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Research Article

Assessment of the Influence of Adiponectin, Nesfatin-1, Insulin Resistance, and Various Biochemical Parameters in Samples of Iraqi Individuals Diagnosed with Type 2 Diabetes Mellitus and Pre-diabetes

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ABSTRACT

Pre-diabetes is characterized by elevated blood sugar levels that are not yet high enough for a Type 2 diabetes diagnosis. Type 2 diabetes results from insufficient insulin production or cellular resistance to insulin. Adiponectin, secreted by adipose tissue, regulates glucose metabolism and energy balance, while nesfatin-1, derived from NUCB2, influences appetite and energy homeostasis. This case-control study at Al-Ramadi Teaching Hospital included 80 participants with diabetes mellitus (DM) or early-stage DM and 40 healthy controls. Demographic data, including age, Body mass index (BMI), and disease duration, were collected. Laboratory tests measured fasting blood glucose (FBG), Glycated hemoglobin (HbA1c%), insulin, adiponectin, and nesfatin-1 levels. Statistical analyses, including ANOVA, Pearson's correlation, and AUC analysis, assessed relationships and diagnostic accuracy. Results showed significantly higher levels of FBG, HbA1c, insulin resistance, and nesfatin-1 in early diabetes and DM type 2 cases compared to healthy controls. Nesfatin-1 concentrations were also notably higher in early diabetes compared to other groups. Adiponectin levels did not significantly differ between early diabetes and the control group but were significantly higher in the DM type 2 group. Both early diabetes and DM type 2 were associated with increased FBG, HbA1c, nesfatin-1, and insulin resistance, while adiponectin levels rose only in DM type 2. These findings suggest adiponectin as a potential biomarker for DM type 2, while nesfatin-1 may aid early diabetes diagnosis.

1. Introduction

Type 2 diabetes mellitus (T2DM) is an increasingly prevalent condition worldwide, characterized by insulin resistance, impaired glucose metabolism, and complex metabolic abnormalities, leading to various complications (Zheng *et al.* 2018). The term "early diabetes" generally refers to blood sugar levels that are consistently higher than normal but not yet high enough for a diabetes diagnosis. This stage is known as pre-diabetes (Duan *et al.* 2021).

Glycated hemoglobin (HbA1c) is a key biomarker for diagnosing and managing diabetes mellitus, as it reflects average blood glucose levels over the past two to three months. It plays a crucial role in assessing long-term glycemic control and predicting diabetes-related complications (Sherwani *et al.* 2016). Additionally, HbA1c is valuable for evaluating treatment effectiveness and guiding therapeutic adjustments to prevent complications (Farhan *et al.* 2018).

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Insulin resistance syndrome refers to a cluster of metabolic abnormalities and associated physiological effects that are more common in insulin-resistant individuals. Due to variations in tissue sensitivity to insulin, the clinical manifestations of insulin resistance syndrome result from the combined effects of excess insulin and variable resistance to its actions (Reaven 2004).

Adipose tissue, or fat cells, is the primary source of the protein hormone adiponectin. Adiponectin is a protein hormone important in controlling the breakdown of fatty acids and glucose levels. Since adiponectin levels and adiposity have an inverse relationship, a rise in fat mass tends to reduce this hormone. Because it reduces inflammation and increases insulin sensitivity, this adipokine—a signaling molecule released by adipose tissue—is frequently seen as advantageous (Nguyen 2020).

In the N terminal region of the protein precursor, Nucliobindin2 (NUCB2) is a polypeptide called nesfatin-1 (Oh *et al.* 2006). Nesfatin stimulates hunger and lipid storage and is generated in pancreatic and hypothalamic cells. The paraventricular nucleus, arcuate nucleus, and tractus solitaries nucleus all contain nesfatin-1, elevate hypothalamic concentration of Nesfatin-1 inhibits appetite and prevents weight gain by inactivating NPY and increasing POM in the CNS's hypothalamus area nesfatin-1 reduces appetite. Additionally, it controls insulin release by pancreatic cells (Garvey *et al.* 2016).

This study aims to investigate the role of adiponectin and nesfatin-1 on early diagnosis of diabetes and diabetes mellitus type 2 patients. In addition, find the role of adiponectin and nesfatin-1 on insulin resistance.

