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# Demographics -Based on Biochemical Markers Levels Among Iraqi Adults with Normal Glucose Tolerance, Impaired Glucose Tolerance, and Type 2 Diabetes Mellitus

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**Abstract**

**Introduction:** Type 2 diabetes mellitus and impaired glucose tolerance conditions result in changes in glucose metabolism, insulin, lipid profile, and adipokine activities. Resistin, visfatin, and apelin are adipokines that might indicate the metabolic imbalance that differs by demographics and anthropometrics, such as age, gender, and BMI.

**Objectives:** To determine the serum levels of resistin, visfatin, and apelin in healthy subjects, patients with impaired glucose tolerance, and patients with type 2 diabetes mellitus, as well as the differences in these indicators according to age, gender, and BMI groups.

**Methodology:**The sample size of 180 subjects consisted of three groups: healthy individuals, patients with impaired glucose tolerance, and patients with type 2 diabetes mellitus with 60 individuals in each group. The serum levels of adipokines, insulin, fasting blood glucose, 2-hour blood glucose, hemoglobin A1C (HbA1c), and lipid profile were measured in addition to dividing them into subgroups based on age, gender, and BMI category. Then, statistical analyses were performed to identify any differences between groups.

**Results:** Significant reductions in the serum levels of resistin and visfatin were recorded according to the metabolic disorder, where the minimum levels of these two indicators were observed in patients with type 2 diabetes mellitus. However, apelin showed an incremental change starting from the healthy control group, reaching a peak in type 2 diabetes mellitus. Also, the glycemic indicators showed significantly high levels among the type 2 diabetes mellitus group compared to the other two groups. Moreover, patients with diabetes mellitus had significantly high levels of total cholesterol and LDL. In the age-based analysis, total cholesterol and LDL levels increased significantly with aging. Differences in the selected metabolic indicators, including lipids, were noticed between females and males; however, these differences could not be generalized because of unequal distribution of the genders. The BMI-based analysis indicated a gradual deterioration of metabolic functions according to BMI. This included higher levels of apelin, insulin, glucose markers, HbA1c, total cholesterol, triglyceride, LDL, and VLDL, and low levels of resistin, visfatin, and HDL.

**Conclusions:** The results showed significant changes in the selected adipokines and metabolic indicators according to the glucose tolerance status, age, gender, and BMI. Therefore, higher BMI was correlated with increased adipokine imbalance, insulin resistance, hyperglycemia, and dyslipidemia.

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**Keywords:** *Resistin; Visfatin; Apelin; Type 2 Diabetes Mellitus; Impaired Glucose Tolerance; Body Mass Index; Age; Gender*

## 1. Introduction

Type 2 diabetes mellitus (T2DM) is considered to be one of the leading chronic metabolic disorders characterized by insulin insensitivity, progressing insulin resistance, abnormalities in glucose regulation, and metabolic complications [1]. Current research has indicated that inflammatory adipokines play a key role in the pathophysiology of T2DM and could be affected by factors such as physical activity and metabolism [1]. One of the most prominent features in the onset and development of T2DM is insulin resistance that leads to hyperglycemia, compensatory hyperinsulinemia, β-cell impairment, and cardiometabolic disturbances [2]. Hence, searching for new biomarkers for detecting early metabolic disorders, including those among individuals with impaired glucose tolerance (IGT), that would allow for a more accurate risk assessment, is of critical importance.

It should be noted that the current scientific community agrees that adipose tissue should be regarded as an active endocrine organ that secretes several metabolically active compounds called adipokines which are essential for regulating glucose metabolism, insulin sensitivity, inflammation, hunger, vascular processes, and lipids' functions [1]. One of the adipokines that has attracted much attention recently due to its involvement in the pathogenesis of T2DM and obesity is visfatin [3]. According to the results obtained by researchers, serum visfatin concentration along with chemerin could potentially be used as clinical biomarkers among individuals with T2DM and obesity [3]. In addition, another promising adipokine is apelin which is thought to play a key role in maintaining insulin sensitivity and glucose uptake and that has been studied experimentally as a prospective approach for improving insulin sensitivity in T2DM [4].

In addition to dietary and behavioral factors, some researchers have considered the possibility of the relationship between visfatin and diet as well as atherogenic effects in patients with T2DM [5]. Other adipocytokines also have been explored as possible predictive factors of metabolic complications, such as metabolic dysfunction-associated steatotic liver disease among patients with T2DM [6]. It suggests that adipokines' dysregulation could also be connected to more general metabolic disturbances, hepatic dysfunction, and adverse vascular outcomes.

A special focus on the evaluation of adipokines' levels among individuals with IGT seems to be relevant since it is regarded as a risk factor of developing T2DM and involves metabolic disorders that can be detected based on adipokine levels. Recent research involving patients with IGT demonstrated the role of apelin and resistin in the metabolism of individuals with metabolic disorders before the development of T2DM [7]. Besides, studies conducted in other countries have also assessed the relationship between adipokines and the prevalence of obesity and T2DM among specific populations, such as in Iraq, where serum nesfatin-1, adiponectin, and resistin were associated with obesity and T2DM [8]. As it could be seen, there is a need for conducting more studies to assess adipokine levels in relation to glucose tolerance status in Iraq.

T2DM is usually accompanied by other endocrine and inflammatory disorders that might affect the levels of adipokines among patients. For instance, it was found that thyroid dysfunction in patients with T2DM is associated with dysregulation of adipokines, such as chemerin, resistin, and visfatin [9]. Visfatin also was found to be correlated with carbohydrate and polyunsaturated fatty acid consumption among patients with T2DM [10]. At the same time, resistin-related parameters were suggested to predict cardiovascular risk in patients with T2DM [11].

Obesity is one of the greatest factors that cause insulin resistance and T2DM. Therefore, many scientists suggest that adipokines should be studied as clinically relevant biomarkers of metabolic disorders, including obesity, in relation to their diagnostic and therapeutic implications [12]. Moreover, the dysregulation of adipokine levels also appears to occur among healthy family members of patients with T2DM, suggesting that adipokines might be altered even before the onset of clinical manifestations of T2DM [13]. Additionally, the presence of both T2DM and thyroid dysfunction was also associated with the increase of inflammatory markers and adipokines [14].

Resistin is one of the most studied adipokines when talking about inflammation, insulin resistance, and metabolic risks. It should be noted that resistin's association with insulin resistance varies depending on the target population and clinical setting. For instance, some studies have found that resistin is mainly associated with inflammation

and renal dysfunction, not with insulin resistance [15]. At the same time, other studies have found changes in apelin and resistin serum levels among patients with impaired fasting glucose, IGT, T2DM, and metabolic syndrome [16].

Another issue worth consideration in regard to adipokines' levels is the influence of obesity on the latter ones. Thus, serum leptin, resistin, and adiponectin levels were found to be different among obese and non-obese T2DM patients with the newly diagnosed form of the disease [17]. In turn, the strong link between T2DM and obesity has been widely discussed by scholars in regard to its epidemiology, mechanisms, and treatment. It was stated that obesity causes insulin resistance, chronic inflammation, lipids' abnormalities, and β-cell malfunctioning [18]. Moreover, novel approaches for treating T2DM, such as increasing the activity of incretin system, indicate the complicated interplay between T2DM, adipocytes' dysfunction, and glucose metabolism [19]. Pharmacological agents for treating diabetes, like metformin, also might influence adipokine's levels, suggesting the impact of treatments on adipokine balance [20].

Although the number of studies exploring the association between adipokines' and glucose tolerance levels has recently become larger, there are still only a few that have assessed serum visfatin, apelin, and resistin concentrations at once among patients with different glucose tolerance and also taking into account age, gender, and body mass index (BMI). Moreover, the simultaneous assessment of these three factors is not typical for the Iraqi scientific community, despite diabetes and obesity being widespread diseases in Iraq. Evaluating the behavior of adipokines among individuals with different glucose tolerance statuses and demographics characteristics will be able to clarify early metabolic changes and reveal at-risk individuals.

Thus, the objective of the present study is to evaluate serum resistin, visfatin, and apelin concentrations among Iraqi adults with normal glucose tolerance, impaired glucose tolerance, and type 2 diabetes mellitus. Specifically, the aim of the study is to compare the aforementioned adipokines and metabolic parameters according to age, gender, and BMI categories.

## 2. Materials and Methods

### *2.1 Study Design*

The current research work was carried out as a case-control analysis to identify serum resistin, visfatin, and apelin levels and assess their relationship with glucose tolerance in Iraqi adults. The sample population consisted of three categories including healthy subjects, subjects with impaired glucose tolerance, and subjects with type 2 diabetes mellitus. In addition, the effects of different age groups, gender, and body mass index on the results were examined as well.

