Research Square

Preprints are preliminary reports that have not undergone peer review.
They should not be considered conclusive, used to inform clinical practice,
or referenced by the media as validated information.

Association of Serum Omentin-1 with Insulin, Hemoglobin A1c, Glucose, and Lipid Profile in Palestinian Women with Type 2 Diabetes Mellitus

Saleh N. Mwafy

smwafy@alazhar.edu.ps

Al-Azhar University – Gaza

Maysaa F. Abu-Namous

Al-Azhar University – Gaza

Research Article

Keywords: Diabetes mellitus, Omentin, Obesity, Lipid profile, HbA1c, Adipokines

Posted Date: May 26th, 2026

DOI: https://doi.org/10.21203/rs.3.rs-9583348/v1

License: This work is licensed under a Creative Commons Attribution 4.0 International License.
Read Full License

Additional Declarations: No competing interests reported.

Version of Record: A version of this preprint was published at SN Comprehensive Clinical Medicine on July 27th, 2026. See the published version at https://doi.org/10.1007/s42399-026-02535-0.

Abstract

Objective

This study aimed to evaluate serum Omentin Levels and its Association with insulin, hemoglobin A1c, glucose, and lipid profile in Palestinian women with type 2 diabetes mellitus.

Methods

This observational case–control study was recruited 200 participants aged (30–60 years) divided into a control and case groups. Cases comprise of 150 type 2 diabetic female categorized according to their BMI as normal weight group (BMI < 25 kg/m^2), overweight group (BMI 25 -29.9 kg/m^2), and obese group (BMI ≥ 30 kg/m^2) with fasting glucose level > 150 mg/dl, and receiving hypoglycemic drug daily all over the study period in addition to 50 of apparently healthy female with normal weight (BMI < 25 kg/m2) and fasting glucose level < 90.0 mg/dl as a control group. Sociodemographic data, clinical history, anthropometric measurements, blood pressure, and pulse rate were collected from all subjects. Serum omentin-1, insulin, hemoglobin A1c, glucose, and lipid profile were measured and statistically analyzed.

Results

Compared with controls, diabetic participants showed significantly increased body weight, waist circumference, BMI, and both systolic and diastolic blood pressure ($P < 0.05$). Serum omentin level was significantly increased in overweight diabetic and obese diabetic patients compared to control ($P < 0.05$). HbA1c and fasting glucose levels were significantly higher across all diabetic groups ($P < 0.001$). All diabetic patients had increased cholesterol and triglyceride levels with decreased HDL values indicating dyslipidemia. Persons Correlation analysis showed significant correlations between BMI and anthropometric measures. Omentin levels were significantly correlated with lipid markers.

Conclusions

The results reveled that serum omentin-1 is associated with HbA1c, glucose, lipid profile and metabolic disturbances in T2DM patients. omentin could be used as a promising biomarker for T2DM patients and may play a role in reducing cardiovascular complications.

Introduction

Type 2 diabetes mellitus (T2DM) is one of the most common chronic metabolic diseases worldwide [1]. The global prevalence of T2DM has increased dramatically in recent decades, largely due to changes in lifestyle, increasing obesity rates, and population aging [2]. Persistent hyperglycemia is associated with

severe microvascular and macrovascular complications, including nephropathy, neuropathy, retinopathy, and cardiovascular diseases [3]. Obesity is widely recognized as one of the most significant risk factors that lead to type 2 diabetes and cardiovascular disease [4]. The accumulation of excess adipose tissue, particularly visceral fat, plays a major role in the pathogenesis of insulin resistance [5]. In recent years, adipose tissue has been identified not only as a site for energy storage but also as an active endocrine organ that secretes a variety of biologically active molecules known as adipokines [6]. These molecules participate in the regulation of glucose metabolism, lipid homeostasis, inflammatory processes, and vascular function [7].