2. Materials and Methods

2.1. Study Design

This study was conducted from November 2023 to March 2024 and included 120 participants, divided into three groups. The patient group consisted of 80 individuals, further categorized into two subgroups: one diagnosed with type 2 diabetes mellitus (T2DM) and the other with early-stage diabetes. The third group comprised 40 healthy controls (both male and female) to assess blood glucose levels, glycated hemoglobin (HbA1c%), adiponectin, insulin, and nesfatin-1. Patients experiencing frequent urination, nausea, or other typical symptoms suggestive of diabetes mellitus sought medical attention at Al-Ramadi Teaching Hospital, Anbar Health Department. Their diagnosis was confirmed through clinical evaluation, medical history, and laboratory tests. The same diagnostic parameters applied to the patient group were also used for the healthy control group. General characteristics recorded in the study included sex, age, body mass index (BMI), and disease duration (measured in weeks). The study received ethical approval from the University of Anbar's Ethical Approval Committee (Project No. 07).

2.2. Procedure

A venous blood sample was collected from the antecubital vein of each participant, including both cases and controls. A total of 5 ml of blood was drawn from each subject. The first portion was placed in an EDTA tube for glycated hemoglobin (HbA1c%) measurement using an Ichroma kit. The remaining blood was collected in a gel tube and left at room temperature for 20 minutes to allow coagulation. Following this, serum was separated by centrifugation at 2000 × g for 10 minutes and then divided into small aliquots. Fasting blood glucose was measured immediately using enzymatic and colorimetric methods.

The remaining serum samples were stored at -20°C until further analysis of adiponectin, insulin, and nesfatin-1 levels. These biomarkers were quantified using enzyme-linked immunosorbent assay (ELISA) kits from ELK Biotechnology (Lot Nos. 46655161, 46664403, and 46672636, respectively).

2.3. Statistical Analysis

The demographic information was presented using a descriptive model and analyzed using the Statistical Package for the Social Sciences (SPSS) version 24. To compare the study groups, a one-way analysis of variance (ANOVA) with Tukey's Honestly Significant Difference (HSD) test was applied. This test was used to identify significant differences among three or more groups. Statistical significance was determined using a confidence interval (CI) of 99.9% and a probability threshold of less than 0.01 ($p<0.01$). Pearson correlation analysis was performed to assess relationships between study parameters. Additionally, the Area Under the Curve (AUC) was calculated to evaluate the performance of classification models. The AUC represents the total area under the Receiver Operating Characteristic (ROC) curve, providing an aggregate measure of classifier performance across all possible thresholds. An AUC of 1.0 indicates a perfect classifier, whereas an AUC of 0.5 suggests no discrimination ability, equivalent to random guessing.

3. Results

Age, sex, BMI, and disease duration for study cases (early DM, DM type 2, and controls) groups were clarified in Table 1 and Figure 1. The result showed that the male participants had 25 samples (62.5%) for early DM, 22 samples (55%) for DM type 2, and 25 samples (62.5%) for the control group. In addition, the female participants for early DM had 15 samples with percent (37.5%), and the DM type 2 was 18 percent (45%), while the control was 15 participants with percent (37.5%). The differences in sex between study groups were non-significant (p-value>0.01).

The duration of the disease is significant and should be noted and studied because it determines whether the disease is in the acute or chronic stage. The study was designed to take the duration of the disease by weeks. Table 1 and Figure 2 showed that 17 participants of the early diabetes samples had a duration of disease with a range from 1 to 2 weeks; also, 9 participants had a duration of disease from 3 to 4 weeks. 9 samples had a duration of disease from 5 to 6 weeks, and finally 5 participants had more than 6 weeks of disease duration. While all the sample groups of diabetes mellitus type 2 have a duration of more than 6 weeks

The mean and standard deviation (SE) with asymptotic significant differences for age and body mass index (BMI) for patients and control groups shown in Table 1 and Figure 3.

