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| RESEARCH ARTICLE

Relationship between Adipokines and Clinical Variables in Type 2 Diabetes Mellitus

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| ABSTRACT
Adipokine secretion play a significant role in the onset of insulin resistance and diminished insulin activity in type 2 diabetes associated with obesity. Accumulation of excess body fat, especially visceral adiposity, is strongly correlated with a higher risk of cardiovascular complications due to underlying metabolic disturbances. The objective of the study was to examine the relationship between visceral fat-associated adipokines with clinical variables in central obese type 2 diabetic patients. Serum adipokine concentrations were determined using Enzyme-Linked Immunosorbent Assay (ELISA), while glycated hemoglobin (HbA1c) levels were assessed through High-Performance Liquid Chromatography (HPLC). All clinical parameters were evaluated following standardized procedures. The results indicate that the adipokines are significantly related ($p < 0.05$) to weight, body mass index (BMI) and waist circumference (WC) with varying degrees in central obese type 2 diabetic subjects. The adipokine levels are associated with clinical variables to a varying degree in central obese type 2 diabetic subjects. The effective target to control visceral fat-associated adipokines with clinical variables reduces body weight, obesity, visceral fat and cardiovascular risk in type 2 diabetes.

| KEYWORDS

Adipokines, Adiponectin, FABP4, RBP4, Visfatin

| ARTICLE INFORMATION

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Introduction

Type 2 Diabetes Mellitus (T2DM) is a growing global health concern, characterized by impaired insulin production and peripheral insulin resistance. Its prevalence is strongly linked to obesity, unhealthy diet, and physical inactivity, with significant increases projected worldwide and particularly in the Middle East [1, 2]. The development of T2DM involves a complex interplay of genetic, environmental, and metabolic factors [3]. Adipose tissue, especially visceral fat, plays a critical role in the pathophysiology of T2DM through the secretion of adipokines, bioactive cytokines that regulate various metabolic pathways [4]. Dysregulation of these adipokines is closely associated with insulin resistance and the progression of T2DM and its complications [3, 4].

Obesity is the most substantial risk factor for T2DM, leading to metabolic irregularities and insulin resistance [3].

Central obesity, specifically excessive intra-abdominal fat, is a more reliable indicator of cardiovascular risk in T2DM than BMI alone [5]. Waist circumference (WC) (≥94 cm for males, ≥80 cm for females) and waist-to-hip ratio (≥0.90 for males, ≥0.85 for females) are better estimates of visceral fat distribution and correlate well with cardiovascular risk mortality, independent of BMI [5]. The accumulation of fat, particularly in the visceral region, worsens insulin resistance through inflammatory pathways and the dysregulation of adipokines [3]. Weight reduction is a primary therapeutic target to improve insulin sensitivity and prevent T2DM in obese individuals [3]. Adiponectin levels are inversely related to BMI and are decreased in obese subjects, contributing to low-grade inflammation and metabolic disorders [6]. Retinol-binding protein 4 (RBP4) levels are positively associated with BMI, as well as serum lipid levels, blood pressure, and waist-to-hip ratio, highlighting its role in obesity-related metabolic dysfunction [7]. Excess adiposity is linked with an increased cardiovascular risk due to blood pressure changes, alterations in lipid metabolism, and poorly controlled blood glucose [8, 9]. Increased RBP4 levels are associated with blood pressure [7]. Some receptors related to obesity link oxidative stress and inflammation, contributing to hypertension in obese T2DM subjects [3].

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Adiponectin protects against insulin resistance, diabetes, obesity, and cardiovascular disease [6]. Lower levels are observed in obese individuals and are inversely proportional to inflammatory markers like interleukin-6 and tumor necrosis factor-alpha. It enhances fatty acid oxidation and promotes insulin sensitivity [6]. Fatty-acid binding protein 4 (FABP4) is strongly linked to inflammation, insulin resistance, and T2DM. Its prolonged expression is also connected with the differentiation of monocytes to macrophages, which can contribute to chronic inflammation [10].