Omentin is a relatively recently identified adipokine that is predominantly produced by visceral adipose tissue [8]. It has been reported that omentin enhances insulin-mediated glucose uptake in adipocytes and may therefore contribute to improved insulin sensitivity [9]. In addition, omentin has been suggested to exert anti-inflammatory and vasoprotective effects, which may have implications for cardiovascular health [10]. Metabolic control in individuals with diabetes is commonly evaluated using biochemical markers such as fasting plasma glucose and glycated hemoglobin (HbA1c). HbA1c reflects the average blood glucose concentration over the preceding two to three months and is widely used for both diagnosis and monitoring of diabetes [11]. Elevated HbA1c levels are associated with an increased risk of chronic diabetic complications [12].

Dyslipidemia is another metabolic abnormality frequently observed in patients with T2DM and is characterized by elevated triglycerides, increased concentrations of low-density lipoprotein (LDL), and reduced levels of high-density lipoprotein (HDL). These lipid alterations contribute significantly to the development of atherosclerosis and cardiovascular disease, which represent the leading causes of mortality among diabetic individuals [13]. Anthropometric measurements such as body mass index (BMI) and waist circumference are commonly used indicators of obesity and central fat accumulation. Numerous epidemiological studies have demonstrated a strong association between increased BMI, abdominal obesity, and the risk of developing T2DM [14]. Despite increasing interest in the role of adipokines in metabolic disorders, the relationship between circulating omentin levels and metabolic parameters in patients with type 2 diabetes remains unclear. Therefore, the present study was designed to evaluate serum omentin levels and its association with insulin, hemoglobin A1c, glucose, and lipid profile in Palestinian women with type 2 diabetes mellitus.

Methods

Study Design and Participants

This observational case–control study included 200 participants aged between (30-60) years in Gaza strip. Cases comprised 150 type 2 diabetic females divided into three subgroups based on body mass index; normal weight diabetes group (BMI < 25 kg/m^2), overweight diabetes group (BMI 25-29.9 kg/m^2), and obese diabetes group (BMI ≥ 30 kg/m2) with fasting glucose level >150 mg/dl, and receiving

hypoglycemic drug daily all over the study period. In addition to ffifty healthy individuals without diabetes, with normal weight (BMI < 25 kg/m^2) and fasting glucose level <90.0 mg/dl as a control group.

Sample size calculation: The sample size was calculated using EPI-INFO statistical package version 3.5.1 that was used with 95% CI, 80% power and 50% proportion as conservative and OR>2. The sample size is in a case of 1:1 ratio of case to control was 46. For a no-response expectation, the sample size was taken as 50 normal weight diabetes group, 50 overweight diabetes group, 50 obese diabetes group and 50 normal controls.

Study variables: Diagnosis of type 2 diabetes was established according to the criteria of the World Health Organization which include a fasting plasma glucose level ≥ 126 mg/dl, 2-hr plasma glucose level ≥ 200 mg/dl and HbA1c value between 5.7%-6.4% [15]. All patients were diagnosed to have diabetes mellitus for at least 5 years. All subjects were subjected to full history taking and clinical examination, and the study was conducted under physician supervision at private centers in Gaza strip. Exclusion criteria included male gender, patients with T1DM, severe kidney or liver diseases, severe cardiovascular diseases, chronic viral or bacterial infection, asthma, tumors, connective tissue diseases and pregnant females were excluded.

Ethics Approval and Consent to Participate: Ethical approval was obtained from Palestinian Health Council, Helsinki committee for ethical approval in Gaza strip under approval number PHRC/HC/899/21 and the study procedures were therefore done in accordance to the accepted ethical standards of research involving human subjects. All participants provided an informed consent after the research protocols were carefully explained to them. The informed consent form was signed by all the participants before joining the study.

Clinical trial number: not applicable

Anthropometric Measurements

The anthropometric measurements involved; weight, height, and waist circumference. BMI was used for the evaluation of the obesity among the diabetic female. To measure BMI, weighed and measured heights of the individuals without shoes using medical balance were used for this purpose. Waist circumference was measured in centimeter using a metric ribbon. Body mass index formula is BMI = kg/m^2 [16].

Blood pressure and pulse monitors: Blood pressure was measured with mercury sphygmomanometer. The pulse rate was determined by counting the pulses at the wrist region at least 15 seconds and then multiplying the result by four to obtain the number of beats per minute.