The results showed the mean age for the early diabetes group was 51.9 years, and the mean age for the diabetes mellitus type 2 group was 45.5 years. The mean for the control group was 42.4 years, with significant differences and a p-value of 0.001f for both groups (Early DM and DM type 2). The skewness value showed the normal distribution of groups. The skewness of age for the early DM group was -0.694, which indicates that the distribution is near normal (Near 1). The distribution of DM type 2 and control

Table 1. Sociodemographic information of the participant

Characteristics		Early DM	DM	Controls
Age (years)	Mean ± SE	51.9±0.94	45.5±1.12	42.4±1.32
	Range	39-60	32-60	30-60
	Skewness	-0.694	0.442	0.466
	p-value	0.0001	0.0001	-
Sex [n(%)]	Male	25 (62.5%)	22 (55)	25 (62.5%)
	Female	15 (37.5%)	18 (45)	15 (37.5%)
	Total sample size	40 (100%)	40 (100)	40 (100%)
	p-value	0.963 NS	0.334 NS	-
BMI (Kg/m^2)	Mean ± SE	32.52±0.974	33.16±1.12	25.3±0.46
	Range	18.5–49.3	20.1–45.3	19.08–32.7
	p-value	0.0001	0.0001	-
Duration of disease (weeks) [n(%)]	1-2	17 (42.5%)	-	-
	3-4	9 (22.5%)	-	-
	5-6	9 (22.5%)	-	-
	More than 6	5 (12.5%)	40 (100%)	-

Statistically significant if P<0.01; *P values from ANOVA test (Age, BMI)

*P value from Z test for two proportions (sex)

Duration of disease (DM and early DM) for cases without a control group

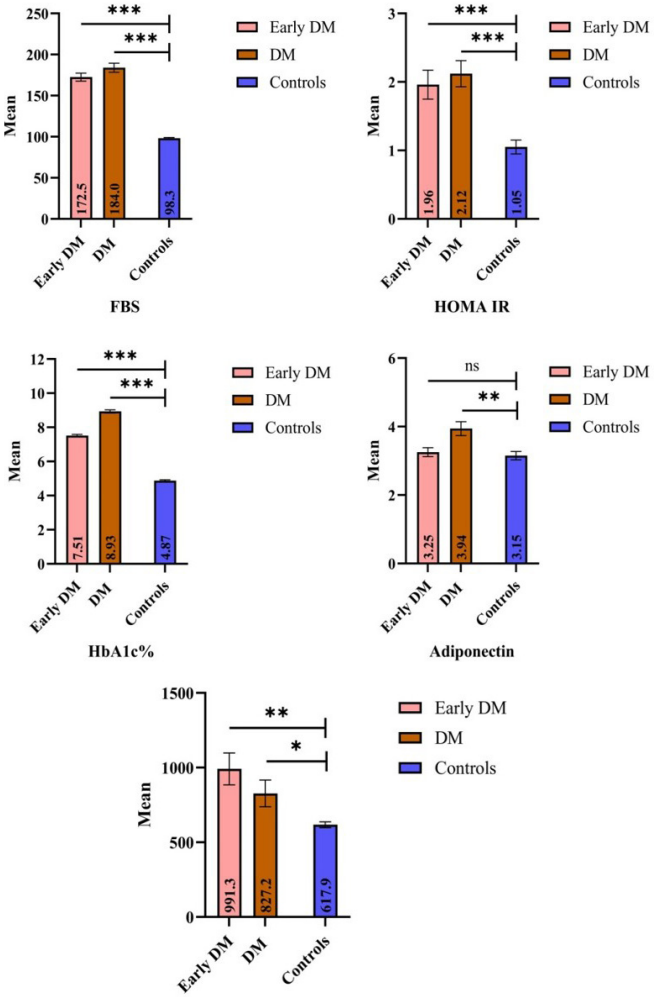

Figure 1. Comparison of parameters among groups using box plots for fasting blood sugar (FBS), glycated hemoglobin (HbA1c%), homeostatic model assessment for insulin resistance (HOMA-IR), adiponectin, and nesfatin-1. Abbreviations: FBS, fasting blood sugar; HbA1c, glycated hemoglobin; HOMA-IR, homeostatic model assessment for insulin resistance; DM, diabetes mellitus; ns, non-significant. Pairwise comparisons were conducted using ANOVA, with a significance level of 0.05 for the adjusted p-value