### *2.2 Study Population*

A total of **180 participants** were included in the study and divided equally into three groups:

| Group | Description | Number |
|---|---|---|
| Healthy control group | Apparently healthy individuals with normal glucose tolerance | 60 |
| Impaired glucose tolerance group | Individuals diagnosed with impaired glucose tolerance | 60 |
| Type 2 diabetes mellitus group | Patients diagnosed with type 2 diabetes mellitus | 60 |

Participants were Iraqi adults recruited for evaluation of adipokine and metabolic markers. The three groups were compared in terms of serum adipokines, glycemic markers, lipid profile, age, gender, and BMI categories.

### *2.3 Inclusion Criteria*

Participants were included if they met the following criteria:

1. Adults within the studied age range.
2. Individuals classified as healthy controls, impaired glucose tolerance, or type 2 diabetes mellitus according to clinical and laboratory evaluation.
3. Participants with available laboratory measurements for resistin, visfatin, apelin, insulin, fasting blood glucose, 2-hour blood glucose, HbA1c, and lipid profile.
4. Participants who provided blood samples suitable for biochemical and immunological analysis.

### *2.4 Exclusion Criteria*

Participants were excluded if they had conditions that could interfere with adipokine or metabolic marker levels, including:

1. Acute infection or inflammatory disease.
2. Chronic renal failure or severe liver disease.
3. Malignancy.
4. Pregnancy.
5. Current use of medications known to strongly affect inflammatory or adipokine levels, unless clinically unavoidable.
6. Incomplete laboratory data.

### *2.5 Clinical and Demographic Data Collection*

Demographic and clinical data were collected for each participant, including **age**, **gender**, and glucose tolerance status. Participants were further categorized according to age groups as follows:

| Age category | Classification |
|---|---|
| 40–49 years | Younger age group |
| 50–59 years | Middle age group |
| ≥60 years | Older age group |

For BMI-based analysis, participants were categorized into three BMI groups:

| BMI category | BMI range |
|---|---|
| Normal BMI | 18.5–24.9 kg/m² |
| Overweight | 25.0–29.9 kg/m² |
| Obese | ≥30.0 kg/m² |

BMI was considered as an anthropometric variable for evaluating the relationship between body weight status, adipokine imbalance, and metabolic disturbance.

### *2.6 Blood Sample Collection*

Venous blood samples were collected from each participant under appropriate clinical and laboratory conditions. Blood samples were used for measurement of adipokines, glycemic markers, and lipid profile parameters. Serum was separated according to standard laboratory procedures and stored under suitable conditions until analysis.

### *2.7 Measurement of Serum Adipokines*

Serum concentrations of **resistin**, **visfatin**, and **apelin** were measured using the enzyme-linked immunosorbent assay (**ELISA**) technique according to the manufacturer's instructions. The measured adipokines were expressed as follows:

| Marker | Unit |
|---|---|
| Resistin | µg/L |
| Visfatin | µg/L |
| Apelin | pg/mL |

These adipokines were selected because of their involvement in adipose tissue activity, insulin resistance, glucose metabolism, inflammation, and obesity-related metabolic dysfunction.

### *2.8 Measurement of Glycemic Markers*

The following glycemic and insulin-related markers were measured:

| Marker | Unit |
|---|---|
| Fasting blood glucose | mg/dL |
| 2-hour blood glucose | mg/dL |
| HbA1c | % |
| Fasting insulin | mU/L |

Fasting blood glucose and 2-hour blood glucose were used to distinguish glucose tolerance status. HbA1c was used as an indicator of longer-term glycemic control, while fasting insulin was included to assess insulin-related metabolic changes.

### *2.9 Measurement of Lipid Profile*

Lipid profile parameters were measured using automated biochemical analysis. The following lipid markers were included:

| Marker | Unit |
|---|---|
| Total cholesterol | mg/dL |
| Triglycerides | mg/dL |
| High-density lipoprotein | mg/dL |
| Low-density lipoprotein | mg/dL |
| Very-low-density lipoprotein | mg/dL |

Lipid profile analysis was performed to evaluate dyslipidemia patterns across glucose tolerance groups and according to age, gender, and BMI categories.

### *2.10 Study Variables*

The main variables analyzed in this study were classified as follows:

| Variable type | Variables |
|---|---|
| Grouping variables | Healthy control, impaired glucose tolerance, type 2 diabetes mellitus |

| | |
|---|---|
| Demographic variables | Age and gender |
| Anthropometric variable | BMI category |
| Adipokine markers | Resistin, visfatin, apelin |
| Glycemic markers | Fasting blood glucose, 2-hour blood glucose, HbA1c, insulin |
| Lipid markers | Total cholesterol, triglycerides, HDL, LDL, VLDL |

### *2.11 Statistical Analysis*

Statistical analysis methods were used to analyze the collected data. The results of measurements for continuous variables were presented in the form of mean ± SD, while for categorical variables, results are given as number (%) of patients with particular features. The comparisons of three main groups in the study were made using one-way analysis of variance (ANOVA). In order to find out whether there are any differences between two groups, post-hoc tests were used after one-way ANOVA test. Independent samples t-test was used to compare gender-related factors. One-way ANOVA was used to compare age and BMI.

### *2.12 Ethical Considerations*

The present study followed the ethical rules and regulations for carrying out clinical studies on human subjects. All subject data were treated in confidence, while all samples collected were used for research purposes only. Subject identity information was anonymized before data analysis to protect the anonymity of the subjects involved.

The present study compared the adipokine and metabolic characteristics of healthy subjects, subjects with IGT, and diabetic subjects. The methodological approach adopted was intended to evaluate differences among subjects with varying glucose status as well as the influence of age, sex, and body mass index on the levels of resistin, visfatin, apelin, glycemic indicators, and lipid profile.

## 3. Results

### *3.1 Baseline Characteristics of the Study Population*

A total of **180 participants** were included in this study and divided equally into three groups: **healthy controls (HC, n = 60)**, **impaired glucose tolerance (IGT, n = 60)**, and **type 2 diabetes mellitus (T2DM, n = 60)**. The mean age was **52.17 ± 6.24 years** in the HC group, **54.82 ± 6.25 years** in the IGT group, and **52.62 ± 6.23 years** in the T2DM group. The difference in age among the three groups was statistically significant (**p = 0.048**), indicating a slight but significant variation in age distribution.

Regarding gender, males represented **75.0%** of the HC group and **100%** of both the IGT and T2DM groups. Females were present only in the HC group, representing **25.0%** of that group. Therefore, gender-based findings were interpreted cautiously due to the unequal distribution of males and females across the study groups.

***Table 1. Baseline characteristics of the study groups***

| Variable | HC n=60 | IGT n=60 | T2DM n=60 | p-value |
|---|---|---|---|---|
| Age, years | 52.17 ± 6.24 | 54.82 ± 6.25 | 52.62 ± 6.23 | 0.048 |
| Male, n (%) | 45 (75.0%) | 60 (100%) | 60 (100%) | — |
| Female, n (%) | 15 (25.0%) | 0 (0%) | 0 (0%) | — |

### *3.2 Comparison of Serum Adipokine Levels Among Study Groups*

The serum adipokines varied significantly among the three groups. For example, the resistin level declined consistently with respect to each group in the following order: HC > IGT > T2DM. The highest level of resistin was noted in HC group at 5.42 ± 1.02 µg/L followed by the IGT group with the value of 4.93 ± 1.06 µg/L. In contrast, the lowest resistin level was recorded in the T2DM group at 3.58 ± 0.99 µg/L ($p < 0.001$).

Similarly, the decline in visfatin was consistent among the three groups in the descending order: HC > IGT > T2DM. The highest visfatin level was found in the HC group with a value of 15.03 ± 3.37 µg/L. It was then followed by the IGT group with the level of 13.77 ± 3.39 µg/L. Conversely, the T2DM group demonstrated the lowest level of 10.56 ± 3.32 µg/L. Thus, the variance between the groups was significant ($p < 0.001$).

On the other hand, apelin showed an inverse trend with respect to the above two adipokines. The apelin level increased gradually from the HC group (385.40 ± 10.88 pg/mL) to the IGT group (461.18 ± 14.10 pg/mL) and to the T2DM group (502.12 ± 16.91 pg/mL) ($p < 0.001$). Such changes in adipokine levels were demonstrated in Figure 1 below.