Specimen collection and processing: Fasting Blood samples were collected from all participants after overnight fasting. Serum was separated by centrifugation for 10 minutes at 2500 rpm and used for the following biochemical parameters. Omentin Human/Intelectin 1 (ITLN1) protein in serum was

determined quantitatively by a solid- phase enzyme-linked immune sorbent assay (ELISA) according to the manufacture instruction. Insulin was quantitative determined by a solid phase enzyme-linked immune sorbent assay (ELISA) (DRG Diagnostic international, USA [17]. The HBA1c was determined by quantitative nephelometric directly for the percentage of glycated hemoglobin (HBA1c) to total hemoglobin (HbT) [18]. Enzymatic colorimetric assays were used to measure biochemical parameters such as fasting blood sugar (FBS) [19], total cholesterol (TC) [20], triglycerides (TG) [21], high density lipoprotein (HDL-C) [22]. Low density lipoprotein (LDL) was calculated by empirical relationship of Friedewald [23].

Statistical analysis: Mean and standard deviation was used to summarize quantitative variables and frequencies and percentages were used to summarize qualitative variables. Independent samples t-test was used in performing group comparison with respect to quantitative variables whereas the Chi-square test was utilized in the process of group comparison with respect to qualitative variables. Associations of quantitative variables were measured using Pearson correlation coefficients, and percentage change was calculated where necessary. A p-value of less than 0.05 was deemed to be statistically significant.

Results

Sociodemographic data in Table 1, showed that there was a significant difference in the age distribution, education level, and place of residency among the studied groups ($P \leq 0.001$). The diabetic participants were generally older than control group with a mean age ranging from 48.1–49.2 years versus 41.1 years among controls. The highest percentage of diabetic individuals was observed in the 41–60 year. Regarding marital status, no statistically significant difference between the groups ($P = 0.274$), while significant difference was detected in educational level ($P = 0.001$). the percentage of bachelor's degrees holders where higher in controls, but the percentages of primary and secondary education levels were higher in diabetic groups especially those that were overweight and obese. Similarly, residency showed a significant difference ($P = 0.001$), with most diabetic participants living in Gaza Governorate.

Metformin is seen as the first-line therapy of type 2 diabetes, particularly in overweight and obese patients (56% each). There were no notable differences ($P = 0.233$) which may indicate that treatment strategies were fairly similar across BMI categories.

Table 2 presented anthropometric measurements that significantly differ between diabetic groups and controls, especially for weight, waist circumference, BMI, blood pressure and pulse. The body weight increased gradually among diabetic populations with obese diabetic, reaching weight of 96.4±15.5 kg which is a 55.9% increase compared with controls ($P = 0.001$). The waist circumference also increased significantly, rising from 79.4±6.3 cm in controls to 109.9±10.4 cm in obese diabetics. BMI followed the same pattern, increasing from 22.6±2.2 kg/m² in controls to 36.7±5.4 kg/m² in obese diabetics, representing a 62.2% increase ($P = 0.001$). These results confirm the relationship between obesity and type 2 diabetes pathogenesis. The diabetic patients had a significant increase in blood pressure. The

systolic blood pressure increased by about 13–16%, and the diastolic pressure increased by 10–13%, while the pulse showed slight significant increase in obese diabetic patients (P = 0.019).

Table 3 showed that the levels of serum omentin levels were found to be significantly high in overweight (36.3**±**8.9 ng/ml) and obese diabetic patients (36.4**±**10 ng/ml) with 50% increase compared with (24.3**±**11.3 ng/ml) in the controls, P≤ 0.01. in diabetic groups, although increase in insulin levels than in controls, these increases were not significant. Controversy, HbA1c levels were significantly elevated in all diabetic groups (P ≤0.001), ranging between 5.2% in controls to 7.6–7.8% in diabetic patients, confirming poor glycemic control. Similarly, the glucose levels were significantly increased, to around 146–161 mg/dL, representing increases of 78–98% compared to controls.