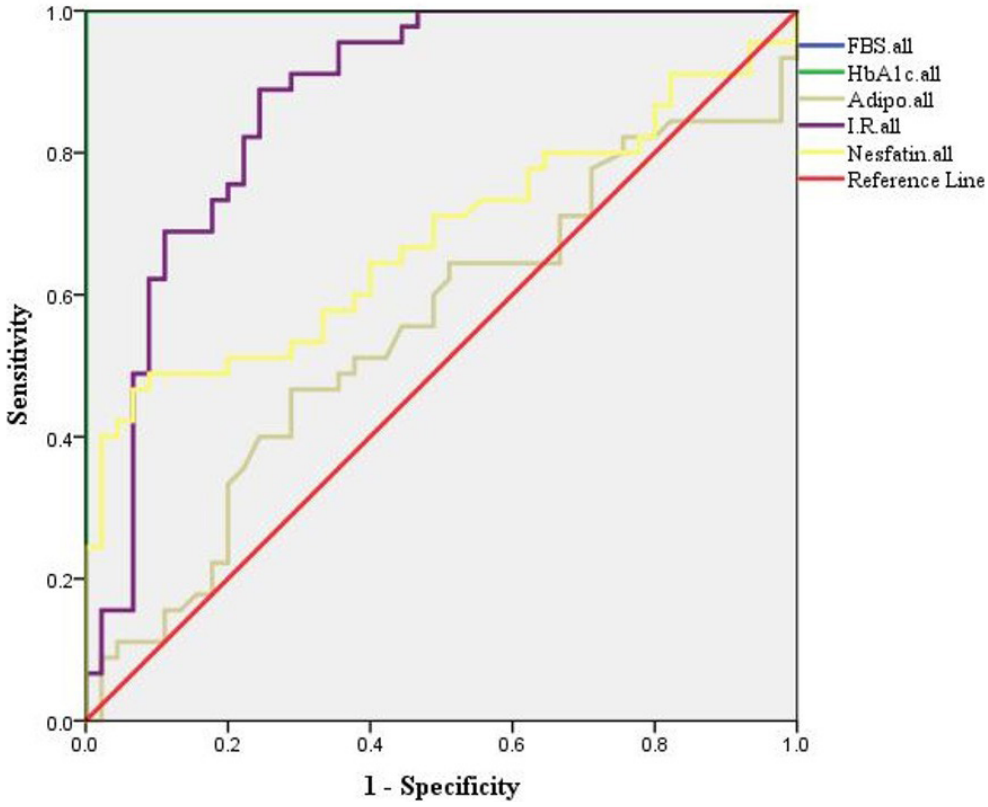

Figure 2. Receiver operating characteristic (ROC) curve analysis for blood glucose, glycated hemoglobin (HbA1c), adiponectin, insulin resistance (HOMA-IR), and nesfatin-1

