	0.628	<0.001
hs-CRP	0.581	<0.001
TyG Index	0.718	<0.001
LAP	0.662	<0.001
Adiponectin	-0.549	<0.001

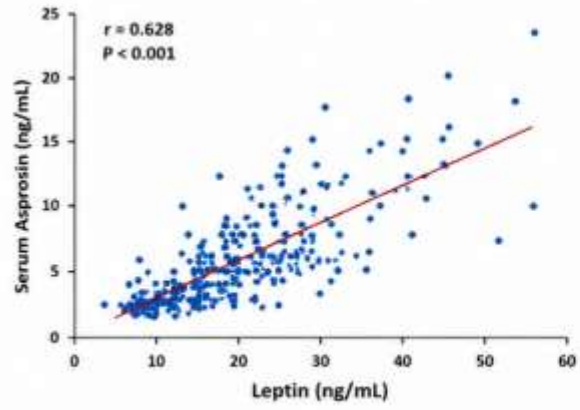

Figure 1. Correlation between Serum Asprosin and Leptin

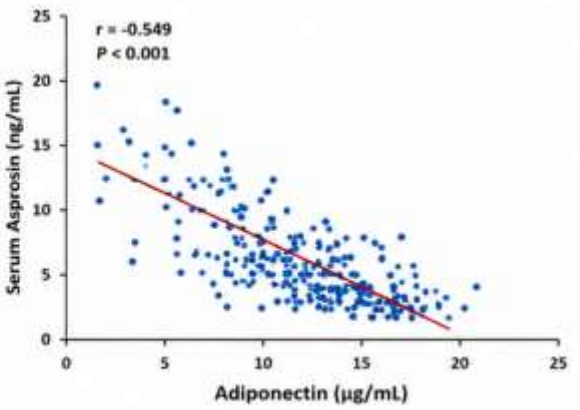

Figure 2. Correlation between Serum Asprosin and Adiponectin

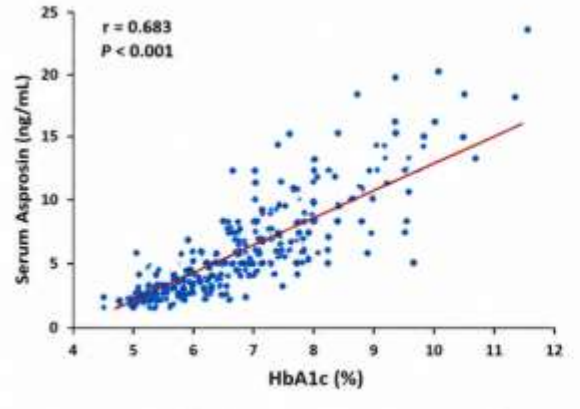

Figure 3. Correlation between Serum Asprosin and HbA1c

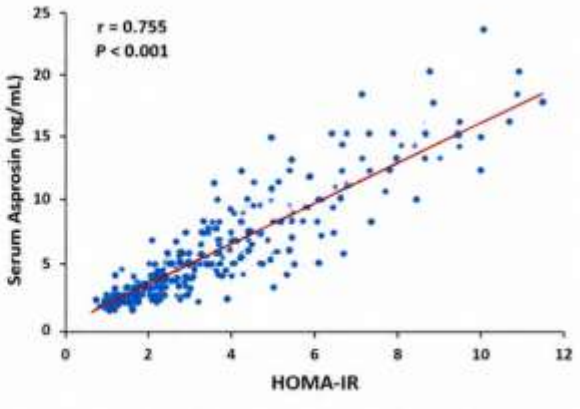

Figure 4. Correlation between Serum Asprosin and HOMA-IR

The multiple linear regression analysis revealed that HOMA-IR, HbA1c, BMI and TyG index were independent predictors of circulating levels of serum asprosin (Fig. 5).