***Table 2. Comparison of serum adipokines among study groups***

| Marker | HC n=60 | IGT n=60 | T2DM n=60 | p-value |
|---|---|---|---|---|
| Resistin, µg/L | 5.42 ± 1.02 | 4.93 ± 1.06 | 3.58 ± 0.99 | <0.001 |
| Visfatin, µg/L | 15.03 ± 3.37 | 13.77 ± 3.39 | 10.56 ± 3.32 | <0.001 |
| Apelin, pg/mL | 385.40 ± 10.88 | 461.18 ± 14.10 | 502.12 ± 16.91 | <0.001 |

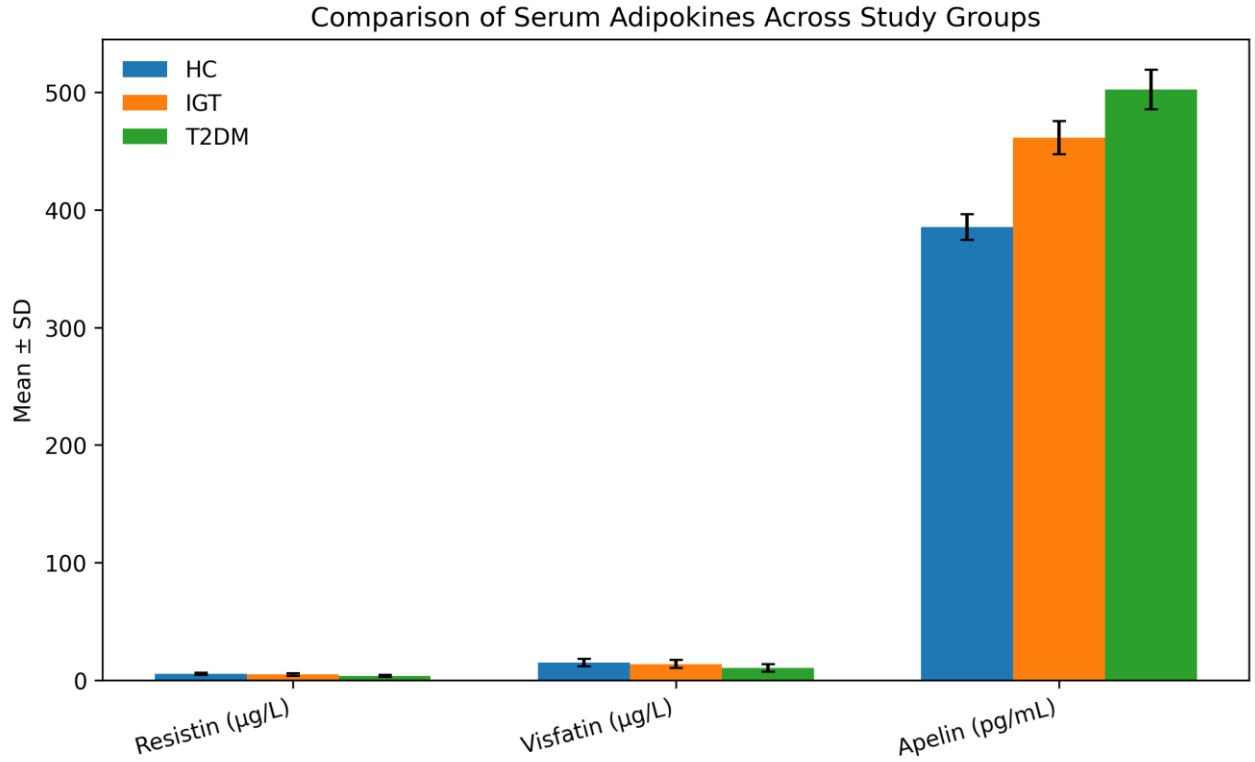


***Figure 1. Comparison of serum adipokine levels among study groups.*** *Serum resistin and visfatin levels decreased progressively from healthy controls to impaired glucose tolerance and type 2 diabetes mellitus groups, whereas serum apelin levels increased across the same groups. Data are presented as mean ± SD. HC: healthy control; IGT: impaired glucose tolerance; T2DM: type 2 diabetes mellitus.*

### *3.3 Comparison of Glycemic Markers Among Study Groups*

There was a notable decrease in the glycemic parameters among the three groups used for the study. Blood glucose values after fasting rose from 92.03 ± 5.95 mg/dL for the HC group to 113.53 ± 11.55 mg/dL for the IGT group and further to 139.03 ± 11.95 mg/dL for the T2DM group ($p < 0.001$). Likewise, blood glucose values after two hours rose significantly from 99.95 ± 7.39 mg/dL for the HC group to 163.00 ± 7.79 mg/dL for the IGT group and up to 279.97 ± 9.63 mg/dL for the T2DM group ($p < 0.001$).

Moreover, there was a progressive increase noted in HbA1c, which increased from 5.45 ± 0.32% for the HC group to 6.05 ± 0.51% for the IGT group and then to 7.50 ± 0.58% for the T2DM group ($p < 0.001$). Insulin values, in turn, were highest in the IGT group at 14.27 ± 1.54 mU/L, followed by the T2DM group at 12.90 ± 0.93 m.

***Table 3. Comparison of glycemic markers among study groups***

| Marker | HC n=60 | IGT n=60 | T2DM n=60 | p-value |
|---|---|---|---|---|
| Insulin, mU/L | 9.01 ± 0.92 | 14.27 ± 1.54 | 12.90 ± 0.93 | <0.001 |
| 2-hour BG, mg/dL | 99.95 ± 7.39 | 163.00 ± 7.79 | 279.97 ± 9.63 | <0.001 |
| FBG, mg/dL | 92.03 ± 5.95 | 113.53 ± 11.55 | 139.03 ± 11.95 | <0.001 |
| HbA1c, % | 5.45 ± 0.32 | 6.05 ± 0.51 | 7.50 ± 0.58 | <0.001 |

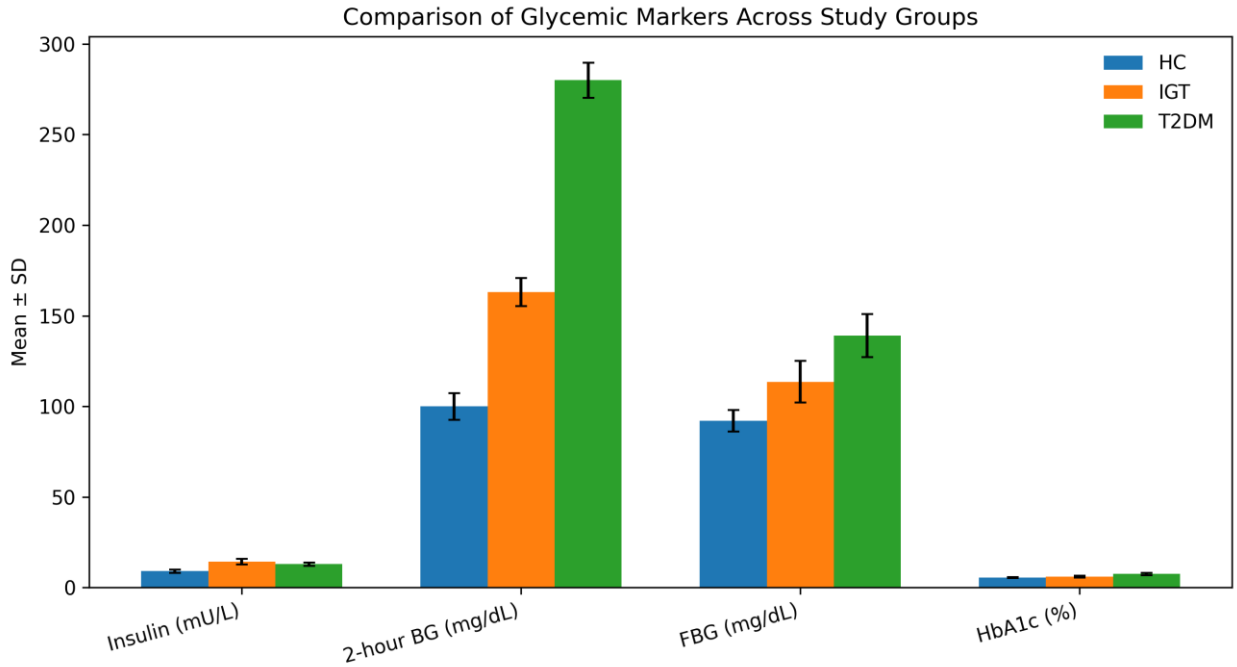


***Figure 2. Comparison of glycemic markers among study groups.*** *Fasting blood glucose, 2-hour blood glucose, and HbA1c increased progressively from healthy controls to impaired glucose tolerance and type 2 diabetes mellitus groups. Insulin levels were highest in the impaired glucose tolerance group. Data are presented as mean ± SD. BG: blood glucose; FBG: fasting blood glucose; HbA1c: glycated hemoglobin.*

### *3.4 Comparison of Lipid Profile Among Study Groups*

The lipid parameters were found to differ greatly between the study groups. For total cholesterol, the highest mean value (107.60 ± 18.06 mg/dL) was recorded in the T2DM group compared to the values in the HC group (92.88 ± 15.85 mg/dL) and the IGT group (89.55 ± 17.71 mg/dL) ($p < 0.001$).