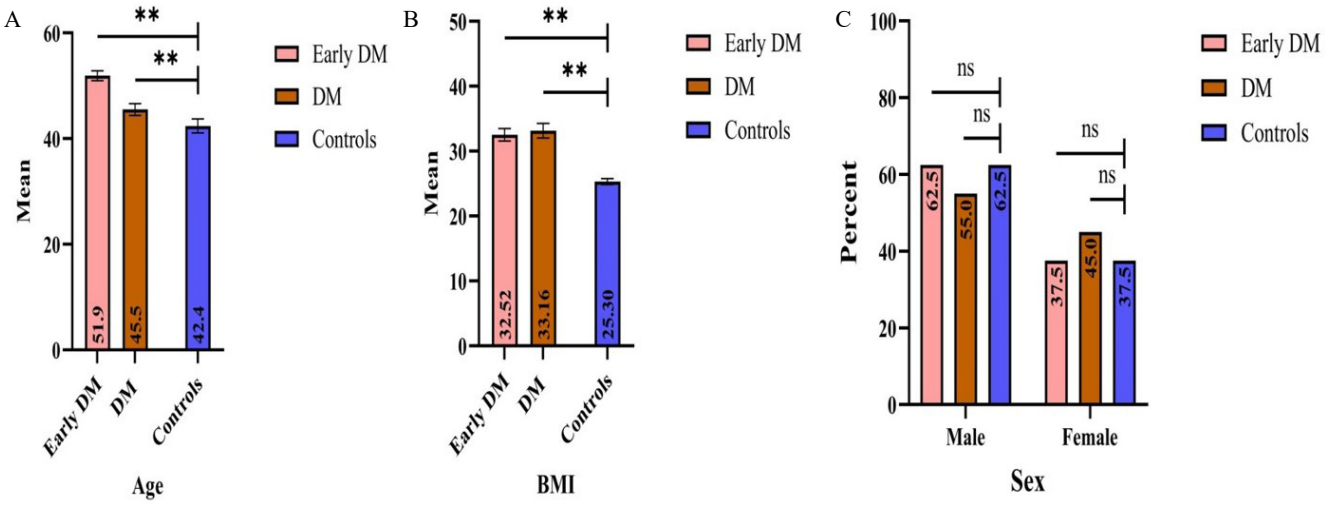

Figure 3. The mean and p-value of (A) age and (B) BMI and (C) the distribution of sex among study participant's BMI, Body Mass Index. Ns: Non-significant

groups was (0.442 and 0.446), respectively, indicating that the distribution is abnormal.

Also, the results of BMI showed that the early diabetes group had a high mean value (32.52), in addition to the diabetes mellitus type 2 (33.16). In contrast, the control group had a BMI value (25.3) with a p-value (0.0001) for both groups compared with the controls.

The result appears that blood glucose mean±SE concentration was higher in early diabetes and DM type 2 than in control, with mean±SE (172.5±4.98) for early DM and (184 ±5.46) for DM type 2. In contrast,

the control group had mean±SE (98.3±0.72) with high significant differences, and the p-value was 0.0001 for both study groups compared with the control group.

Serum glycated hemoglobin is considered an indicator of diabetes disease and is used as a monitoring and diagnosis parameter for Diabetes mellitus. The result in Table 2 shows the mean±SE of percent for early DM patients was (7.51±0.074), and for DM type 2 patients were (8.93±0.091) while the mean±SE of percent for the control group was (4.87±0.056) with significant differences and p-value (0.0001) for both study group.

The result of the study in Table 2 showed that the mean±SE concentration of adiponectin for early DM was (3.25±0.130), the mean±SE concentration for the control group was (3.15±0.122) with non-significant differences, and the p-value was (0.605). Conversely, the mean±SE concentration of adiponectin for DM type 2 patients was (3.94±0.204) with significant differences between patients with DM type 2 compared to control with p-value (0.0002).

HOMA IR results showed that the mean±SE of increase in early DM (1.96±0.212) while the mean±SE in the control group was (1.05±0.102) with high significant differences and p-value was (0.0001). In addition, the mean±SE of HOMA-IR in DM type 2 was (2.12±0.192) with significant differences compared with the control with a p-value (0.0001). The results are explained in Table 2.

Table 2. The mean ± SD, range, and differences of study parameters for each study group

Characteristics		Early DM	DM	Controls
FBG (mg/dl)	Mean ± SE	172.5±4.98	184±5.46	98.3±0.72
	Range	130-260	142-323	85-110
	p-value	0.0001	0.0001	-
HbA1c (%)	Mean ± SE	7.51±0.074	8.93±0.091	4.87±0.056
	Range	6.60-8.50	6.67-11.12	4.10-5.70
	p-value	0.0001	0.0001	-
Adiponectin (ng/ml)	Mean ± SE	3.25±0.130	3.94±0.204	3.15±0.122
	Range	2.21-6.67	2.42-8.56	2.29-7.76
	p-value	0.605	0.0002	-
Insulin resistant (HOMA IR)	Mean ± SE	1.96±0.212	2.12±0.192	1.05±0.102
	Range	0.89-8.42	0.93-8.22	0.65-4.36
	p-value	0.0001	0.0001	-
Nesfatin-1 (pg/ml)	Mean ± SE	991.3±107.4	827.2±89.65	617.9±19.37
	Range	399.2-3688	289.6-2212.4	414.07-1098.7
	p-value	0.002	0.0221	-

Statistically significant if P<0.01; *P values from ANOVA test (FBG, HbA1c%, adiponectin, HOMA IR, Nesfatin-1)

The study results showed that the mean±SE of nesfatin-1 for early DM was more than the control group (991.3±107.4) while the mean±SE concentration for control groups was (617.9±19.37) with significant differences between these two groups and the p-value was (0.002). Also, the mean±SE of nesfatin-1 for DM type 2 was (827.2±89.65) with significant differences, and the p-value was (0.0221). The result of nesfatin-1 in DM type 2 was less than early diabetes. The results are explained in Table 2.