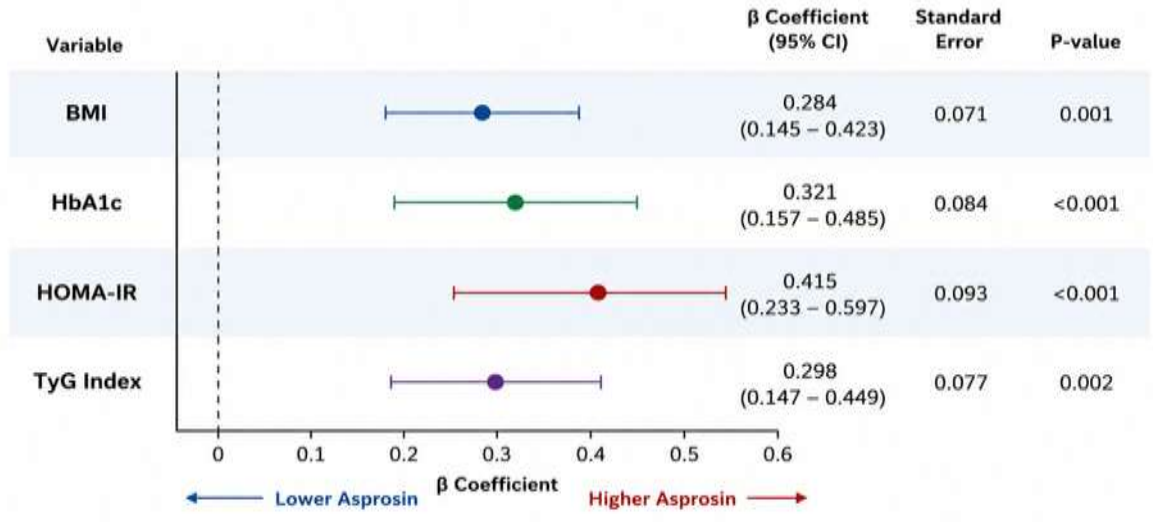

Figure 5. Multiple Linear Regression Analysis for Predictors of Serum Asprosin Levels.

Discussion

Body weight, BMI, waist circumference, waist to hip ratio, and waist to height ratio were significantly higher in the obese participants, especially the obese participants with T2DM compared to healthy participants. The results are consistent with the well established correlation between over-adiposity and metabolic dysfunction. However, higher concentrations of asprosin in the blood of obese people [14,15] might be due to higher fat mass, and asprosin is produced mainly in white adipose tissue.

In diabetic groups, as expected, fasting blood glucose, HbA1c, fasting insulin and HOMA-IR were all significantly elevated, the highest levels being measured in the obese diabetic group. These results suggest a glucose regulation problem and high insulin resistance. Asprosin stimulates glucose production by stimulating cAMP pathway in the liver, so that high serum levels of asprosin may lead to chronic hyperglycemia and glycemic control disorders [16,17].

The first main conclusion of this study was that asprosin levels rose in healthy controls, further up to obese persons, and lastly to obese patients with T2DM. This is similar to the findings from past clinical studies and recent meta-analyses that have shown significantly higher asprosin levels in obesity, metabolic syndrome, and T2DM [18–21]. Overall, these results reinforce the notion that asprosin is a marker of metabolic decline.

Next, correlation analysis revealed positive associations between serum asprosin and obesity parameters such as BMI and waist circumference, indicating that higher levels of adipose tissue are linked with higher levels of asprosin. The same finding was previously reported and it is suggested that asprosin is involved in the metabolic dysfunction associated with obesity [22].

One particularly noteworthy note was the correlation between serum asprosin and insulin resistance parameters such as fasting insulin, HOMA-IR and TyG index. Of these factors, HOMA-IR had the highest correlation with circulating asprosin. Excessive levels of asprosin have been proposed to cause insulin resistance via several pathways, including oxidative stress, endoplasmic reticulum stress and inflammation pathways, in experimental studies [23,24].

The present study also showed remarkable abnormalities in lipid metabolism in obese and diabetic participants. High levels of triglycerides, total cholesterol, LDL-C and VLDL-C were correlated with high levels of serum asprosin, which was correlated positively with triglycerides. These observations are similar to previous studies that correlated elevated asprosin levels to dyslipidemia, and elevated cardiovascular risk [25,26].