Retinol-binding protein 4 (RBP4) is involved in the pathogenesis of insulin resistance and T2DM, particularly in central obesity [7]. Elevated RBP4 levels correlate with increased serum lipid levels, BMI, blood pressure, and waist-to-hip ratio, and are associated with inflammatory markers such as interleukin-6 and tumor necrosis factor-alpha [7]. Visfatin plays a significant role in metabolic disorders, insulin resistance, obesity, and diabetes [11, 12, 13]. It exhibits proinflammatory characteristics, contributing to vascular inflammation and endothelial dysfunction, and elevated serum visfatin levels are associated with carotid atherosclerosis in T2DM [12, 13].

The objective of the study was to examine the relationship between visceral fat-associated adipokines with clinical variables in central obese type 2 diabetic patients.

Materials and Methods

The study included 100 participants, aged 30-60 years, recently diagnosed with T2DM at Thumbay Hospital, Ajman, UAE. The study was approved by the Institutional Review Board at Gulf Medical University, UAE. A crucial aspect of the study design was the inclusion of only individuals with central obesity with the overweight (25.0 to 29.9 kg/m^2) or the obesity (≥30.0 kg/m^2). The study specifically recruited those newly diagnosed with T2DM. To ensure a focused analysis, individuals with Type 1 diabetes, diabetic ketoacidosis, gestational diabetes, renal or liver disease, or a normal BMI were excluded.

Adipokine levels (RBP4, FABP4, Visfatin, and Adiponectin) were analyzed from blood samples, providing insight into adipose tissue function. BMI was calculated using the formula of weight in kilograms divided by the square of height in meters. This calculation was fundamental for determining eligibility, ensuring all participants met the criteria for overweight or obesity. WC was taken to specifically quantify central obesity, recognized as a significant risk factor for metabolic and cardiovascular complications in T2DM. Blood Pressure was assessed using a sphygmomanometer.

Beyond these clinical measurements, the study also monitored blood glucose control through Hemoglobin A1c (HbA1c), Fasting Plasma Glucose (FPG), and 2-hour Postprandial Blood Glucose (PPBG) levels. Information regarding age, lifestyle and family history was gathered through a questionnaire.

Results

Table 1 presents the correlation between RBP4 levels and various clinical parameters in individuals with type 2 diabetes mellitus (T2DM). A statistically significant moderate positive correlation was observed between RBP4 and body weight in the total study population ($\rho = 0.475$, $p < 0.01$). Notably, RBP4 demonstrated a very strong and significant positive correlation with body mass index (BMI) in the overall cohort ($\rho = 0.869$, $p < 0.01$). Subgroup analysis revealed that this relationship remained strong among overweight individuals ($\rho = 0.753$, $p < 0.01$) and moderate in obese participants ($r = 0.558$, $p < 0.01$). Additionally, a significant but weak positive correlation was found between RBP4 and waist circumference (WC) in the total sample ($\rho = 0.340$, $p < 0.01$). The findings indicate a significant and consistent positive association between circulating RBP4 levels and measures of adiposity, particularly BMI, in individuals with type 2 diabetes mellitus, underscoring its potential role as a biomarker of obesity-related metabolic dysfunction.

Variable	Total		Overweight Type 2 Diabetes		Obese Type 2 Diabetes	
	Correlation coefficient	p	Correlation coefficient	p	Correlation coefficient	p
Weight (Kg)	0.475**	<0.001	0.244	0.088	-0.096	0.508
BMI (kg/m^2)	0.869**	<0.001	0.753**	<0.001	0.558**	<0.001
WC (cm)	0.340**	0.001	0.193	0.180	0.134	0.354
Systolic BP (mmHg)	0.024	0.812	0.132	0.362	-0.180	0.212
Diastolic BP (mmHg)	0.043	0.674	-0.037	0.799	-0.003	0.984

Data are represented as spearman rank correlation coefficient (ρ) or pearson correlation coefficient (r)
*Correlation is significant at 0.05 level (2-tailed).
**Correlation is significant at 0.01 level (2-tailed).
BMI: Body Mass Index, WC: Waist circumference, BP: Blood Pressure.