To find the correlation between the study parameters for the patient, the researchers used Spearman's correlation analysis. The results are explained in Table 3.

The study results showed a high correlation between age with (Blood glucose, HbA1c, Insulin resistance, and nesfatin-1) with r = 0.413, 0.481, 0.299, and 0.391, and the p-values were 0.0001, 0.0001, 0.002, and 0001 respectively. In addition, fast blood glucose had a high correlation with (glycated hemoglobin, Insulin resistance, and nesfatin-1) with r = 0.797, 0.625, and 0.228, while the p-value was 0.0001, 0.0001, and 0.012, respectively. The explanation for this result is that blood glucose concentration is related to glycated hemoglobin when the glucose level in red blood cells is increased; also, blood glucose increase leads to insulin resistance—the blood glucose relation with nesfatin-1 because of the role of nesfatin-1 in carbohydrate metabolism.

The study also found a high correlation between glycated hemoglobin with (adiponectin, Insulin

Table 3. Spearman's correlation coefficient (ρ) between early diabetes mellitus (DM) and type 2 DM clinical parameters, demographic factors, Adiponectin, and Nesfatin-1

Parameters / Patients	Pearson r	P-value
Age -– FBS	0.413**	0.0001
Age – HbA1c	0.481**	0.0001
Age – I.R	0.299**	0.002
Age – Nesfatin-1	0.391**	0.0001
FBS - HbA1c	0.797**	0.0001
FBS – IR	0.625**	0.0001
FBS – Nesfatin-1	0.228*	0.012
HbA1c - Adiponectin	0.195*	0.033
HbA1c – I.R	0.523**	0.0001
HbA1c – Nesfatin-1	0.342**	0.0001
Nesfatin-1 – I.R	0.259**	0.007

**Correlation is significant at the 0.01 level (1-tailed)

* Correlation is significant at the 0.05 level (1-tailed)

resistance, and nesfatin-1) with r = 0.195, 0.523, and 0.342 with p-values 0.033, 0.0001,0.0001, respectively.

Nesfatin-1 concentration correlated with Insulin resistance with r = 0.259 and p-values (0.007) and clarify in Table 4. The AUC for fast blood sugar (FBS) and glycated hemoglobin (HbA1c) were 1.0 with a cut-off value of 120 for FBS and 6.15 for HbA1c and specificity and sensitivity were (100%) for both, and the significant differences were (p-value 0.0001) for both. Adiponectin has less sensitivity and specificity as a parameter for early diabetes diagnosis and diabetes type 2, and the cut-off value was 3.19, while the AUC was 0.548 with a p-value of 0.431. In addition, insulin resistance (I.R) has a cut-off value 1.03, and the AUC was 0.871 with a p-value of 0.0001. At last, the nesfatin-1 cut-off value was 771.5, and the AUC was 0.678, with a p-value of 0.004.

The cut-off value indicates that the value is considered a critical value for diabetes mellitus disease.

4. Discussion

The prevalence of type 2 diabetes is predicted to rise in the upcoming years, making it a worldwide health concern. To prevent and control diabetes, it is crucial to look into the risk factors for the disease and find appropriate biomarkers. Pre-diabetes is a high-risk state for the development of diabetes, usually characterized as blood glucose readings greater than normal but below the diabetes threshold (Tabák *et al.* 2012).

Our results display the FSG distribution (mean ± SE) in mg/dl for T2DM, early DM patients, and normal healthy samples. The ANOVA test results showed a substantial variance ($p<0.001$) across the three groups, according to the FSG results. Both patient groups exhibited a highly significant increase ($p<0.001$) compared to the control group. In the present investigation, the HbA1c values of both patient groups were significantly higher ($p<0.001$) than those of the control group. In T2DM patients, inadequate glycemic management is the cause of elevated HbA1c levels. This result is consistent with that of Khan *et al.* who found a high correlation between FPG and HbA1c levels (Khan *et al.* 2007).