For triglycerides, the highest mean concentration was observed in the IGT group (49.33 ± 23.55 mg/dL) followed by the T2DM group (43.00 ± 18.92 mg/dL) and HC group (36.77 ± 23.70 mg/dL) (p = 0.009). The highest level of HDL was observed in the HC group (41.92 ± 5.71 mg/dL) while the lowest was noted in the IGT group (35.58 ± 5.83 mg/dL); the mean for the T2DM group fell between (38.18 ± 5.97 mg/dL) (p < 0.001).

Regarding LDL, its highest mean was recorded in the T2DM group (60.82 ± 19.23 mg/dL) compared to the HC group (43.61 ± 18.61 mg/dL) and IGT group.

***Table 4. Comparison of lipid profile among study groups***

| Marker | HC n=60 | IGT n=60 | T2DM n=60 | p-value |
|---|---|---|---|---|
| Total cholesterol, mg/dL | 92.88 ± 15.85 | 89.55 ± 17.71 | 107.60 ± 18.06 | <0.001 |
| Triglycerides, mg/dL | 36.77 ± 23.70 | 49.33 ± 23.55 | 43.00 ± 18.92 | 0.009 |
| HDL, mg/dL | 41.92 ± 5.71 | 35.58 ± 5.83 | 38.18 ± 5.97 | <0.001 |
| LDL, mg/dL | 43.61 ± 18.61 | 44.10 ± 19.15 | 60.82 ± 19.23 | <0.001 |
| VLDL, mg/dL | 7.35 ± 4.74 | 9.87 ± 4.71 | 8.60 ± 3.78 | 0.009 |

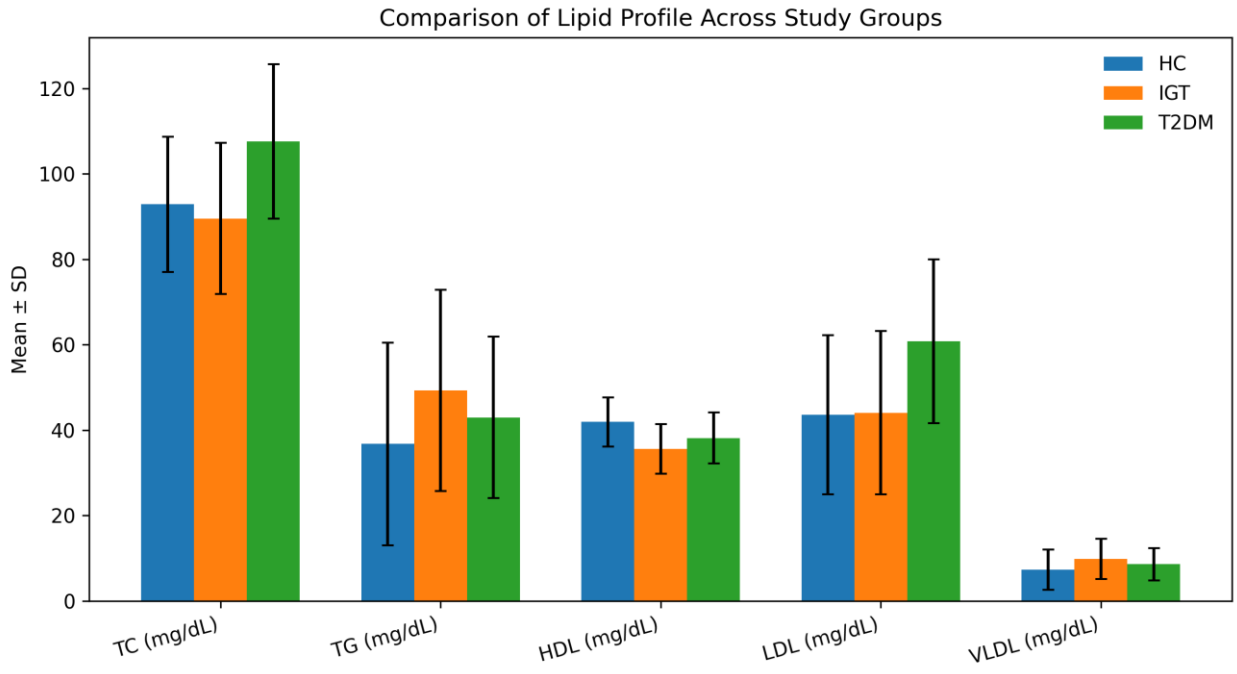


***Figure 3. Comparison of lipid profile parameters among study groups.*** *The figure shows differences in total cholesterol, triglycerides, HDL, LDL, and VLDL among healthy controls, impaired glucose tolerance, and type 2 diabetes mellitus groups. Total cholesterol and LDL were highest in the type 2 diabetes mellitus group, while HDL was lowest in the impaired glucose tolerance group. Data are presented as mean ± SD. TC: total cholesterol; TG: triglycerides; HDL: high-density lipoprotein; LDL: low-density lipoprotein; VLDL: very-low-density lipoprotein.*

### *3.5 Age-Based Comparison of Adipokines and Metabolic Markers*

The participants were divided into three age groups, which included ages of 40-49, 50-59, and those aged more than or equal to 60 years. Both total cholesterol and LDL were found to be significantly age-dependent and showed an increase with age. The total cholesterol values rose from 89.24 ± 17.20 mg/dL in the first age group to 98.93 ±

17.81 mg/dL in the second age group and 102.87 ± 21.34 mg/dL in the third age group (p=0.001). An increase in LDL was also noticed, from 41.29 ± 18.69 mg/dL in the first age group to 51.53 ± 20.07 mg/dL in the second age group and 57.85 ± 20.78 mg/dL in the third age group (p=0.0).

***Table 5. Age-based comparison of adipokines and metabolic markers***

| Marker | 40–49 years n=54 | 50–59 years n=96 | ≥60 years n=30 | p-value |
|---|---|---|---|---|
| Age, years | 46.11 ± 2.77 | 54.02 ± 2.74 | 63.33 ± 2.32 | <0.001 |
| Resistin, µg/L | 4.34 ± 1.31 | 4.78 ± 1.23 | 4.77 ± 1.35 | 0.107 |
| Visfatin, µg/L | 13.38 ± 3.92 | 12.92 ± 4.02 | 13.28 ± 3.07 | 0.761 |
| Apelin, pg/mL | 449.28 ± 56.73 | 451.06 ± 48.37 | 445.30 ± 46.54 | 0.862 |
| Insulin, mU/L | 11.44 ± 2.36 | 12.34 ± 2.53 | 12.29 ± 2.63 | 0.094 |
| 2-hour BG, mg/dL | 179.69 ± 78.97 | 182.57 ± 75.69 | 178.17 ± 69.04 | 0.951 |
| FBG, mg/dL | 113.20 ± 20.63 | 115.89 ± 22.58 | 114.60 ± 21.65 | 0.769 |
| HbA1c, % | 6.33 ± 1.04 | 6.32 ± 0.97 | 6.37 ± 0.98 | 0.972 |
| Total cholesterol, mg/dL | 89.24 ± 17.20 | 98.93 ± 17.81 | 102.87 ± 21.34 | 0.001 |
| Triglycerides, mg/dL | 41.96 ± 20.65 | 45.23 ± 25.97 | 37.93 ± 11.36 | 0.281 |
| HDL, mg/dL | 39.56 ± 6.34 | 38.35 ± 6.33 | 37.43 ± 6.46 | 0.308 |
| LDL, mg/dL | 41.29 ± 18.69 | 51.53 ± 20.07 | 57.85 ± 20.78 | 0.001 |
| VLDL, mg/dL | 8.39 ± 4.13 | 9.05 ± 5.19 | 7.59 ± 2.27 | 0.281 |

