Evaluation of serum Asprosin in patients with Prediabetes and newly diagnosed type 2 Diabetes Mellitus

Salam Adil Raheem[1]*, Mohammed Imran Hamzah[2], Mahmood Shakir Khudhair[3]

[1]Department of Chemistry and Biochemistry, College of Medicine, Al-Nahrain University, Baghdad, Iraq

[2]Department of Chemistry and Biochemistry, College of Medicine, Al-Nahrain University, Baghdad, Iraq

3Internal Medicine and Endocrinology, College of Medicine, Al-Nahrain University, Baghdad, Iraq

Email: SalamAR11@yahoo.com

Background Mammal white adipose tissue secretes asprosin, a protein hormone that causes the liver to release glucose. Asprosin, a protein produced by the gene FBN1, is released from the C-terminus of profibrillin via targeted proteolysis. Asprosin stimulates a cyclic adenosine monophosphate (cAMP)-dependent mechanism that results in fast glucose release in the liver. Objective This study's aim was to provide an estimation of serum concentrations of asprosin in prediabetes, newly diagnosed diabetes mellitus type 2 patients, & healthy controls by ELISA technique. Methods Case-control research carried out at the Teaching Hospital of the Medical City of Baghdad and the AL-Imamain AL-Kadhmain Medical City between March 2021 and March 2022. It included 40 diabetics with type 2 recently diagnosed, forty people who have prediabetes and 40 healthy control persons. Patients with diabetes, prediabetes, and healthy controls each had around 5 ml of blood drawn from a vein. After 15 minutes at room temperature, the 5 mL of blood were analyzed. Centrifugation of the sera about 3000 rpm for ten minutes was used to separate the sera after coagulation. Sera were put in -20 C storage until they could be tested with ELISA kits. Results When compared to patients with prediabetes and controls, individuals with newly diagnosed diabetes type 2 had serum asprosin concentrations that were considerably greater. Conclusion When compared to prediabetic patients and the control group, Type 2 diabetic mellitus newly diagnosed patients had serum asprosin levels that were significantly higher, with a high sensitivity and specificity of the cutoff value for the biomarker.

List of abbreviations

ADA (American Diabetes Association)
ANOVA (Analysis of variance)
BMI (Body mass index)
cAMP (Cyclic adenosine monophosphate)
ELISA (Enzyme linked immunosorbent assay)
FBG (Fasting blood glucose)
FBN1 (Fibrillin1 gene)
HbA1c (Hemoglobin 1 c)
HOMA-IR (Homeostatic model assessment of insulin resistance)
mRNA (Messenger RNA)
OR (Odds ratio)
RPM (Round per minute)
SD (Standard deviation)
SPSS (Statistical Package for the Social Sciences)
T2DM (Type 2 diabetes mellitus)
WAT (White adipose tissue)

1. Introduction

Hyperglycaemia, caused by abnormalities in insulin secretion, insulin transport, or both, is a hallmark of the metabolic diseases that make up the diabetes spectrum. Other organs suffering permanent harm, becoming dysfunctional, or failing, Insulin shortage can be caused by the immune system attacking and killing off pancreatic cells, or it can be caused by abnormalities that make the body resistant to the effects of insulin including the eyes, kidneys, nerves, heart, and blood arteries, are related with the persistent hyperglycaemia of diabetes. Several underlying pathogenic mechanisms. Insulin shortage can be caused by the immune system attacking and killing off pancreatic cells, or it can be caused by abnormalities that make the body resistant to the effects of insulin. Diabetes' irregularities in glucose,

doi.org/10.31838/hiv23.02.36 Received: 12.10.22, Revised: 16.11.22, Accepted: 12.12.22

fat, Insulin shortage can be caused by the immune system attacking and killing off pancreatic cells, or it can be caused by abnormalities that make the body resistant to the effects of insulin and protein metabolism are founded on insulin's inability to reach the correct tissues. Impaired insulin secretion and/or reduced tissue reactions to insulin at different stages along the complex pathways of hormone activation are two possible explanations of lack of insulin action. There is frequently confusion over which aberration, if each abnormality by itself, is the major cause of the hyperglycaemia when both impaired Insulin shortage can be caused by the immune system attacking and killing off pancreatic cells, or it can be caused by abnormalities that make the body resistant to the effects of insulin shortage can be caused by the immune system attacking and killing off pancreatic cells, or it can be caused by abnormalities that make the body resistant to the effects of insulin (1). White adipose tissue (WAT) in mammals contains a unique hormone called asprosin, which was recently identified (2). After a fast, it is secreted from WAT to maintain daily meal thermal energy standards (3). It is also now recognized as a glucogenic hormone that controls glucose homeostasis (4). Additionally, the Fibrillin1 gene (Fbn1), which is the precursor to the recently discovered glucogenic hormone, encodes asprosin (26-27). After examining Fbn1 mRNA expression, prior studies verified the highest levels of expression in WAT from humans and/or mice, demonstrating the crucial function of Fbn1 in adipogenesis. (5). Thus, type 2 diabetic mellitus (T2DM) and obese are thought to have particularly sensitive biomarkers for asprosin. (6).