Lipid profile analysis revealed that diabetic patients have a significant dyslipidemia demonstrated in Table 4. Diabetic groups had significantly higher levels of total cholesterol (P < 0.05), with percentage of increase from 14–18% compared to the controls. Similarly, there was a significant increase in triglyceride levels especially in the overweight diabetic patients, where the levels increased by over 114%. A moderate increase in LDL cholesterol levels and it was only statistical significance in the overweight group (P ≤ 0.05). In the other hand, there was a decrease in HDL cholesterol levels, especially in overweight diabetic patients (P≤ 0.01).

Correlation analysis in Table 5, showed that there is a strong positive correlation between BMI and body weight and waist circumference in all groups (P ≤ 0.001). BMI also showed a significant positive correlation with the levels of cholesterol in the control group (P < 0.01). Strong positive correlations were found between BMI with weight, waist circumference, omentin in the female among normal weight group. There was a strong positive correlation between female weight and waist circumference with overweight and obese groups of females (*P*<0.01). However, BMI was not significantly correlated with glucose, HbA1c, insulin, or triglycerides in most diabetic groups.

Discussion

The present study demonstrated that there were significant differences in age, educational level, and place of residency between the studied groups. Diabetic patients were significantly older than the control group, and most of cases fell within the 41-60 years old range. These findings are consistent with other epidemiological studies that indicated the prevalence of type 2 diabetes mellitus increases with advancing age due to progressive insulin resistance and pancreatic β-cell dysfunction. Zheng et al., in 2018 and Sun et al., in 2022, who reported that middle-aged and old people have much higher incidence of T2DM than younger populations [24] and [25]. The study also revealed a significant association between lower educational level and the presence of diabetes. Participants with diabetes were more likely to have primary or secondary education compared with the control group, where higher education levels predominated. This finding is in agreement with the study conducted by Agardh et al. in 2011 and later confirmed by Hill-Briggs et al. in 2020, who reported that low level of education is linked to higher risk of diabetes because of low health literacy, unhealthy lifestyle behaviors, and insufficient access to

preventive healthcare services [26, 27]. Additionally, the distribution of residency differed significantly among groups, with more patients with diabetic being residing in Gaza Governorate. The same geographic differences in the prevalence of diabetes were reported in regional studies from the Middle East and North Africa, where environmental, urbanization conditions, and socioeconomic disparities contribute to differences in the distribution disease [28].

Metformin is seen as the first-line therapy of type 2 diabetes, particularly in overweight and obese patients (56% each). There were no notable differences which may indicate that treatment strategies were fairly similar across BMI categories. The most frequently used medication in diabetic patients regardless of the BMI status is metformin which is consistent with the current international treatment guidelines which recommend metformin to be the first-line therapy when treating T2DM due to its effectiveness, safety profile and positive effects on insulin sensitivity [29]. The results showed significant increases in body weight, waist circumference, BMI, and blood pressure among diabetic patients compared to the control group. These results indicated that obesity and T2DM have strong association. Waist circumference is a sign of central obesity, which is known to be a key importance in the mechanism of insulin resistance development. Kohlgruber & Lynch in 2015 and Hruby and Hu in 2015, also reported similar results and revealed that excess adiposity, especially the presence of visceral fat tissue is a strong predictor of T2DM on the basis of inflammatory processes and insulin signaling [30, 31]. The elevated blood pressure observed among diabetic patients in the present study also supports the previous studies showing that hypertension is a common comorbidity in diabetes. According to Fox et al. (2015) hypertension combined with diabetes is a significant risk factor for cardiovascular complications, such as coronary artery disease and stroke [32].

Biochemical examination revealed that HbA1c and fasting glucose levels were significantly higher in diabetic patients than controls, thus showing that the patients had poor glycemic control. These findings are in accordence with the diagnostic criteria and pathophysiological characteristics of T2DM. Similar increases in HbA1c and fasting glucose levels have been reported [33, 34]. Interestingly, the present study showed that the levels of omentin was significantly elevated in overweight and obese diabetic patients. Omentin is an adipokine released by visceral adipose tissue primarily and has been involved in glucose metabolism and insulin sensitivity [7].

