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Afamin level and their correlation with insulin resistance in patients with type 2 diabetes

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Abstract

Afamin can be defined as a bioactive protein that is produced by the liver. Afamin levels are high in obesity and metabolic syndrome, and they have a strong correlation with metabolic syndrome components. A total of 39 patients who have diabetes mellitus type 2 (T2DM) who were either hospitalized to the department or who attended the diabetic clinic participated in the current work. In the presented research, specific tests were performed, including glycated haemoglobin, Fasting Blood Sugar (FBS), magnesium, creatinine, and calcium Homeostatic Model for Insulin Resistance (HOMA-IR). As opposed to the control group, type 2 diabetic patients had significantly higher levels of insulin, HbA1c, FBS, afamin, HOMA-IR, cortisol, calcium, triglycerides (TG), Total Cholesterol (TC), VERY Low-Density Lipoprotein (VLDLc), Low Density Lipoprotein (LDLc), creatinine, and urea. In contrast, the diabetic group's High-Density Lipoprotein (HDL-c) and magnesium levels are significantly lower when compared to those of the control group.

Keywords: Afamin, Type 2 D.M, Insulin resistance, Lipid profile and cortisol

Introduction

Persistent hyperglycemia is a Diabetes Mellitus (DM) hallmark, a chronic metabolic disorder. Insulin resistance (IR), impaired insulin secretion, or the two could be to blame. In the year 2015, a total of 415 million persons aged between 20 and 79 had diabetes mellitus, based on the International Diabetes Federation (IDF). Given that this number is expected to increase to an additional 200 million by the year 2040, DM is proving to be a global public health concern (Zheng *etal*., 2018). Chronic hyperglycemia as well as other metabolic aberrations in patients experiencing DM might harm organ systems, resulting in life-threatening and disabling health complications, most notably diabetes (diabetes, retinopathy, and nephropathy) and cardiovascular complications (a 2- to 4-fold increased risk of cardiovascular diseases (CVD)). The diagnosis, pathogenesis, management principles of diabetes, and clinical manifestation are briefly discussed in the presented work. The term "insulin resistance" (IR) describes a reduction in the metabolic response regarding insulin-responsive cells to the insulin or, on a systemic level, an impaired metabolic response to circulating insulin that is caused by low levels of blood glucose. IR, or insulin deficient conditions, can be divided to 3 main groups, which are: (1) reduced secretion of insulin through β-cells; (2) insulin antagonists in plasma, stemming from the non-hormonal bodies or counter-regulatory hormones disrupting insulin receptors or signaling; and (3) impaired response of insulin in the target tissues (Pearson et al., 2016).

Human afamin can be defined as a glycoprotein with apparent molecular weight of 87kDa and an amino acid sequence similarity to albumin of 55% (34% identity). With 4 or 5 possible N-glycosylation sites, Human afamin represents a previously identified albumin gene family member. A unique binding protein for vitamin E was identified as human plasma afamin. Afamin was found to have no more than 18 binding sites for vitamin E per molecule (Erol et al., 2021). It was suggested that Afamin is a very good clinically significant indicator of metabolic disease in the general population. Afamin plasma concentrations are definitely linked to the occurrence and progression of metabolic syndrome, according to meta-analyses of large population studies. Afamin's substantial correlation with IR and its potential as an independent, powerful predictor of T2D were verified by a pooled analysis involving over 20,000 participants. Independent of the other main metabolic risk variables, afamin can predict a higher T2D incidence and raises the probability of metabolic

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syndrome by 79% for every 10 mg/L rise in afamin. In patients who have polycystic ovarian syndrome, elevated afamin concentrations were positively correlated with IR and were found to be an early biomarker for pre-eclampsia and gestational diabetes. Gender, age, prandial state, menstrual cycle, and circadian rhythms have no effect on the circulating levels of afamin, which could be tested in plasmaor serum. All things considered, afamin is a great potential biomarker for elevated metabolic disease risk (Timea et al., 2021; Kanval, 2025).

Materials and methods

Design of the study

This research has been conducted at the University of Baghdad's Department of Biotechnology, College of Science. A total of 39 T2DM patients who have been either hospitalized to the department or who visited the diabetic clinic ranged in age from 19 to 42. The presented work included certain tests like HbA1c, FBS, urea, serum afamin, BMI, creatinine, and the HOMA-IR. Samples have been gathered. The samples were analyzed at University of Baghdad's Chemical Laboratory, College of Science, and Department of Biotechnology. From the subjects in the morning, 16 hours of fasting were followed by the collection of blood samples. The individuals were given a 5 ml blood sample with the use of a needle and syringe.