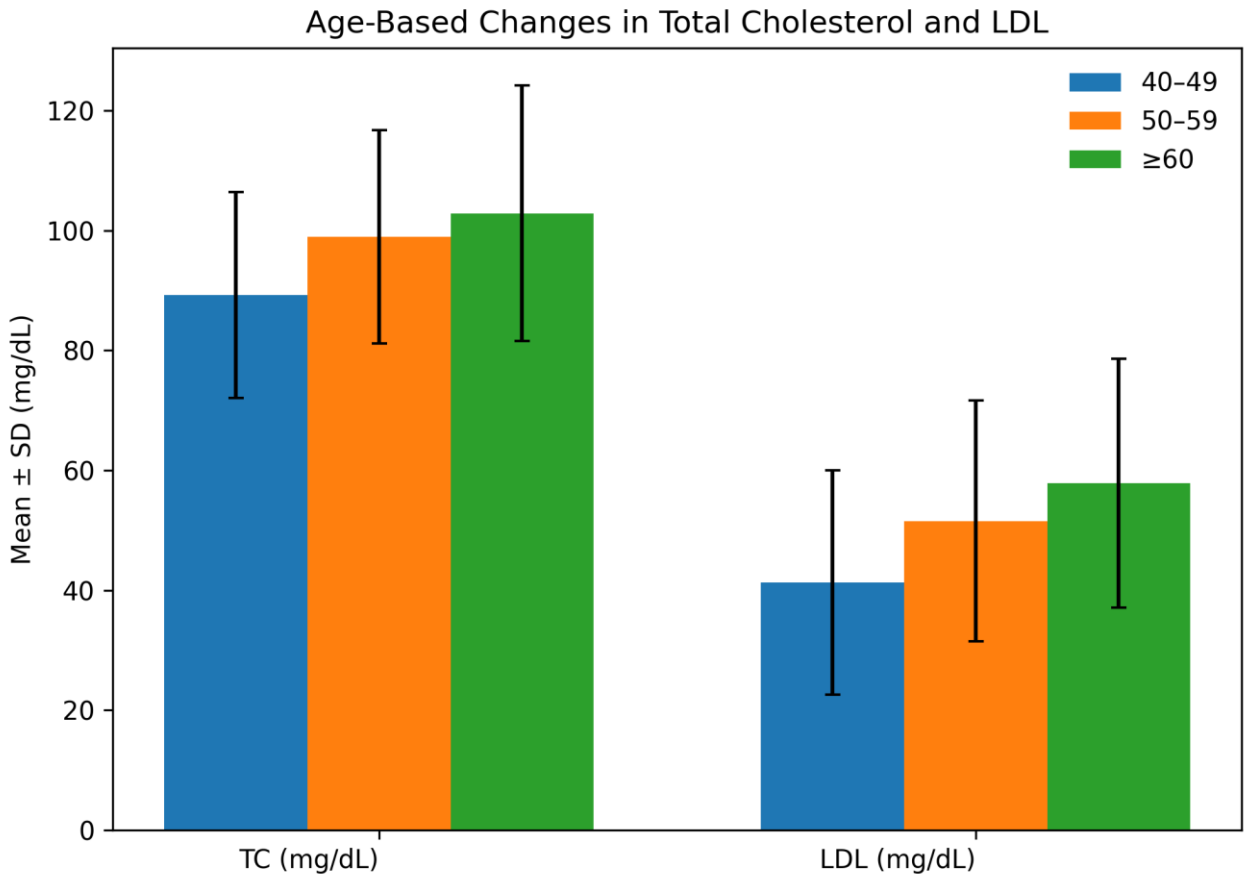


***Figure 4. Age-based comparison of total cholesterol and LDL levels.*** *Total cholesterol and LDL levels increased with advancing age, with the highest values observed among participants aged ≥60 years. Data are presented as mean ± SD. LDL: low-density lipoprotein.*

### *3.6 Gender-Based Comparison of Adipokines and Metabolic Markers*

The overall analyses of the gender factors showed significant differences for adipokines and metabolic markers between the groups. The level of male serum resistin (4.58 ± 1.28 µg/L) was significantly lower compared to that in females (5.32 ± 1.08 µg/L), and the level of male serum visfatin (12.87 ± 3.78 µg/L) was significantly lower compared to that in females (15.84 ± 3.51 µg/L).

On the contrary, males had significantly higher levels of apelin (454.93 ± 49.24 pg/mL) compared to that in females (390.60 ± 11.49 pg/mL). Moreover, insulin, 2h-BG, FBG, and HbA1c levels were significantly higher in men compared to women. Nevertheless, the presence of women was noted in the HC group only.

***Table 6. Overall gender-based comparison***

| Marker | Male n=165 | Female n=15 | p-value |
|---|---|---|---|
| Age, years | 53.29 ± 6.36 | 52.20 ± 5.81 | 0.499 |
| Resistin, µg/L | 4.58 ± 1.28 | 5.32 ± 1.08 | 0.024 |
| Visfatin, µg/L | 12.87 ± 3.78 | 15.84 ± 3.51 | 0.006 |
| Apelin, pg/mL | 454.93 ± 49.24 | 390.60 ± 11.49 | <0.001 |
| Insulin, mU/L | 12.36 ± 2.40 | 8.81 ± 0.96 | <0.001 |
| 2-hour BG, mg/dL | 188.47 ± 74.11 | 98.53 ± 10.84 | <0.001 |
| FBG, mg/dL | 116.92 ± 21.45 | 92.27 ± 8.42 | <0.001 |
| HbA1c, % | 6.41 ± 1.00 | 5.53 ± 0.29 | <0.001 |
| Total cholesterol, mg/dL | 97.27 ± 18.91 | 90.13 ± 17.49 | 0.151 |
| Triglycerides, mg/dL | 43.92 ± 21.44 | 33.27 ± 32.50 | 0.232 |
| HDL, mg/dL | 38.41 ± 6.43 | 40.20 ± 5.47 | 0.248 |
| LDL, mg/dL | 50.08 ± 20.52 | 43.28 ± 20.22 | 0.230 |
| VLDL, mg/dL | 8.78 ± 4.29 | 6.65 ± 6.50 | 0.232 |

### *3.7 Gender-Based Comparison Within Healthy Controls*

A gender-specific comparison was also performed among the healthy control (HC) subjects since females were present only among this category of subjects. In this subset of subjects, no gender difference was observed with respect to adipokines and metabolic parameters except apelin, which was higher in females (390.60 ± 11.49 pg/mL) than males (383.67 ± 10.22 pg/mL) although not reaching the level of statistical significance (p = 0.050).

***Table 7. Gender-based comparison within healthy controls***

| Marker | Male HC n=45 | Female HC n=15 | p-value |
|---|---|---|---|
| Age, years | 52.16 ± 6.43 | 52.20 ± 5.81 | 0.980 |
| Resistin, µg/L | 5.46 ± 1.01 | 5.32 ± 1.08 | 0.664 |
| Visfatin, µg/L | 14.76 ± 3.32 | 15.84 ± 3.51 | 0.305 |
| Apelin, pg/mL | 383.67 ± 10.22 | 390.60 ± 11.49 | 0.050 |
| Insulin, mU/L | 9.08 ± 0.91 | 8.81 ± 0.96 | 0.363 |
| 2-hour BG, mg/dL | 100.42 ± 5.89 | 98.53 ± 10.84 | 0.528 |
| FBG, mg/dL | 91.96 ± 4.98 | 92.27 ± 8.42 | 0.894 |
| HbA1c, % | 5.42 ± 0.33 | 5.53 ± 0.29 | 0.258 |
| Total cholesterol, mg/dL | 93.80 ± 15.37 | 90.13 ± 17.49 | 0.477 |

| | | | |
|---|---|---|---|
| Triglycerides, mg/dL | 37.93 ± 20.29 | 33.27 ± 32.50 | 0.607 |
| HDL, mg/dL | 42.49 ± 5.73 | 40.20 ± 5.47 | 0.178 |
| LDL, mg/dL | 43.72 ± 18.29 | 43.28 ± 20.22 | 0.941 |
| VLDL, mg/dL | 7.59 ± 4.06 | 6.65 ± 6.50 | 0.607 |

### *3.8 BMI-Based Comparison of Adipokines and Metabolic Markers*

The participants were further classified according to their BMI, categorizing them into normal-BMI, overweight, and obese individuals. Adipokine composition, glycemia, and lipids showed differential characteristics when analyzed according to BMI category.

Resistin levels were found to be significantly lower in overweight and obese participants compared to normal-BMI subjects; resistin was 5.10 ± 1.02 µg/L in normal-BMI subjects, 4.68 ± 1.13 µg/L in overweight, and 4.05 ± 1.25 µg/L in obese participants ($p = 0.002$). Similarly, visfatin also showed a decreasing trend with an increase in BMI, from 14.50 ± 3.20 µg/L in normal-BMI patients to 13.20 ± 3.65 µg/L in overweight and 11.60 ± 3.85 µg/L in obese individuals ($p < 0.001$). On the contrary, apelin levels increased significantly with BMI from 420.30 ± 45.80 pg/mL in normal-BMI subjects to 451.60 ± 52.40 pg/mL in overweight and 482.90 ± 55.10 pg/mL in obese participants ($p < 0.001$). This can be observed in figure 7.

Insulin, 2-hour plasma glucose, fasting blood glucose, and HbA1c values showed a progressive increase with an increase in BMI category. Furthermore, the same trend was also seen with lipids: an increase in total cholesterol, triglycerides, LDL, and VLDL levels and a decrease in HDL (from 41.20 ± 5.90 mg/dL in the normal-BMI category to 36.40 ± 6.35 mg/dL in the obese group). Figure 5 shows the relative differences in these metabolites according to BMI.