Although insulin levels were slightly elevated in diabetic groups, these differences were not statistically significant. This observation may reflect underlying insulin resistance, where increased insulin production fails to maintain normal glucose levels. Similar patterns have been reported in patients with T2DM [35, 36]. The current study reported high levels of dyslipidemia among diabetic patients, which manifested by increased levels of total cholesterol and triglycerides, and decreased HDL levels. These findings are in agreements with the typical lipid abnormalities observed in T2DM, which known as diabetic dyslipidemia. The high triglyceride levels found in overweight diabetic patients, which are closely linked to hypertriglyceridemia, which is strongly associated with insulin resistance and increased cardiovascular risk. According to Mooradian (2009), diabetic dyslipidemia is a major cause of atherosclerosis and cardiovascular disease among diabetic populations [37]. Correlation analysis

demonstrated that BMI and anthropometric measures including weight and waist circumference had a strong positive relationship. This relationship is expected since BMI is directly influenced by body mass and fat accumulation. BMI was found to have strong positive correlation with cholesterol in the control group, suggesting that increase in body weight may influence lipid metabolism even in non-diabetic individuals. Similar results were described by Després (2012), who reported that obesity causes dyslipidemia due to the change lipid metabolism and increased hepatic lipid synthesis .[38]

Conclusion

In conclusion, individuals with type 2 diabetes mellitus exhibited significant differences in anthropometric measurements, glycaemic markers, and lipid profile compared with healthy subjects. Elevated omentin concentrations observed among overweight and obese diabetic patients suggest a potential role for this adipokine in metabolic regulation. These findings highlight the possible importance of omentin as a biomarker associated with metabolic disturbances in T2DM and may play a role in reducing cardiovascular complications. Further investigations are required to clarify the mechanisms underlying the relationship between omentin and metabolic disorders.

Declarations

Funding agencies: The researcher did not receive any grant money and this work did not support by grant funding

Conflicts of interest: The authors declare no conflicts of interest.

Ethical consideration: the necessary approval to conduct this study was obtained from the Helsinki Committee for Ethical Approval number PHRC/HC/899/21, in the Gaza Strip, and all study participants signed an informed consent form.

Consent to Participate: Written informed consent was obtained from all participants prior to their inclusion in the study.

Written Consent for Publication: Not applicable.

Availability of Data and Material: The data analyzed during the current study are available at the corresponding author upon reasonable request.

Code Availability: Not applicable.

Author contributions: All authors contributed to writing and reviewing the manuscript, and approved the final version. Authors' Contributions: S.N.M. led the study, with primary responsibility for conceptualization, formal analysis, investigation, supervision, visualization, and preparation of the

original manuscript. M.F.N. performed the experiments, contributed reagents, materials, analysis tools or data and contributed to drafting and editing the manuscript