Serum total cholesterol (T. chol) determination

The commercially available (bio-Merieux) kit has been utilized in order to assess T. chol concentration with using enzymatic approach (Richmonds, 1973). At 500nm, T. chol value is determined spectrophotometrically.

Determining serum High Density Lipoprotein (HDL-c)

Bio-Merieux kit has been utilized for the measuring levels of the HDL-c by using enzymatic technique (Burstein, 1970). The concept behind this method is to add phosphotungestic acid with the existence of the ions of magnesium, which precipitates lipoproteins as well as chylomicrons of low-density lipoprotein and VLDL. Following centrifuging, HDL was included in the supernatant, which could contain cholesterol and phospholipids. HDL was measured at 500 nm using spectrophotometry.

Serum triglycerides (TG) determination

Using Bio-Merieux kit as well as enzymatic approach of Prencipel & Fossati (1982), the total serum TG concentration had been determined. At 500nm, TG total serum concentration was established.

Determining serum VLDL-c

Friedewald et al. (1972) used the classical equation to specify the VLDL. VLDLc (mg. dl^{-1}) = 0.2xTG (mg/dl).

Serum LDL-c determination

Friedewald's equation has been used in order to specify the serum LDL. LDL-c = T.Chol. - (VLDLc + HDLc).

FBS determination

Using glucose oxidase GOD PAP (Kit) (Liquid) GL2624, FBG is calculated enzymatically.

Serum insulin determination

The AESKULISA ELISA kit from Germany is used to measure serum insulin. In microplates that have been coated with the particular antigen, serum samples that have been diluted 1:101 are incubated. In a case when the specimen contains the antibodies of the patient, they bind to antigen. In the subsequent step, the unbound fraction is rinsed out. The antigen-antibody complex of samples in micro-plates is reacted after that by anti-human immunoglobulins conjugated to horseradish peroxidase (i.e., conjugate), which is then incubated. This is followed by washing off the unbound conjugate. An enzymatic colorimetric (i.e., blue) reaction is triggered by the addition of TMB-substrate gene and is stopped by diluted acid (color changes to yellow). The conjugate amount that is linked to antigen-antibody complex is responsible for how intensely the chromogen produces color, and this is proportionate to initial concentration of corresponding antibodies in the patient sample.

Determination of HOMA-IR

The next formula has been utilized to determine

HOMA-IR: (fasting glucose (mg/dl) × fasting insulin) / 405 or (fasting glucose (mmol/L) × fasting insulin) /22.5.

Determination of glycated hemoglobin (HbA1c)

The quantitative colorimetric measurement regarding glycohemoglobin in whole blood (StanbioGlycohemoglobin – pre-fil-procedure No. P350) was used to quantify (HbA1c).

Serum creatinine determination

Commercially available (BIOLABO) kits were utilized to colorimetrically quantify the creatinine levels in serum. The kidneys eliminate creatinine, which is released throughout creatine phosphate metabolism. Alkaline picrate and creatinine are combined in a 1:1 ratio to generate the colored creatinine picrate complex, which has ionic limits. The rate at which the colored complex forms is proportional to the creatinine concentration.

Serum urea level determination

According to Scott and Fawcett's (1960) colorimetric method, Serum Urea was quantified, utilizing a Randox kit.

Determination of serum afamin level

Afamin Human ELISA, cat. number: RD194428100R, BioVendor, Asheville, NC, U.S., was used to assess serum afamin concentrations in accordance with the manufacturer's instructions.

Determination of serum calcium

According to Tietz, serum calcium has been measured color metrically, using a Biolabo kit.

Determination of serum cortisol

The competitive immune detection approach of the ichroma kit was used to measure Serum Cortisol.

Determination of serum magnesium

The Chem CHEK (AGAPPE) colorimetric approach has been used to determine the serum magnesium levels (Young, 2001).

Statistical analyses

The statistical analysis has been carried out with the use of SPSS for Windows, v. 22. Mean ± Standard Deviation (SD) has been utilized in order to depict the data. To find out if the studied parameters have been following gaussian distribution, Shapiro-Wilk normality test has been employed. Following ANOVA testing, the Bonfferoni Post Hoc test has been used for multiple comparisons. Pearson's correlation analysis has been used in order to examine the association levels. A significance level of $p<0.05$ has been established (Mitchell and Glover, 2008).