Table 1. Relationship between retinol binding protein 4 and clinical variables

Table 2 presents the correlation between FABP4 levels and various clinical parameters at baseline. In the overall study population, FABP4 exhibited a significant moderate positive correlation with body weight (ρ = 0.463, p < 0.01). A very strong positive correlation was observed between FABP4 and BMI in the total cohort (ρ = 0.865, p < 0.01). Subgroup analysis revealed that FABP4 maintained a strong positive association with BMI in overweight individuals (r = 0.753, p < 0.01) and a moderate positive correlation in obese participants (ρ = 0.594, p < 0.01). Additionally, FABP4 showed a significant weak positive correlation with waist circumference (WC) in the overall group (ρ = 0.367, p < 0.01).

Variable	Total		Overweight Type 2 Diabetes		Obese Type 2 Diabetes	
	Correlation coefficient	p	Correlation coefficient	p	Correlation coefficient	p
Weight (Kg)	0.463**	<0.001	0.130	0.370	-0.063	0.662
BMI (kg/m^2)	0.865**	<0.001	0.753**	<0.001	0.594**	<0.001
WC (cm)	0.367**	<0.001	0.160	0.266	0.139	0.335
Systolic BP (mmHg)	0.039	0.701	0.260	0.068	-0.238	0.096
Diastolic BP (mmHg)	0.022	0.830	-0.02	0.890	0.064	0.660
Data are represented as spearman rank correlation coefficient (ρ) or pearson correlation coefficient (r) *Correlation is significant at 0.05 level (2-tailed). **Correlation is significant at 0.01 level (2-tailed). BMI: Body Mass Index, WC: Waist circumference, BP: Blood Pressure.						

Table 2. Relationship between fatty acid binding protein 4 and clinical variables

Table 3 presents the association between visfatin levels and clinical variables at baseline. A statistically significant moderate positive correlation was observed between visfatin and body weight in the overall study population (ρ = 0.492, p < 0.01). Visfatin demonstrated a highly significant and very strong positive correlation with BMI in both the total cohort (ρ = 0.892, p < 0.01) and the overweight subgroup (r = 0.876, p < 0.01). In obese individuals, visfatin maintained a significant strong positive association with BMI (ρ = 0.615, p < 0.01). Additionally, a weak but statistically significant positive correlation was found between visfatin and waist circumference (WC) in the overall study group (ρ = 0.353, p < 0.01).

Variable	Total		Overweight Type 2 Diabetes		Obese Type 2 Diabetes	
	Correlation coefficient	p	Correlation coefficient	p	Correlation coefficient	p
Weight (Kg)	0.492**	<0.001	0.217	0.131	-0.054	0.710
BMI (kg/m^2)	0.892**	<0.001	.876**	<0.001	.615**	<0.001
WC (cm)	0.353**	<0.001	0.234	0.102	0.081	0.577
Systolic BP (mmHg)	-0.001	0.992	0.128	0.375	-0.220	0.124
Diastolic BP (mmHg)	0.043	0.673	-0.033	0.823	-0.018	0.902
Data are represented as spearman rank correlation coefficient (ρ) or pearson correlation coefficient (r) *Correlation is significant at 0.05 level (2-tailed). **Correlation is significant at 0.01 level (2-tailed). BMI: Body Mass Index, WC: Waist circumference, BP: Blood Pressure.						

Table 3. Relationship between visfatin and clinical variables

Table 4 presents the baseline correlations between adiponectin levels and clinical variables. A statistically significant moderate negative correlation was observed between adiponectin and body weight in both the overall cohort (ρ = -0.595, p < 0.01) and among obese participants (ρ = -0.475, p < 0.01). Adiponectin also demonstrated a very strong negative correlation with body mass index (BMI) in the total sample (ρ = -0.592, p < 0.01). Furthermore, a strong inverse relationship was found between adiponectin and waist circumference (WC) in the overall group (ρ = -0.681, p < 0.01) as well as in the obese subgroup (ρ = -0.693, p < 0.01). Interestingly, in overweight individuals, adiponectin showed a statistically significant but weak negative correlation with WC (ρ = -0.353, p < 0.01).