***Table 8. BMI-based comparison of adipokines and metabolic markers***

| Marker | Normal BMI | Overweight | Obese | p-value |
|---|---|---|---|---|
| Resistin, µg/L | 5.10 ± 1.02 | 4.68 ± 1.13 | 4.05 ± 1.25 | 0.002 |
| Visfatin, µg/L | 14.50 ± 3.20 | 13.20 ± 3.65 | 11.60 ± 3.85 | <0.001 |
| Apelin, pg/mL | 420.30 ± 45.80 | 451.60 ± 52.40 | 482.90 ± 55.10 | <0.001 |
| Insulin, mU/L | 10.30 ± 1.90 | 12.20 ± 2.35 | 13.80 ± 2.20 | <0.001 |
| 2-hour BG, mg/dL | 145.20 ± 68.50 | 181.60 ± 76.20 | 218.40 ± 82.10 | <0.001 |
| FBG, mg/dL | 102.80 ± 18.10 | 115.30 ± 22.40 | 126.70 ± 24.60 | <0.001 |
| HbA1c, % | 5.90 ± 0.78 | 6.35 ± 0.95 | 6.85 ± 1.05 | <0.001 |
| Total cholesterol, mg/dL | 91.40 ± 16.80 | 96.70 ± 18.50 | 104.20 ± 19.70 | 0.004 |
| Triglycerides, mg/dL | 38.80 ± 18.60 | 43.90 ± 21.50 | 49.60 ± 24.10 | 0.032 |
| HDL, mg/dL | 41.20 ± 5.90 | 38.50 ± 6.15 | 36.40 ± 6.35 | <0.001 |
| LDL, mg/dL | 43.20 ± 18.30 | 50.10 ± 20.20 | 58.60 ± 21.10 | 0.001 |
| VLDL, mg/dL | 7.76 ± 3.72 | 8.78 ± 4.30 | 9.92 ± 4.82 | 0.032 |

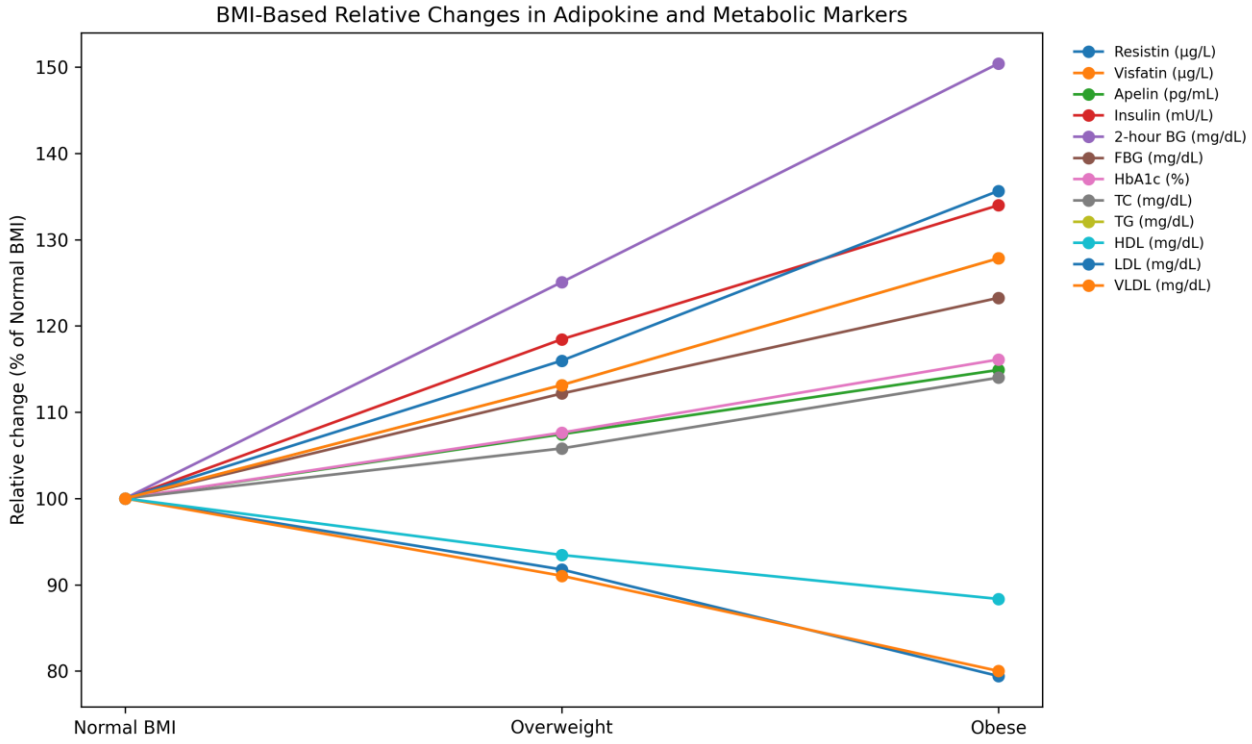


***Figure 5. BMI-based relative changes in adipokine and metabolic markers.*** *The figure illustrates relative changes in adipokines, glycemic markers, and lipid profile parameters across normal BMI, overweight, and obese categories. Increasing BMI was associated with higher apelin, insulin, glucose markers, HbA1c, total cholesterol, triglycerides, LDL, and VLDL, along with lower resistin, visfatin, and HDL levels. BMI: body mass index.*

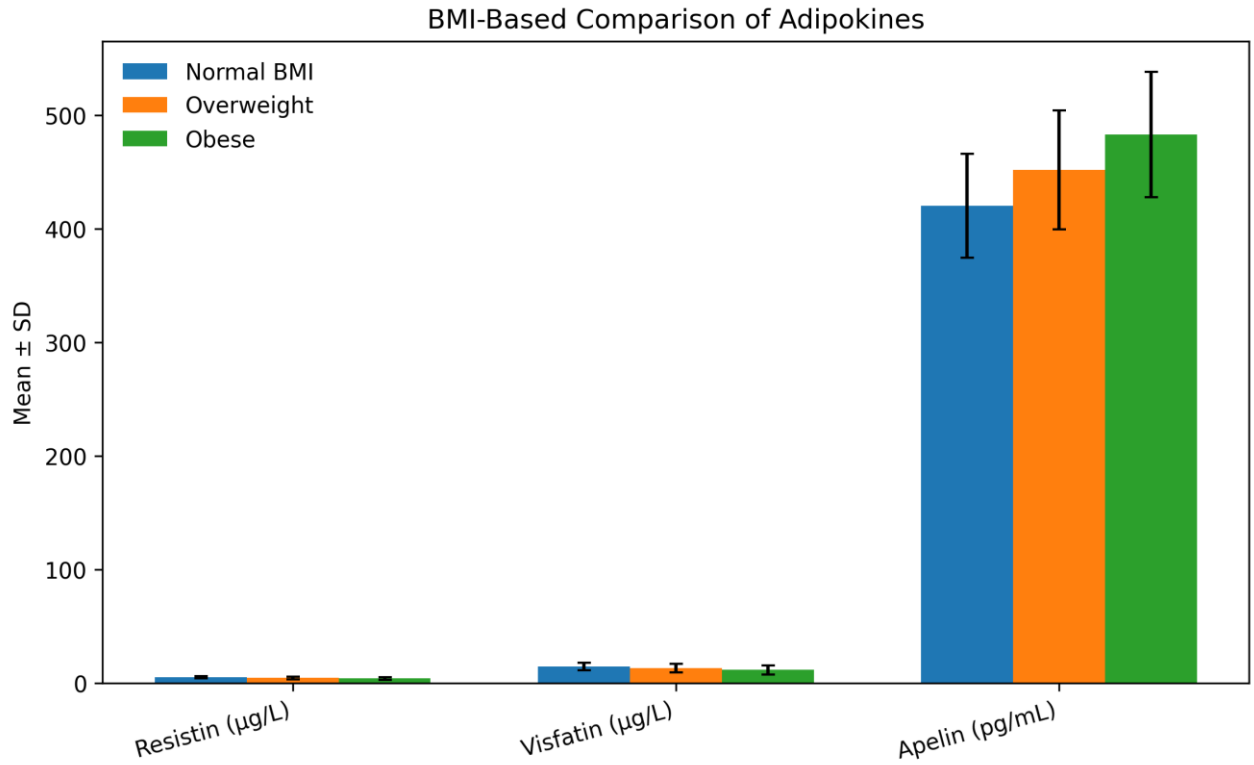


***Figure 7. BMI-based comparison of serum adipokine levels.*** *Serum resistin and visfatin levels declined progressively with increasing BMI category, while serum apelin levels increased from normal BMI to overweight and obese participants. Data are presented as mean ± SD. BMI: body mass index.*

### *3.9 Correlation Analysis*

Correlations were established between adipokines and metabolic factors. In the IGT group, the following correlations were observed for apelin: a positive correlation between apelin and resistin (r = 0.259), and a positive correlation between apelin and insulin (r = 0.339), as well as a negative correlation with HDL levels (r = −0.222). In the T2DM group, age demonstrated positive correlations with total cholesterol (r = 0.548) and LDL (r = 0.540). Also, positive correlations were observed for resistin with total cholesterol (r = 0.355) and LDL (r = 0.344). On the other hand, the negative correlations included those between age and visfatin (r = −0.258), and between apelin and insulin (r = −0.290) as well as 2-hour plasma glucose levels (r = −0.259).