Results and discussion

Effects of afamin and some of the biochemical parameters

Table 1 indicates that the diabetic group differs significantly in a few biochemical parameters. When comparing the diabetic group to control group, there is a significant rise (P≤ 0.05) in Afamin, cortisol, and calcium (Ca). In contrast, diabetic individuals had lower magnesium (Mg) levels (P≤ 0.05) than healthy controls.

Table -1-Effect of afamin level and a few biochemical parameters in study groups

Parameters	Control subjects	Diabetic subjects	P value
Afamin (µg/mL)	42.9 ± 3.3	63.3 ± 6.6	≤ 0.05
Mg (mg/dl)	2.10 ± 0.30	1.35 ± 0.4	≤ 0.05
Ca (mmol/L)	1.87 ± 0.3	3.7 ± 0.7	≤ 0.05
Cortisol (ng/ml)	7.8 ± 1.2	11.7 ± 2.1	≤ 0.05

As far as we are aware, this present data on higher afamin levels in diabetic patients. Comparing diabetic patients to control patients, afamin levels have been greater. This increase is comparable to what was seen in earlier studies comparing diabetes to healthy people (Kollerits et al., 2017). This shows that afamin could have a role in the early onset of IR. Afamin can be specified as a polysaccharide-protein primarily released by liver and has a physiologically significant function as a carrier of vitamin E in plasma and other bodily fluids. According to studies, hyperglycemia, obesity, IR, and metabolic syndrome are all strongly associated with higher plasma afamin

levels (Zafar et al., 2018). According to this study, patients with T2DM who have low levels of serum Mg are more likely to experience poor glycemic control. The prevalence of DM is rising quickly, making it a public health concern. One necessary mineral is Mg. Hypomagnesemia is frequently linked to T2DM (Ketteler and Jahnen-Dechent, 2012; Dasgupta et al., 2012). The association between dietary magnesium consumption and risks of getting diabetes was assessed in a cohort study that included 17.592 people without diabetes or any other chronic diseases. After five years of followup, 459 diabetes cases have been found in this study, and it has been determined that dietary magnesium intake decreased the chance of developing diabetes (Kirii et al., 2010). A total of 8.587 incidences of diabetes has been seen following 14 years of follow-up in a study that involved 75.512 people in Hawaii who were between the ages of 45 and 75. High Mg levels lower the risk of diabetes, according to the study's findings (Kostov, 2019). In a different study of 189 T2DM patients, the hypomagnesemic group (n = 64) had significantly higher fasting glucose as well as HbA1c levels compared to the normomagnesemic group (n = 125) (Misirlioğlu et al., 2020).

In intracellular regions as well as membrane channels, Mg acts as a Ca antagonist. According to Tengholm and Idevall-Hagren (2020), low Mg is linked to excessive Ca. According to one study, patients with diabetes and chronic kidney disease (CKD) had increased rates of osteoporosis and hypomagnesemia (Hu et al., 2021). Intake of Mg and potassium was found to be inversely correlated with HbA1c in a study involving 102 patients with T2DM (Brandão-Lima et al., 2018). In their study of 138 T1DM patients, Galli-Tsinopoulou et al. (2014) discovered that the group with poor glycemic control had low Mg levels. Additionally, they have compared magnesium by dividing it to 4 quarts and discovered that, similar to our study's findings, HbA1c level dropped as the patients' Mg levels rose (Galli-Tsinopoulou et al., 2014). Our investigation revealed that the group of diabetic patients had much higher calcium levels and significantly lower Mg levels, which is consistent with the information in the literature. Increased hepatic gluconeogenesis as well as glycogenolysis are linked to elevated cortisol, which in turn causes hyperglycemia. The purpose of glucocorticoid cortisol is to maintain an increased blood glucose level. Yet, because it contributes to the maintenance of hyperglycemia, its role in DM could be unfavorable. According to Mohanad (2016), this result suggests that measuring this hormone in diabetics is necessary for control and monitoring. Through its effects on glucose transporters in peripheral tissues including skeletal muscle and fat, cortisol modifies blood glucose levels. (Mohanad, 2016; Jam et al., 2025) Cortisol was connected to obesity. It increases the number of the adipocytes in visceral depots, promotes appetite, and ultimately leads to obesity by stimulating the production of hepatic triglycerides (Mohanad, 2016). Additionally, it causes IR, most likely due to the opposite impact of insulin (Mohanad, 2016). According to a study, eating around lunchtime increases the release of stress hormone cortisol (Maha et al., 2023).