Adipokines, obese diabetic patients had significantly higher level of leptin and lower level of adiponectin. Asprosin serum levels were positively associated with leptin and negatively associated with adiponectin suggesting considerable adipose tissue dysfunction. The results indicate that there is a possibility of coexistance of elevated asprosin with impaired adipokine balance, which may lead to insulin resistance and metabolic impairment.

There is a known association between obesity and T2DM with chronic low-grade inflammation. As such, serum asprosin levels were significantly positively correlated with hs-CRP levels. There is some previous experimental data that asprosin is capable of triggering inflammatory cytokines and inflammatory signalling pathways, suggesting its involvement in obesity-associated inflammation [27]

In addition, there was a significant elevation in cardiometabolic risk indices, such as TyG, AIP and LAP, among obese diabetic patients and a positive correlation between these indices and serum asprosin levels. As this study adds to the growing body of evidence suggesting that asprosin may have a role as a combined measure of cardiometabolic dysfunction, these indices are deemed to be good markers of insulin resistance and cardiovascular risk.

Liver and kidney function parameters were not significantly elevated but elevated liver enzymes were noted in diabetic patients with obesity. The positive association with serum asprosin could be indicative of underlying hepatic steatosis and metabolic liver dysfunction, as previous reports have shown that elevated circulating asprosin is associated with metabolic liver dysfunction [28,29].

Overall, serum asprosin was found to have positive correlations with obesity, hyperglycemia, insulin resistance, dyslipidemia, inflammatory markers, and cardiometabolic risk indices, and negative correlations with adiponectin. Multiple linear regression also showed that BMI, HbA1c, HOMA-IR and

TyG index were independent factors associated with circulating asprosin concentrations, confirming previous clinical findings [30]. These results indicate that asprosin is related to metabolic dysfunction and could be a promising biomarker of the relationship between obesity, insulin resistance and T2DM.

Conclusion

In the present study, a significant increase in the serum levels of asprosin was found in obese individuals and T2DM patients, and the highest levels were seen in obese diabetic patients. Higher levels of asprosin were found to be related to higher adiposity levels, glycemic control, insulin resistance, dyslipidaemia, peripheral inflammation and unfavorable cardiometabolic risk phenotypes. HbA1c, HOMA-IR, triglycerides, leptin, hs-CRP, and TyG index had significant positive correlations, while adiponectin had a significant inverse correlation.

Furthermore, BMI, HbA1c, HOMA-IR and TyG index were independent predictors of circulating asprosin, suggesting asprosin as a new potential biomarker for metabolic dysfunction. Additional prospective and mechanistic studies are justified to further understand the causation of asprosin and to assess its therapeutic value to obesity and T2DM.

REFERENCES

[1] R. Ruze, T. Liu, X. Zou, J. Song, Y. Chen, R. Xu, X. Yin, and Q. Xu, "Obesity and type 2 diabetes mellitus: Connections in epidemiology, pathogenesis, and treatments," *Front. Endocrinol.*, vol. 14, p. 1161521, Feb. 2023, doi: 10.3389/fendo.2023.1161521.

[2] P. Chandrasekaran and R. Weiskirchen, "The role of obesity in type 2 diabetes mellitus—An overview," *Int. J. Mol. Sci.*, vol. 25, no. 3, p. 1882, Feb. 2024, doi: 10.3390/ijms25031882.

[3] L. Luo and M. Liu, "Adipose tissue in control of metabolism," *J. Endocrinol.*, vol. 231, no. 3, pp. R77–R99, Dec. 2016, doi: 10.1530/JOE-16-0211.

[4] V. J. Clemente-Suárez, L. Redondo-Flórez, A. I. Beltrán-Velasco, A. Martín-Rodríguez, I. Martínez-Guardado, E. Navarro-Jiménez, C. C. Laborde-Cárdenas, and J. F. Tornero-Aguilera, "The role of adipokines in health and disease," *Biomedicines*, vol. 11, no. 5, p. 1290, Apr. 2023, doi: 10.3390/biomedicines11051290.