References

1. Galicia-Garcia, U., et al., *Pathophysiology of type 2 diabetes mellitus.* International journal of molecular sciences, 2020. **21**(17): p. 6275.
2. Gong, J.Y., et al., *Variation in type 2 diabetes prevalence across different populations: the key drivers.* Diabetologia, 2025. **68**(11): p. 2327-2339.
3. Islam, K., et al., *Diabetes mellitus and associated vascular disease: pathogenesis, complications, and evolving treatments.* Advances in therapy, 2025. **42**(6): p. 2659-2678.
4. Shahid, I., et al., *Obesity and atherosclerotic cardiovascular disease: A review of social and biobehavioral pathways.* Methodist DeBakey cardiovascular journal, 2025. **21**(2): p. 23.
5. Packer, M., *Causal demonstration of adiposity-induced adipose-specific signaling derangements in the pathogenesis of the clinical features of the cardiovascular-kidney metabolic syndrome.* Cardiovascular Diabetology, 2026. **25**(1): p. 86.
6. Zadgaonkar, U., *The interplay between adipokines and body composition in obesity and metabolic diseases.* Cureus, 2025. **17**(1).
7. Pedro, W.J.S., et al., *Role of adipokines chemerin, visfatin, and omentin in obesity and their inflammatory and metabolic implications.* Biomedicines, 2025. **13**(10): p. 2321.
8. Biegański, H.M., K.M. Dąbrowski, and A. Różańska-Walędziak, *Omentin—General overview of its role in obesity, metabolic syndrome and other diseases; problem of current research state.* Biomedicines, 2025. **13**(3): p. 632.
9. Hussein, A.A., et al., *Omentin roles in physiology and pathophysiology: an up-to-date comprehensive review.* Archives of physiology and biochemistry, 2024. **130**(6): p. 800-813.
10. Sena, C.M., *Omentin: a key player in glucose homeostasis, atheroprotection, and anti-inflammatory potential for cardiovascular health in obesity and diabetes.* Biomedicines, 2024. **12**(2): p. 284.
11. Eyth, E., M. Zubair, and R. Naik, *Hemoglobin A1c*, in *StatPearls [Internet]*. 2025, StatPearls Publishing.
12. Boye, K.S., et al., *The association between sustained HbA1c control and long-term complications among individuals with type 2 diabetes: a retrospective study.* Advances in Therapy, 2022. **39**(5): p. 2208-2221.
13. Goldberg, I.J., *Diabetic dyslipidemia: causes and consequences.* The journal of clinical endocrinology & metabolism, 2001. **86**(3): p. 965-971.
14. Janssen, I., P.T. Katzmarzyk, and R. Ross, *Waist circumference and not body mass index explains obesity-related health risk.* The American journal of clinical nutrition, 2004. **79**(3): p. 379-384.
15. WHO, *World Health Organization. Definition and diagnosis of diabetes mellitus and intermediate hyperglycaemia: report of a WHO/IDF consultation.* 2006.

16. Mohajan, D. and H.K. Mohajan, *Body mass index (BMI) is a popular anthropometric tool to measure obesity among adults.* Journal of Innovations in Medical Research, 2023. **2**(4): p. 25-33.
17. MacDonald, M.J. and J.P. Gapinski, *A rapid ELISA for measuring insulin in a large number of research samples.* Metabolism, 1989. **38**(5): p. 450-452.
18. Weykamp, C.W., et al., *Carbamylated Hemoglobin Interference in Glycohemoglobin Assays.* Clinical Chemistry, 1999. **45**(3): p. 438-440.
19. Barham, D. and P. Trinder, *An improved colour reagent for the determination of blood glucose by the oxidase system.* Analyst, 1972. **97**(1151): p. 142-145.
20. Richmond, W., *Preparation and properties of a cholesterol oxidase from Nocardia sp. and its application to the enzymatic assay of total cholesterol in serum.* Clinical chemistry, 1973. **19**(12): p. 1350-1356.
21. Cole, T.G., S.G. Klotzsch, and J.R. McNamara, *Measurement of triglyceride concentration.* Handbook of lipoprotein testing, 2000. **8**.
22. Bachorik, P.S., *Measurement of low-density-lipoprotein cholesterol.* Handbook of lipoprotein testing, 2000. **2**: p. 245-64.
23. Friedewald, W.T., R.I. Levy, and D.S. Fredrickson, *Estimation of the concentration of low-density lipoprotein cholesterol in plasma, without use of the preparative ultracentrifuge.* Clinical chemistry, 1972. **18**(6): p. 499-502.
24. Zheng, Y., S.H. Ley, and F.B. Hu, *Global aetiology and epidemiology of type 2 diabetes mellitus and its complications.* Nature reviews endocrinology, 2018. **14**(2): p. 88-98.
25. Sun, H., et al., *IDF Diabetes Atlas: Global, regional and country-level diabetes prevalence estimates for 2021 and projections for 2045.* Diabetes research and clinical practice, 2022. **183**: p. 109119.
26. Agardh, E., et al., *Type 2 diabetes incidence and socio-economic position: a systematic review and meta-analysis.* International journal of epidemiology, 2011. **40**(3): p. 804-818.
27. Hill-Briggs, F., et al., *Social determinants of health and diabetes: a scientific review.* Diabetes care, 2020. **44**(1): p. 258.
28. Namazi, N., et al., *Burden of type 2 diabetes mellitus and its risk factors in North Africa and the Middle East, 1990–2019: findings from the Global Burden of Disease study 2019.* BMC Public Health, 2024. **24**(1): p. 98.
29. Jimoh, A.O., et al., *Comparative Efficacy, Safety, and Cost-Utility of DPP-4 Inhibitors and Metformin Combination Therapy in Type 2 Diabetes: A Systematic Review of Real-World Clinical and Economic Outcomes.* Journal of Diabetes Research, 2026. **2026**(1): p. 8464330.
30. Kohlgruber, A. and L. Lynch, *Adipose tissue inflammation in the pathogenesis of type 2 diabetes.* Current diabetes reports, 2015. **15**(11): p. 92.
31. Hruby, A. and F.B. Hu, *The epidemiology of obesity: a big picture.* Pharmacoeconomics, 2015. **33**(7): p. 673-689.
32. Fox, R., et al., *The State of the UK's Butterflies 2015.* 2015.