Effect of glycated hemoglobin and IR in patients with T2D

Differences in IR among the group of diabetic patients are significant (Table 2). There is an increase in significance P ≤ 0.05 in the insulin (13.1 ± 1.4), FBS (243.86 ± 11.2), HbA1c (7.2 ± 0.9), and HOMA -IR (7.88 ± 0.7) in the diabetic control group in comparison with the controls, (insulin 9.2 ± 1.1), (FBS 87.33 ± 6.7), (HbA1c 5.1 ± 0.5), and (HOMA-IR 1.98 ± 0.3).

Table -2- Effect of HOMA-IR and HbA1c in T2DM patients

Parameter	Control subjects	Diabetic subjects	P_{value}
FBS (mg/dl)	87.33 ± 6.7	243.86 ± 11.2	≤ 0.05
Insulin (µIU/ml)	9.20 ± 1.10	13.1 ± 1.4	≤ 0.05
HOMA-IR	1.98 ± 0.3	7.88 ± 0.7	≤ 0.05
HbA1c %	5.1 ± 0.5	7.2 ± 0.9	≤ 0.05

IR is significantly higher in patients with DM, according to the current research. Our data support the study's findings that type-2 diabetics' sera had considerably greater concentrations ($p < 0.05$) of HbA1c and FBS than controls (Mohanad, 2022). Impaired biologic response of the target tissues to insulin stimulation is known as IR. All tissues that have insulin receptors have the potential to develop IR, however the skeletal muscle, liver, and adipose tissue are the main indicators of IR. As a result of impaired glucose elimination caused by IR, beta-cell

insulin synthesis rises in response, leading to hyperinsulinemia. Given that hyperinsulinemia is a contributing factor to IR, recent research has questioned whether hyperinsulinemia precedes IR. This idea could be useful in clinical settings since it implies that the metabolic dysfunction that is linked to IR could be fueled by the hyperinsulinemia that is brought on by an excessive calorie consumption. Hyperglycemia, dyslipidemia, hypertension, increased inflammatory markers, hyperuricemia, endothelial dysfunction, and a prothrombotic state are some metabolic effects of IR. T2DM, Nonalcoholic Fatty Liver Disease (NAFLD), as well as metabolic syndrome could develop because of insulin resistance. (Seong *etal*., 2019; Brown et al., 2019). Additionally, the results of the present research are consistent with research showing that a ketogenic diet and a reduction in carbohydrates in the diet lower IR in those individuals (Mohanad, 2023).

Effect of lipid profile in type 2 diabetic

In the current research, the lipid profiles of diabetic patients and the control group differ significantly. In comparison with control group (TG 99.8 ± 8.2, TC 103 ± 7.8, LDL-C 54.54 ± 5.4, and VLDL-C 19.96 ± 2.1, respectively), the diabetic group had higher levels of TG (192.6 ± 9.4), TC (243 ± 11.2), LDL-C (171.68 ± 11.6), and VLDL-C (38.52 ± 3.9). In contrast, the HDL-C in diabetic participants decreased significantly (P ≤ 0.05) to 32.8 ± 4.3 from 37.5 ± 2.7 in control subjects.

Table3- Levels of lipid profile in groups of control and diabetic subjects

Parameter	Controls	Diabetic subjects	P_{value}
TC mg/dl	103 ± 7.80	243 ± 11.2	≤ 0.05
TG mg/dl	99.8 ± 8.2	192.6 ± 9.4	≤ 0.05
HDL-C mg/dl	37.5 ± 2.7	32.8 ± 4.3	≤ 0.05
VLDL-C mg/dl	19.96 ± 2.10	38.52 ± 3.9	≤ 0.05
LDL-C mg/dl	54.54 ± 5.4	171.68 ± 11.6	≤ 0.05

The levels of LDL, HDL, TC, and TG are known risk factors for complications from diabetes, including CVD and Coronary Heart Disease (CHD). Our findings support a study conducted by Ahmed et al. in 2023. Lipid abnormalities are common in diabetic people, and T2DM patients are no different. Since IR decreases insulin-dependent muscle-free fatty acid uptake, increases release of fatty acids, and increases hepatic fatty acid synthesis in liver, it was previously associated with the T2DM aberrant lipid profile. High LDL and triacylglycerol levels, as well as low HDL, are common in diabetes. According to the findings of the current study, people with diabetes had higher lipid profile (5). Poor glycemic control in particular often leads to hyperlipidemia, a primary risk factor for CHD and atherosclerosis and a common secondary cause of DM. The great majority of previous research revealed a link between lowering the risk of CVD and the importance of achieving optimal glycemic control. Adult diabetic patients should aim for HbA1c levels below 7% since a single HbA1c reduction reduces the risk of micro-vascular problems by around 37% and myocardial infarction by about 14%. In fact, it is thought that high blood glucose raises the risk of high blood LDL and other atherosclerosis-causing disorders (Ahmed et al., 2023).