[5] E. A. Al-Suhaimi and A. Shehzad, "Leptin, resistin and visfatin: The missing link between endocrine metabolic disorders and immunity," *Eur. J. Med. Res.*, vol. 18, no. 1, p. 12, Apr. 2013, doi: 10.1186/2047-783X-18-12.

[6] A. I. Mazur-Bialy, "Asprosin—a fasting-induced, glucogenic, and orexigenic adipokine as a new promising player: Will it be a new factor in the treatment of obesity, diabetes, or infertility? A review of the literature," *Nutrients*, vol. 13, no. 2, p. 620, Feb. 2021, doi: 10.3390/nu13020620.

[7] M. Farrag *et al.*, "Asprosin in health and disease, a new glucose sensor with central and peripheral metabolic effects," *Front. Endocrinol.*, vol. 13, p. 1101091, Dec. 2022, doi: 10.3389/fendo.2022.1101091.

[8] N. Gozel and F. Kilinc, "Investigation of plasma asprosin and saliva levels in newly diagnosed type 2 diabetes mellitus patients treated with metformin," *Endokrynol. Pol.*, vol. 72, no. 1, pp. 37–43, 2021, doi: 10.5603/EP.a2020.0059.

[9] K. Shabir *et al.*, "Asprosin exerts pro-inflammatory effects in THP-1 macrophages mediated via the Toll-like receptor 4 (TLR4) pathway," *Int. J. Mol. Sci.*, vol. 24, no. 1, p. 227, Dec. 2022, doi: 10.3390/ijms24010227.

[10] M. Kościuszko, A. Buczyńska, E. Duraj, A. Adamska, K. Siewko, A. J. Krętowski, and A. Popławska-Kita, "Associations between asprosin, insulin resistance, and oxidative stress in adults with obesity," *Sci. Rep.*, vol. 15, no. 1, p. 40849, 2025, doi: 10.1038/s41598-025-24648-7.

[11] M. E. Cerf, "Beta cell dysfunction and insulin resistance," *Front. Endocrinol.*, vol. 4, p. 37, Mar.

2013, doi: 10.3389/fendo.2013.00037.

[12] Z. S. Y. Hammo, A. J. Mansoor, and M. D. Mustafa, "The role of obesity in the development of type 2 diabetes," *Dijlah J. Med. Sci.*, vol. 2, no. 1, 2025, doi: 10.65204/DJMS-TRO-DT.

[13] L. Ma *et al.*, "Association analysis between serum asprosin and metabolic characteristics, complications in type 2 diabetic patients with different durations," *J. Diabetes Investig.*, vol. 15, no. 12, pp. 1781–1787, Dec. 2024, doi: 10.1111/jdi.14313.

[14] Q. X. Yi, L. N. Zhu, J. Ma, X. J. Yu, L. Liu, and J. Shen, "Use of anthropometric measures of obesity to predict diabetic retinopathy in patients with type 2 diabetes in China," *Diabetes Metab. Syndr. Obes.*, vol. 14, pp. 4089–4095, Sep. 2021, doi: 10.2147/DMSO.S321030.

[15] Y. Zheng, S. H. Ley, and F. B. Hu, "Global etiology and epidemiology of type 2 diabetes mellitus and its complications," *Nat. Rev. Endocrinol.*, vol. 14, no. 2, pp. 88–98, Feb. 2018, doi: 10.1038/nrendo.2017.151.

[16] B. Açmaz, S. Bahçebaşı, E. Aydın, Ş. Yıldız, and İ. Biçer, "Comparison of HbA1c and OGTT results in obese and morbidly obese patients: A single-center evaluation from an obesity clinic," *J. Diabetes Res.*, vol. 2025, p. 9933219, 2025, doi: 10.1155/jdr/9933219.

[17] A. Hruby and F. B. Hu, "The epidemiology of obesity: A big picture," *PharmacoEconomics*, vol. 33, no. 7, pp. 673–689, Jul. 2015, doi: 10.1007/s40273-014-0243-x.