2. Methods

Between the years of 2021 and 2022, case-control research was done. This study involved 40 participants with newly diagnosed type 2 diabetes, 40 participants with prediabetes, and 40 healthy controls. Samples were collected between March 2021 and March 2022 from the Medical City of Baghdad Teaching Hospital and the AL-Imamain AL-Kadhmain Medical City. According to the standards established by the American Diabetes Association (ADA) in 2020, all samples were collected from participants who were fasting and examined for indicators of pre-diabetes and type 2 diabetes mellitus.

1. The participants in this study fall into three categories:
2. 40 individuals with recently diagnosed diabetes mellitus type 2 were included in the diabetic group.
3. Prediabetic group: Included 40 persons with prediabetes.
4. Control group: Included 40 healthy control persons.
5. The patients' ages range from (35-70).

Serum asprosin measurements

A commercial enzyme-linked immunosorbent test was used to measure the levels of serum endocan. The detection threshold was 0.23 ng/ml.

3. Statistical Analysis

In order to determine whether or not there were significant differences in the study's continuous variables between groups, an analysis of variance (ANOVA) was performed, with the p-value adjusted using the Tukey HSD method to account for the potential impact of multiple comparisons on the family-wise error rate. The Statistical Package for the Social Sciences (SPSS, Version 21 for Windows; SPSS, Inc., Chicago, IL, USA) and GraphPad prism Software, United States, were used for data analysis and graphical presentations, respectively, with a significance level of 0.05.

4. Results

In comparison to prediabetic and healthy healthy controls, the average of human asprosin was considerably greater in recently diagnosed patients with type 2 diabetes (Table 1).

Table 1. Asprosin levels in diabetic subgroups and control.

Variable	Control Mean± SD	Diabetic Mean± SD	Prediabetic Mean± SD	F	p
Asprosin	14.023± 2.82	27.089± 5.293	15.913± 5.12	96.18	< .001

5. Discussion

Asprosin blood concentrations were found to be considerably higher in T2DM patients in a case-control study (4.18 [IQR: 4.4] vs. 3.5 [IQR: 1.85], $P < 0.001$) than in healthy controls. In healthy participants, the concentrations of asprosin substantially linked with BMI, fasting blood glucose, hemoglobin A1c, and the homeostatic model assessment of insulin resistance (HOMA-IR). Asprosin serum concentrations increased the odds ratio (OR) for patients with type 2 diabetes by about 1.547 (95% CI 1.293-1.850, $P < 0.001$) versus the control group in the fully adjusted model. Asprosin was independently correlated with FBG and HOMA-IR in T2DM, according to multiple stepwise regression analysis (7).

6. Conclusion

Asprosin levels were significantly greater in patients with type 2 diabetes who had recently received their diagnosis than they were in prediabetic patients or healthy controls. It performs effectively as a biomarker and has good specificity and sensitivity at its diagnostic threshold for the purpose of diagnosing people with type 2 diabetes.

References

American Diabetes Association. Diagnosis and classification of diabetes mellitus. Diabetes care.

2010 Jan 1;33(Supplement_1): S62-9.
Wang Y, Qu H, Xiong X, Qiu Y, Liao Y, Chen Y, Zheng Y, Zheng H. Plasma asprosin concentrations are increased in individuals with glucose dysregulation and correlated with insulin resistance and first-phase insulin secretion. Mediators of inflammation. 2018 Oct;2018.
Zhang L, Chen C, Zhou N, Fu Y, Cheng X. Circulating asprosin concentrations are increased in type 2 diabetes mellitus and independently associated with fasting glucose and triglyceride. Clinica chimica acta. 2019 Feb 1; 489:183-8.
Romere C, Duerrschmid C, Bournat J, Constable P, Jain M, Xia F, Saha PK, Del Solar M, Zhu B, York B, Sarkar P. Asprosin, a fasting-induced glucogenic protein hormone. Cell. 2016 Apr 21;165(3):566-79.
Davis MR, Arner E, Duffy CR, De Sousa PA, Dahlman I, Arner P, Summers KM. Datasets of genes coexpressed with FBN1 in mouse adipose tissue and during human adipogenesis. Data in brief. 2016 Sep 1; 8:851-7.
Schumann U, Qiu S, Enders K, Bosnyák E, Laszlo R, Machus K, Trájer E, Jaganathan S, Zügel M, Steinacker JM. Asprosin, A Newly Identified Fasting-Induced Hormone Is Not Elevated in Obesity and Is Insensitive to Acute Exercise: 3592 Board# 39 June 3 8: 00 AM-9: 30 AM. Medicine & science in sports & exercise. 2017 May 1;49(5S):1023.
Naiemian S, Naeemipour M, Zarei M, Lari Najafi M, Gohari A, Behroozikhah MR, Heydari H, Miri M. Serum concentration of asprosin in new-onset type 2 diabetes. Diabetology & Metabolic Syndrome. 2020 Dec;12(1):1-8.