Regarding IR, diabetes types T2DM and early DM are linked to IR and beta-cell dysfunction. A compensatory rise in insulin production initially maintains blood glucose levels within the normal range. As the illness progresses, beta cells change, making insulin secretion inadequate to maintain glucose homeostasis and leading to hyperglycemia. The majority of people with type 2 diabetes are obese or have higher body fat percentages, with the majority of this fat being stored in the abdomen. This adipose tissue induces IR (Galicia-Garcia *et al.* 2020). Given that insulin resistance is most likely the first metabolic aberration in early DM and DM type 2, this data can be explained.

According to cohort research, significant variations were noticed between T2DM patients and the controls in several metabolic markers. A study by (American Diabetes Association 2018) found that HbA1c and FPG were significantly higher in the T2DM group. While adiponectin levels dramatically dropped (Abudalo *et al.* 2024). This finding contradicts our research, which showed that T2DM patients had higher adiponectin levels. Several studies have reported that individuals diagnosed with type 2 diabetes mellitus exhibited reduced adiponectin concentrations in their bloodstream (Eltahir *et al.* 2020). Notably, type 2 diabetes mellitus, which exhibited adiponectin, a recently discovered biomarker, allows for a statistically significant improvement over the use of conventional risk variables in assessing the risks of diabetes and pre-diabetes (Li *et al.* 2009). Adiponectin improves insulin sensitivity by promoting glucose absorption in peripheral tissues, including the liver and muscle, and inhibiting the liver's ability to produce glucose. This system assists in preserving appropriate blood glucose levels and averts insulin resistance, a defining feature of type 2 diabetes (Yamauchi & Kadowaki 2013). Adiponectin regulates insulin sensitivity glucose and lipid metabolism, alters glucose homeostasis,

Table 4. Receiver operating characteristic (ROC) curve analysis of study parameters for diagnosing early diabetes mellitus (DM) and type 2 DM

Parameter	Cut-off value	Specificity (%)	Sensitivity (%)	Area-under curve	Sig.
FBS	120	100.0	100.0	1.0	0.0001
HbA1c	6.15	100 .0	100.0	1.0	0.0001
Adiponectin	3.19	71.1	46.7	0.548	0.431
Insulin resistant	1.03	75.6	88.9	0.871	0.0001
Nesfatin-1	771.5	93.3	46.7	0.678	0.004

promotes adipose tissue growth, and prevents the renin-angiotensin system activation. It has anti-inflammatory and antiatherogenic properties (Kim *et al.* 2007).

One study by Kern *et al.* (2003) found that adiponectin levels tend to decrease with age in individuals with type 2 diabetes. The researchers observed lower adiponectin concentrations in older diabetic patients than in younger ones. This decline in adiponectin levels with age may contribute to the progression of insulin resistance and metabolic dysfunction commonly observed in elderly individuals with diabetes (Kern *et al.* 2003). Another study by (Hotta *et al.* 2000) reported no significant correlation between age and adiponectin levels in individuals with impaired glucose tolerance (IGT), a pre-diabetic condition. This study corresponds with our finding that there is no relation between age and adiponectin level. Previous research found that plasma adiponectin levels were lower in elderly subjects with pre-diabetes than in subjects with normal glucose tolerance (Kong *et al.* 2015). This study conflicts with our study, which indicates no relation between age and adiponectin level. According to the results of a ten-year prospective trial including 912 people, adiponectin was linked to T2DM and newly-onset pre-diabetes. (Cho *et al.* 2020) study contradicts our findings about early diabetes outcomes and is consistent with type 2 diabetes. Several studies have investigated the relationship between age and insulin resistance in early diabetes.

For example, Abbasi *et al.* (1998) found that older age was independently associated with insulin resistance, even after adjusting for other risk factors such as obesity and physical activity levels. In addition, a meta-analysis study revealed a consistent correlation between age and insulin resistance in early diabetes, indicating a substantial link between age and insulin resistance across various investigations. (Petersen *et al.* 2007) This study supports the findings of our investigation. Several studies have demonstrated adiponectin levels and HbA1c levels to be inversely correlated. This condition indicates that adiponectin levels tend to decline with increasing HbA1c levels, which indicates worse glycemic management. This negative association raises the possibility that reduced adiponectin levels in diabetics may be a factor in insulin resistance and worse glycemic management (Vendramini *et al.* 2006). Our findings indicated a favorable relationship between adiponectin and HbA1c.