Effect of kidney function in T2DM

Significant differences in kidney function between diabetic and control groups are displayed in Table (4). Diabetic group's creatinine (1.8 ± 0.3) and urea (42.4 ± 5.6) have been much higher (p ≤ 0.05) when compared to those of the controls (0.85 ± 0.1 and 21.4 ± 3.2, respectively).

Table 4- Level of creatinine and urea in type 2 diabetic

Parameters	Control subjects	Diabetic subjects	P_{value}
Urea (mg/dl)	21.4 ± 3.2	42.4 ± 5.6	≤ 0.05
Creatinine (mg/dl)	0.85 ± 0.1	1.8 ± 0.3	≤ 0.05

Kidney Disease (KD) might affect one in three persons with T2D, and up to 90% of those with KD and 40% of those with severe KD are not aware that they have the condition. The value of KD screening should be communicated to both professionals and patients with diabetes, as these numbers highlight. Both presentations might be linked to consequences including metabolic disease and CVD, and abnormalities in kidney structure could occur before declines in renal function. The Glomeruli Filtration Rate (GFR) is the most popular and well accepted measure of kidney function (Javier et al., 2023) and is a gene. The current research found that individuals

with T2DM had higher levels of creatinine and urea. Those results are consistent with research that has been carried out by Maha et al. (2023) that explains that a diet that is high in sugars and carbohydrates raises the percentage of creatinine, urea, and lipid profile in such individuals.

Correlation of afamin protein with other parameters in diabetic group

Fig (4-1) shows negative significance regarding afamin protein with IR represented by HOMA-IR.

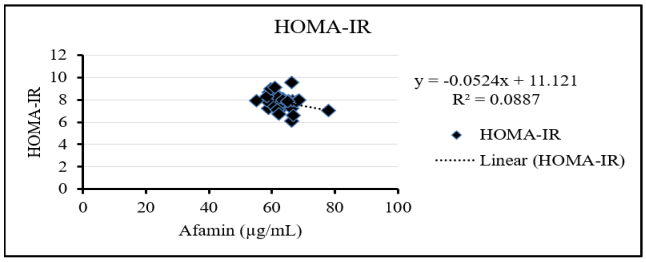

Fig 4-1: The relation of afamin with HOMA-IR in diabetic group

Figure (4-2) positive correlation regarding afamin protein with TC.

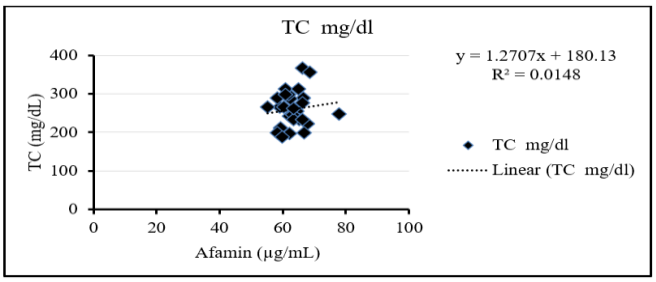

Fig 4-2: The relation of afamin with TC in diabetic group

Figure (4-3) no correlation of afamin with serum urea in diabetic group.

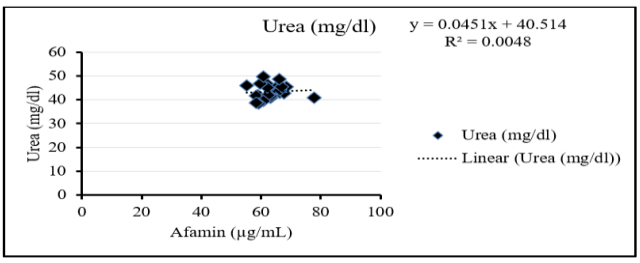

Fig 4-3: The relation of afamin with urea in diabetic group

Conclusion

Afamin can be defined as a liver-produced bioactive protein that is enhanced in the group of diabetic type 2 patients. We also discovered a substantial increase in insulin, FBS, HOMA-IR, HbA1c, TC, Ca, LDL-c, TG, urea, VLDL-c, and creatinine in this study. In contrast, the diabetic group's HDL-c and Mg levels are much lower than those of the control group.

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