***Table 9. Main significant correlations among adipokines and metabolic markers***

| Group | Correlation | r-value | Direction |
|---|---|---|---|
| IGT | Apelin with resistin | 0.259 | Positive |
| IGT | Apelin with insulin | 0.339 | Positive |
| IGT | Apelin with HDL | −0.222 | Negative |
| T2DM | Age with total cholesterol | 0.548 | Positive |
| T2DM | Age with LDL | 0.540 | Positive |
| T2DM | Total cholesterol with resistin | 0.355 | Positive |
| T2DM | Resistin with LDL | 0.344 | Positive |
| T2DM | Age with visfatin | −0.258 | Negative |
| T2DM | Insulin with apelin | −0.290 | Negative |
| T2DM | 2-hour BG with apelin | −0.259 | Negative |

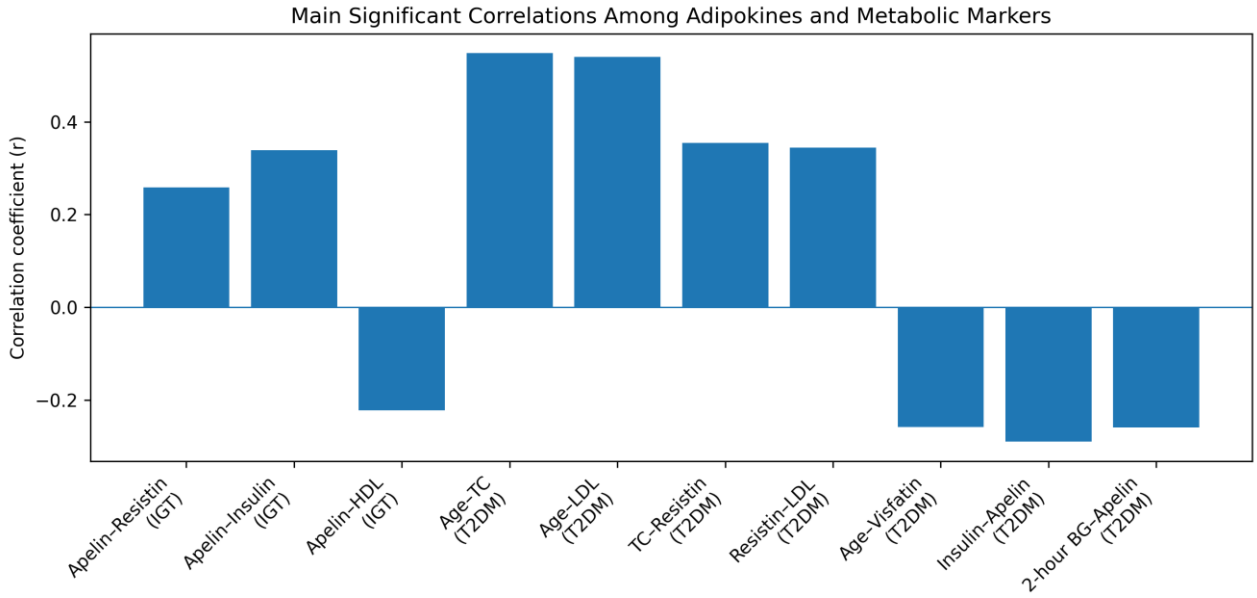


***Figure 6. Main correlation coefficients among adipokines and metabolic markers.*** *The figure summarizes the principal positive and negative correlations observed among adipokines, age, glycemic markers, and lipid profile parameters. Positive correlations were observed for apelin with resistin and insulin in the impaired glucose tolerance group, and for age with total cholesterol and LDL in the type 2 diabetes mellitus group. Negative correlations were observed for apelin with HDL, insulin, and 2-hour blood glucose, and for age with visfatin.*

All in all, the results show significant changes in the levels of adipokines and metabolic factors among the tested groups. Resistin and visfatin showed progressive reductions from HC through IGT to T2DM subjects while apelin showed significant increase from one group to the next as displayed in Figure 1. Glycemic factors showed progressive deterioration in terms of the levels of FBG, 2 hours post glucose, and HbA1c that were highest in the T2DM group as illustrated in Figure 2. There was significant alteration in lipid factors such as elevated levels of total cholesterol and LDL cholesterol in T2DM subjects as indicated in Figure 3.

Age-related analysis showed significant elevation in the levels of total cholesterol and LDL cholesterol with advanced ages as illustrated in Figure 4. The results for BMI-related analysis indicate an association between high BMI, adipokine imbalance, hyperglycemia, insulin, and lipids as reflected in Figures 5 and 7. There was also a correlation between adipokines and metabolism as displayed in Figure 6. Overall, the results reveal that glucose tolerance, age, gender, and BMI might jointly affect adipokine expression and metabolism among Iraqis.

## 4. Discussion

The present study assessed serum resistin, visfatin, and apelin levels in adults with normal glucose tolerance, impaired glucose tolerance, and type 2 diabetes mellitus. The study also focused on differences in these variables based on participant age, gender, and BMI categories. Main results revealed progressive decreases in resistin and visfatin, and a progressive increase in apelin in the study population. Parallel changes in the glycemic and lipid profile parameters indicated worsening metabolic status, especially in T2DM. The above results agree well with accumulating literature on the involvement of adipokines in insulin resistance, glucose dysregulation, obesity-related metabolic disturbance, and progression of diabetes mellitus [1,2].

Among the main results obtained, the most striking one was related to the progressive decrease in the serum resistin level between healthy controls and IGT, and especially between IGT and T2DM groups. As a pro-inflammatory adipokine commonly associated with metabolic dysfunction and insulin resistance, resistin may show variable changes in different populations depending on numerous factors, including kidney function, insulin resistance, inflammatory status, obesity degree, and medication. In accordance with the above data, recent studies have demonstrated relationships between resistin and insulin resistance, inflammation, renal function, and cardiovascular risk in T2DM patients [11,15]. Therefore, the current finding of decreased resistin levels in T2DM cannot necessarily mean progression from health to IGT to T2DM.

It has been shown that adipokine profiles may differ between obese and non-obese T2DM patients based on their metabolic phenotypes [17]. In addition, alterations in resistin concentration have been described in individuals with impaired fasting glucose, IGT, T2DM, and metabolic syndrome [16]. The results of positive correlation between resistin and total cholesterol and LDL are in line with existing data on the relationship between resistin-related indices and cardiovascular risk in T2DM patients [11].

As another adipokine of interest, visfatin also exhibited a progressive decline in serum concentration among healthy controls, IGT, and T2DM patients. This result indicates possible alteration of the studied adipokine during the transition from normal glucose tolerance to IGT and T2DM. Visfatin has been described as an adipokine involved in glucose metabolism, inflammation, and pathways of insulin action. In addition, visfatin has been proposed as a possible biomarker in obesity and T2DM [3]. Nevertheless, the behavior of visfatin in T2DM appears to be controversial. For example, increased visfatin levels have been detected in obese and diabetic patients, while reduced and variable concentrations were found depending on dietary intake, endocrine disorders, and inflammatory status [5,9,10].

Interestingly, the negative correlation between visfatin and age among T2DM participants may suggest age-related changes in visfatin regulation. Previous research has shown that visfatin is modulated by carbohydrate and polyunsaturated fatty acid intake and is linked with vascular risk in patients with T2DM [5,10]. Moreover, alterations in visfatin levels have been found in T2DM patients with thyroid dysfunction [9,14]. Thus, the current

decrease in visfatin serum concentration may be caused by different metabolic processes and interactions among obesity, T2DM, aging, and other variables.

Contrary to the results found in resistin and visfatin groups, apelin showed a progressive increase in concentration from healthy controls to IGT, and especially T2DM. Such a finding may indicate a compensatory response to insulin resistance, hyperglycemia, and vascular/metabolic stress. Apelin has recently attracted great attention as an adipokine involved in glucose uptake and utilization, insulin sensitivity, metabolic functions, and cardiovascular regulation [4,7]. Experimental and clinical investigations have also demonstrated the involvement of apelin in glucose metabolism by improving insulin sensitivity in T2DM [4]. The presence of positive correlations between apelin and impaired glucose regulation has also been revealed, and these results indicate the involvement of apelin in earlier pathophysiological processes before the development of T2DM [7,16].

It should be noted that a positive correlation between apelin and insulin among IGT patients may suggest the close relationship between apelin and compensatory hyperinsulinemia. Indeed, in IGT patients, the increased insulin level often serves as a compensatory factor for insulin resistance. Apelin may be involved in metabolic adjustment aimed at restoring glucose utilization. On the other hand, the negative correlation between apelin and insulin in the T2DM group may suggest alterations in compensatory processes after the onset of diabetes. Therefore, the results indicate variable behavior of adipokines in various stages of metabolic disorders [2,16].