Another study found that T2DM patients had lower serum nesfatin-1 levels than the healthy group. This study's findings are at odds with ours, which supports the idea that nesfatin-1 increases the risk of T2DM and early diabetes. Lower nesfatin-1 levels in the T2DM group may account for the significantly greater insulin resistance in this group since nesfatin-1 has been demonstrated to modify glucose metabolism by improving insulin sensitivity and lowering insulin resistance (Khalili *et al.* 2017). A study that first investigated the fasting plasma levels nesfatin-1 in type 2 diabetes patients found that fasting nesfatin-1 levels were significantly lower in the type 2 diabetes group than in healthy subjects (Li *et al.* 2010). This study conflicts with our study that the result nesfatin-1 in early DM and T2DM was higher in patients than in the healthy group.

Previous studies found that only newly diagnosed type 2 diabetes patients showed increased circulating nesfatin-1 levels compared to the controls. This study corresponds with our result (Zhang *et al.* 2012; Guo *et al.* 2013).

A study showed that circulating nesfatin-1 levels were significantly lower in type 2 diabetes patients receiving anti-diabetic treatment, but newly diagnosed type 2 diabetes patients exhibited considerably higher levels of circulating nesfatin-1. The significantly reduced heterogeneity in both groups suggested that treatment for two types of diabetes is the primary source of heterogeneity (Zhai *et al.* 2017). The study by (Matta *et al.* 2022) found significantly decreased serum nesfatin-1 levels among newly diagnosed drug-naive patients with either pre-diabetes or DMT2 than healthy control. Hence, the results of this study are in contrast to our results. Another study in newly diagnosed treatment-naive diabetic and pre-diabetic patients (one group) in Jordan reported elevated nesfatin-1 levels compared to euglycemic subjects as a control group (Akour *et al.* 2017). This study agrees with our study.

Elevated nesfatin-1 levels increase the amount of insulin secreted in response to glucose by inducing Ca+2 influx via an L-type channel (Nakata *et al.* 2011). Additionally, it has been discovered that insulin increases the expression of nesfatin-1 in adipose tissue. Thus, it is possible to conclude that nesfatin-1's effects on glucose metabolism depend solely on insulin (Ayada *et al.* 2015). A study by (Mohammad & Gallaly 2020) showed a significant difference between patients with type 2 diabetes and healthy controls (P value = 0.004) and exhibited a decrease in the concentration of nesfatin-1 in patients. This result is contrary to the results of our study. This study showed that the ROC curve is a biomarker for type 2 diabetes, with an area under the curve of 0.64 and a P value = 0.0042. This finding is consistent with our study, which showed that the AUC value was 0.678 and a P

value = 0.004. The same previous study demonstrated that serum NES-1 level negatively correlates with all the FSG, HbA1C, FSI, HOMA-IR, and BMI. These negative correlations can be explained by the role NES-1 in glucose metabolism and its anorexigenic effect (Mohammad & Gallaly 2020). This finding agreed with the results of a study done by Tsuchiya and his coworkers (Tsuchiya *et al.* 2010). These two studies conflict with our study in the correlation (negatively), with our result finding a positive correlation between nesfatin-1 and FBG, HOMA IR, and HbA1c.

In conclusion, the study showed the effect of adiponectin, nesfatin-1, and some diagnostic parameters on early diabetes and DM type 2. The results prove that nesfatin-1 concentration increases in both study groups with highly significant differences compared to the control, and it has a very important role in the prognosis or diagnosis of disease. Nesfatin-1 has a good correlation with insulin resistance in the study sample, and this result indicates that nesfatin-1 has a good role in insulin sensitivity and resistance in diabetes mellitus disease. Adiponectin doesn't affect early diabetes, unlike the DM type 2 group, which has a higher concentration than the control group. This finding indicates that adiponectin concentration is affected as diabetes progresses, given its effect on adipose tissue and its association with obesity.

Conflict of Interests

The authors declare no conflict of interest.

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