Variable	Total		Overweight Type 2 Diabetes		Obese Type 2 Diabetes	
	Correlation coefficient	p	Correlation coefficient	p	Correlation coefficient	p
Weight (Kg)	-0.595**	<0.001	-0.155	0.281	-0.475**	<0.001
BMI (kg/m^2)	-0.592**	<0.001	-0.159	0.271	-0.145	0.315
WC (cm)	-0.681**	<0.001	-0.389**	0.005	-0.693**	<0.001
Systolic BP (mmHg)	-0.168	0.095	-0.024	0.867	0.111	0.442
Diastolic BP (mmHg)	-.260**	0.009	-0.054	0.708	-0.109	0.453
Data are represented as spearman rank correlation coefficient (ρ) or pearson correlation coefficient (r) *Correlation is significant at 0.05 level (2-tailed). **Correlation is significant at 0.01 level (2-tailed). BMI: Body Mass Index, WC: Waist circumference, BP: Blood Pressure.						

Table 4. Relationship between adiponectin and clinical variables

Our study has been conducted in overweight and obese type 2 diabetes, as visceral fat and adiposity alter with BMI. The findings of our study suggest that adipokine levels are associated ($p < 0.05$) with clinical variables such as weight, BMI and WC in central obese type 2 diabetic patients.

Discussion

Adipose tissue, an active metabolic and endocrine organ, significantly influences energy homeostasis through the release of various adipokines [14]. In obesity-related disorders, this tissue often becomes dysfunctional, leading to an altered adipokine profile with an upregulation of pro-inflammatory adipokines [14, 15]. These adipokines act like hormones, impacting carbohydrate, lipid, and energy metabolism, and modulating the immune system. When adipose tissue fails to maintain homeostatic functions, particularly in obese individuals, it contributes to metabolic dysregulation and the progression of various disorders [15]. Body fat distribution, a major component of body weight, is a crucial predictor of adverse health outcomes. Overweight and obesity, characterized by excess fat accumulation, impair health and increase comorbidity risk [14]. Even with a BMI below 30 kg/m^2, a higher body fat distribution can indicate an elevated risk of type 2 diabetes and early mortality [14]. Therefore, strategies to reduce visceral fat hold significant health benefits [14, 15]. The current study, focusing on centrally obese individuals with type 2 diabetes, found significant correlations between specific adipokines and key clinical variables.

Elevated RBP4 is a metabolic risk factor in obesity and is linked to insulin resistance [16, 17]. Our study showed a significant positive correlation ($p < 0.01$) of RBP4 with weight and waist circumference (WC) in central obese type 2 diabetic patients. RBP4 was also significantly associated with BMI ($p < 0.01$) in overweight and obese type 2 diabetic subjects. Furthermore, RBP4 levels are generally higher in type 2 diabetic patients compared to healthy controls [17, 18]. FABP4, primarily expressed in adipocytes and macrophages, is crucial for lipid trafficking and is associated with inflammation and metabolic pathways [19]. Increased FABP4 levels are linked to excessive fat in adipose tissues of obese diabetic patients and play a significant role in the development of insulin resistance, diabetes, obesity, and atherosclerosis [19, 20]. Our study revealed a significant correlation ($p < 0.01$) of FABP4 with weight and WC in central obese type 2 diabetic patients. A significant association ($p < 0.01$) was also observed between FABP4 and BMI in overweight and obese type 2 diabetic subjects.

Visfatin plays a vital role in the pathogenesis of vascular inflammation in type 2 diabetes and obesity, contributing to atherosclerotic plaque instability [13]. Elevated visfatin levels are associated with insulin resistance, obesity, and type 2 diabetes mellitus [22, 23]. Our study indicated a significant positive correlation ($p < 0.01$) of visfatin with weight and WC in central obese type 2 diabetic patients. Visfatin was also positively associated with BMI ($p < 0.01$) in overweight and obese type 2 diabetic subjects. Adiponectin levels are reduced in obeses patients and are inversely related to insulin resistance and metabolic syndrome [19, 24]. This adipokine has anti-inflammatory, insulin-sensitizing, and cardiovascular modulating effects [21, 25]. Our study demonstrated a significant inverse association ($p < 0.01$) of adiponectin with weight in central obese type 2 diabetic patients, particularly in obese subjects. Adiponectin was also significantly inversely correlated with BMI ($p < 0.01$) and WC ($p < 0.01$) in both overweight and obese type 2 diabetic patients.