[18] N. Ouchi, J. L. Parker, J. J. Lugus, and K. Walsh, "Adipokines in inflammation and metabolic disease," *Nat. Rev. Immunol.*, vol. 11, no. 2, pp. 85–97, Feb. 2011, doi: 10.1038/nri2921.

[19] D. P. Guh, W. Zhang, N. Bansback, Z. Amarsi, C. L. Birmingham, and A. H. Anis, "The incidence of co-morbidities related to obesity and overweight: A systematic review and meta-analysis," *BMC Public Health*, vol. 9, p. 88, Mar. 2009, doi: 10.1186/1471-2458-9-88.

[20] I. Mishra *et al.*, "Asprosin-neutralizing antibodies as a treatment for metabolic syndrome," *eLife*, vol. 10, p. e63784, Apr. 2021, doi: 10.7554/eLife.63784.

[21] J. R. Ulloque-Badaracco *et al.*, "Asprosin levels in patients with type 2 diabetes mellitus, metabolic syndrome and obesity: A systematic review and meta-analysis," *Diabetes Metab. Syndr. Clin. Res. Rev.*, vol. 18, no. 7, p. 103095, Jul. 2024, doi: 10.1016/j.dsx.2024.103095.

[22] S. Mottillo, K. B. Filion, J. Genest, *et al.*, "The metabolic syndrome and cardiovascular risk," *J. Am. Coll. Cardiol.*, vol. 56, no. 14, pp. 1113–1132, Sep. 2010, doi: 10.1016/j.jacc.2010.05.034.

[23] T. V. Kirichenko, Y. V. Markina, A. I. Bogatyreva, T. V. Tolstik, Y. R. Varaeva, and A. V. Starodubova, "The role of adipokines in inflammatory mechanisms of obesity," *Int. J. Mol. Sci.*, vol. 23, no. 23, p. 14982, Dec. 2022, doi: 10.3390/ijms232314982.

[24] T. W. Jung *et al.*, "Asprosin attenuates insulin signaling pathway through PKCδ-activated ER stress and inflammation in skeletal muscle," *J. Cell. Physiol.*, vol. 234, no. 11, pp. 20888–20899, Nov. 2019, doi: 10.1002/jcp.28694.

[25] H. A. Silitonga, J. M. Siahaan, and E. J. Anto, "Correlation between obesity and lipid profile in type 2 diabetes mellitus patients at the endocrine and metabolic polyclinic in General Hospital Pirngadi Medan," *Open Access Maced. J. Med. Sci.*, vol. 7, no. 8, pp. 1309–1313, Apr. 2019, doi: 10.3889/oamjms.2019.312.

[26] American Diabetes Association, "Diagnosis and classification of diabetes mellitus," *Diabetes Care*, vol. 37, no. Suppl. 1, pp. S81–S90, Jan. 2014, doi: 10.2337/dc14-S081.

[27] J. Klein, N. Perwitz, D. Kraus, and M. Fasshauer, "Adipose tissue as source and target for novel therapies," *Trends Endocrinol. Metab.*, vol. 17, no. 1, pp. 26–32, Jan. 2006, doi: 10.1016/j.tem.2005.11.008.

[28] J. Stanimirović *et al.*, "Role of C-reactive protein in diabetic inflammation," *Mediators Inflamm.*, vol. 2022, p. 3706508, 2022, doi: 10.1155/2022/3706508.

[29] D. Zheng *et al.*, "The association of liver enzymes with diabetes mellitus risk in different obesity subgroups: A population-based study," *Front. Endocrinol.*, vol. 13, p. 961762, Aug. 2022, doi:

10.3389/fendo.2022.961762.

[30] M. Mirr, A. Braszak-Cymerman, A. Ludziejewska, M. Kręgielska-Narożna, P. Bogdański, W. Bryl, and M. Owecki, "Serum asprosin correlates with indirect insulin resistance indices," *Biomedicines*, vol. 11, no. 6, p. 1568, May 2023, doi: 10.3390/biomedicines11061568.