The results on glycosylated hemoglobin and fasting/2-hr blood glucose are predictable and logically consistent with metabolic disorders. In the order of healthy controls, IGT, and T2DM, each group demonstrated increased glucose values, confirming the gradually worsening glycemic status. Similarly, insulin levels were maximal in IGT patients and were indicative of compensation due to insulin resistance in this case [2]. Thus, these results confirm the significance of the IGT condition, which requires prompt diagnosis.

The analysis of lipid profile parameters showed increased total cholesterol and LDL, and decreased HDL in the T2DM group compared with the other two groups. This means that changes in the lipid profile begin with IGT and progress towards T2DM. Lipid disorders have been considered an important component of diabetes-related cardiometabolic complications that are closely linked with insulin resistance, obesity, hepatocyte metabolism, and inflammation [6,18]. Therefore, the current relationships between adipokines and lipid parameters suggest that adipocytokines may have a broader impact on metabolic processes, including those related to glucose and lipid dysregulation [6,12].

Based on age differences in adipokine concentration, it has been demonstrated that serum concentrations of total cholesterol and LDL increased with advancing age. Patients older than 60 showed the highest cholesterol/LDL levels. This fact is important since aging is associated with the development of insulin resistance, altered lipid metabolism, cardiovascular problems, and changed body composition. Even though there were no significant differences in adipokine concentration in the current age analysis, the above finding is clinically relevant. It is important to note that T2DM and obesity-related complications tend to be more severe in older patients [2,18].

Analysis of gender-specific changes in adipokine levels and metabolic markers revealed several statistically significant differences between men and women. Specifically, men had significantly decreased serum concentrations of resistin and visfatin. At the same time, men had increased levels of apelin, insulin, glucose markers, and HbA1c in comparison with women. This finding needs to be interpreted carefully because women appeared to be absent from groups of IGT and T2DM. That is, the above gender differences may indicate imbalances among disease groups rather than real gender-related differences. After analysis of healthy controls, it became clear that apart from a marginal elevation of apelin, no significant differences occurred between men and women. Literature reviews suggested variable relationships between adipokines and gender based on different metabolic factors [13,17].

BMI-based analysis of metabolic disorders demonstrated significant worsening with increasing BMI. Serum concentrations of resistin and visfatin were found to decrease in the normal, overweight, and obese groups, whereas the apelin concentration progressed accordingly. In addition, obesity was accompanied by the increased serum concentrations of insulin, glucose markers, HbA1c, total cholesterol, triglycerides, LDL, and VLDL, as well as reduced levels of HDL. In other words, obesity is known to be highly associated with metabolic dysregulation, including changes in adipokine concentration [12,18]. New developments in this area show that adipokines may be used as valuable targets in obesity and T2DM therapy [12].

The increase in apelin concentration with BMI may reflect a compensatory response to obesity-associated metabolic disorders. The decrease in visfatin and resistin concentrations at the increase of BMI may seem to differ from published results, although the adipokine behavior may be quite variable, depending on many factors, including obesity stage, inflammation degree, obesity type, and pharmacotherapy [3,5,10,17]. It is worth noting that adipokine alterations have been detected in obese and non-obese T2DM patients, suggesting possible differences in the metabolic effect on adipokines [17]. Metformin has also been shown to affect adipokine concentrations in recent investigations [20].

Overall, the current findings are clinically valuable from the perspective of further evaluation of adipokine dysregulation as a component of metabolic disturbance. Namely, the assessment of resistin, visfatin, and apelin as adipokines should not be performed separately but together with other variables, such as insulin, glucose markers, HbA1c, and lipid profile. The current findings are consistent with recent research emphasizing the contribution of adipokines to metabolic dysfunction complications and possibilities to assess and intervene with adipokines as biomarkers/therapeutic targets [6,12,19]. Incretin-based and metabolic therapies continue shaping the management of obesity and T2DM, and adipokine studies are expected to provide additional insights in the future [19,20].

From the perspective of clinical practice in Iraq, the results are valuable because data on the simultaneous assessment of resistin, visfatin, and apelin in adults with normal glucose tolerance, IGT, and T2DM are scarce in this population. Published regional studies have already indicated the association between adipokines and obesity/T2DM [8]. The current findings complement this literature by evaluating multiple adipokines along with other glycemic and lipid markers, while also accounting for age, gender, and BMI differences. These results will hopefully contribute to future diabetes prevention efforts.

There are several limitations to the current study. First, the use of a cross-sectional design precludes any conclusions about causality. Second, the gender difference among groups prevented proper analysis of gender-specific differences. Third, BMI-based analysis should be interpreted with caution and needs to be verified based on actual measurements of height and weight. Finally, potential confounders, including medication use, nutritional status, physical exercise, renal and thyroid function, and inflammatory state, may affect the adipokine profile. As shown in recent publications, adipokine concentration may be modified by treatment, diet, exercise, thyroid dysfunction, renal function, and inflammation [1,9,10,15,20].

Despite the above limitations, the present study offers interesting results on the adipokine differences among patients with various degrees of metabolic disorders. Analysis of multiple adipokines and accompanying parameters, including insulin, glucose markers, lipid profile, age, gender, and BMI, adds value to this research. It would be useful to perform a future study with a large sample size, equal gender ratio, verified BMI data, inclusion of inflammatory parameters, evaluation of renal and thyroid function, and follow-up of IGT patients to evaluate apelin and resistin/visfatin changes prior to T2DM onset.

To conclude, the present study shows that adipokines resistin, visfatin, and apelin are significantly different between individuals with normal glucose tolerance, IGT, and T2DM. The decreases in resistin and visfatin, together with a progressive increase in apelin, indicate particular adipokine imbalance in relation to worsening glucose metabolism. Older age correlated with lipid disorder, whereas increased BMI accounted for adipokine

alterations, insulin, glucose markers, and dyslipidemia. These findings suggest the potential clinical significance of adipokines for evaluating metabolic dysfunctions in Iraqi adults.

## 5. Conclusion

The findings of the current study confirmed significant changes in serum adipokine levels among healthy controls, patients with impaired glucose tolerance, and patients with type 2 diabetes mellitus. It was discovered that serum levels of resistin and visfatin were significantly reduced while serum apelin levels were significantly elevated among patients with type 2 diabetes compared to healthy controls and impaired glucose tolerance patients. Therefore, metabolic dysfunction and adipokine imbalance can be interrelated, and the progression of glucose metabolism disturbance can be associated with changes in adipokine levels.

Furthermore, glycemic factors such as fasting blood glucose, 2-hours blood glucose, and HbA1c were significantly higher for patients with type 2 diabetes, and insulin serum levels were significantly elevated among patients with impaired glucose tolerance, which can be explained as the result of compensatory hyperinsulinemia. Metabolic disorder and dysregulation associated with lipids were discovered among patients with type 2 diabetes, as their total cholesterol and LDL values were significantly increased in comparison with other study groups.

Age-based analysis revealed that with the increase of age total cholesterol and LDL values become significantly higher. Gender-based results revealed some differences in selected adipokines and metabolic parameters; yet, these results require verification since patients did not have similar genders. Finally, analysis based on the BMI revealed that metabolic dysfunction is intensified with increasing BMI, since serum levels of insulin, glucose markers, HbA1c, total cholesterol, triglycerides, LDL, and VLDL become significantly higher, while serum levels of resistin, visfatin, and HDL become significantly lower.

Therefore, the presented findings can prove that resistin, visfatin, and apelin can be used as complementary biomarkers of metabolic dysfunction among individuals with impaired glucose tolerance and type 2 diabetes mellitus. The obtained results also show the necessity to pay attention to such variables as age, gender, and BMI in terms of evaluating changes associated with metabolic disturbance. Future research with an equal number of males and females, confirmed BMI measurement, and longitudinal follow-up is highly needed.

## Declarations

***Data Availability Statement:*** The datasets generated and/or analyzed during the current study are available from the corresponding author upon reasonable request. Due to privacy and ethical considerations, individual participant data are not publicly available.

***Conflict of Interest:*** The author declares that there is no conflict of interest regarding the publication of this study.

***Funding:*** This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

***Ethical Approval:*** The study was conducted in accordance with the ethical principles for medical research involving human participants. All procedures involving human participants were performed according to institutional and ethical research standards.

***Informed Consent:*** Informed consent was obtained from all participants before sample collection and data analysis. Participant confidentiality was maintained throughout the study.

***Consent for Publication:*** Not applicable, as the manuscript does not contain identifiable personal information, images, or individual participant details.

***Competing Interests:*** The author declares no competing interests.

**Abbreviations**

BMI: Body mass index
FBG: Fasting blood glucose
HbA1c: Glycated hemoglobin
HC: Healthy control
HDL: High-density lipoprotein
IGT: Impaired glucose tolerance
LDL: Low-density lipoprotein
T2DM: Type 2 diabetes mellitus
TC: Total cholesterol
TG: Triglycerides
VLDL: Very-low-density lipoprotein

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