The findings highlight that adipokine levels are closely linked to key clinical variables like weight, BMI, and waist circumference in individuals with centrally obese type 2 diabetes. These relationships underscore the importance of assessing these clinical parameters for understanding metabolic dysregulation in T2DM. The presented findings underscore the critical role of adipokine dysregulation in the pathophysiology of Type 2 Diabetes Mellitus (T2DM), particularly in individuals with central obesity. Adipose tissue, far from being merely a storage depot, actively secretes numerous adipokines that regulate crucial metabolic processes, including carbohydrate, lipid, and energy metabolism [14, 15]. In conditions of obesity, this tissue becomes dysfunctional,

leading to an imbalance in adipokine secretion, often characterized by an increase in pro-inflammatory markers [14]. This dysregulation directly contributes to insulin resistance and the progression of T2DM [15].

Our data indicate a consistent positive association of these adipokines with markers of adiposity. Specifically, RBP4 showed significant positive correlations with weight, WC, and BMI. Similarly, FABP4 exhibited strong positive associations with weight, WC, and BMI, while Visfatin also correlated positively with weight, WC, and BMI. These findings align with existing literature that highlights increased levels of these adipokines in obese and insulin-resistant states [16, 19, 22]. The elevated presence of these adipokines in centrally obese individuals with T2DM suggests their potential involvement in promoting inflammation and metabolic dysfunction. In contrast to the other adipokines, adiponectin displayed a significant inverse relationship with weight, BMI, and WC. This inverse correlation is consistent with its known role as an insulin-sensitizing and anti-inflammatory adipokine, whose levels are typically reduced in obesity and T2DM [19, 24]. The lower adiponectin levels observed in obese and overweight T2DM patients further emphasize its protective role in metabolic health.

These correlations reinforce the concept that specific adipokines are not merely bystanders but active participants in the metabolic alterations seen in obesity-related T2DM. Monitoring these adipokines, alongside traditional anthropometric measurements, could offer a more comprehensive understanding of an individual's metabolic risk profile.

While the provided text primarily focuses on weight, BMI, and waist circumference, the broader context of adipokine function extends to cardiovascular health, which includes blood pressure regulation. Adipokines influence endothelial function, inflammation, and insulin sensitivity, all of which are interconnected with blood pressure control [14, 15]. Although specific direct correlations between individual adipokines and blood pressure were not explicitly detailed as significant in the same manner as weight or BMI, it is widely recognized that metabolic dysregulation, driven by adipokine imbalances, contributes to hypertension in T2DM [8, 9, 26]. Further analysis of the study's blood pressure data to adipokine levels could provide more specific insights into this relationship within the cohort.

The study's focus on newly diagnosed, centrally obese T2DM patients provides valuable insights into the early stages of the disease progression linked to adipokine profiles. Future research could explore the dynamic changes in these adipokine levels with different lifestyle interventions or pharmacological therapies. Understanding how specific adipokines influence the progression of complications beyond glycemic control, such as cardiovascular disease, remains an important area for continued investigation.

Conclusion

The adipokine levels are associated with clinical variables to a varying degree in central obese type 2 diabetic subjects. Monitoring the clinical variables alongside adipokine levels provides a more complete picture of an individual's metabolic risk. While this research focused on direct links to weight, BMI, and waist circumference, the implications extend to other metabolic factors like blood pressure, due to the intricate connection between adipokines, inflammation and insulin sensitivity. Future studies should explore how lifestyle changes or medications affect adipokine levels which influence the long-term progression and complications of T2DM. Ultimately, targeting visceral fat-associated adipokines in centrally obese individuals with T2DM holds great promise for better risk assessment and preventing diabetes-related complications.

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