33. Sheng, L., et al., *Glycemic variability evaluated by HbA1c rather than fasting plasma glucose is associated with adverse cardiovascular events.* Frontiers in Endocrinology, 2024. **15**: p. 1323571.
34. Rasoulinejad, S.A., et al., *High levels of FBS and HbA1c and their association with diabetic retinopathy: a study in the north of Iran.* Journal of Diabetes & Metabolic Disorders, 2022. **21**(1): p. 399-406.
35. Sancar, G. and A.L. Birkenfeld, *The role of adipose tissue dysfunction in hepatic insulin resistance and T2D.* Journal of Endocrinology, 2024. **262**(3).
36. Vajje, J., et al., *Comparison of the efficacy of metformin and lifestyle modification for the primary prevention of type 2 diabetes: a meta-analysis of randomized controlled trials.* Cureus, 2023. **15**(10).
37. Mooradian, A.D., *Dyslipidemia in type 2 diabetes mellitus.* Nature Reviews Endocrinology, 2009. **5**(3): p. 150-159.
38. Després, J.-P., *Body fat distribution and risk of cardiovascular disease: an update.* Circulation, 2012. **126**(10): p. 1301-1313.

Tables

Table 1: Sociodemographic characteristics of study population.

Parameter		Control		Diabetic patients						*P*-value
				Normal weight		Overweight		Obese		
		N	%	N	%	N	%	N	%	
Age (years)	30 - 40	34	68.4 %	5	10%	7	14.6 %	7	14%	0.001
	41 - 50	11	21.1%	25	50%	18	35.4 %	19	38 %	
	51 - 60	5	10.5%	20	40%	25	50 %	24	48 %	
	Mean ± SD	41.1±6.5		49.2±8.1		48.5±6.3		48.1±6.3		0.001
Marital status	Married	45	90%	37	74 %	44	88 %	44	88 %	0.274
	Widow	0	0.0%	5	10%	5	10 %	2	4 %	
	Divorced	0	0.0%	0	0.0 %	1	2 %	1	2 %	
	Single	5	10 %	8	16 %	0	0.0 %	3	6%	
Education	illiteracy	5	10%	5	10 %	2	4 %	2	4 %	0.001
	Primary	13	26 %	13	26 %	26	52 %	21	42 %	
	Secondary	5	10 %	15	30 %	12	24 %	23	46 %	
	Bachelor's	27	54 %	17	35%	10	20 %	4	8 %	
Residency	North Gaza Governorate	34	68 %	18	36%	18	36%	10	20%	0.001
	Gaza Governorate	16	32%	32	64%	32	64 %	40	80 %	

N: number of the subject; **SD:** standard deviation,

The significant of difference was checked by checked by chi square test, significant at $P \leq 0.05$.

Table 2: anthropometric, blood pressure (BP) and pulse measurements among study population

Parameters	Control	Diabetic patients Means ± SD				
	Means ± SD	Normal weight	Overweight	Obese	% Change	P-value
Weight (kg)	61.9±8.5	62.2±6.6	76.0±6.7	96.4 ±15.5	0.6% 22.9 % 55. 9 %	0.887 0.001 0.001
Length (cm)	164.9±6.5	162.3±4.8	164.4±5.4	162.0±5.8	- 1.6 % - 0.3 % - 1.8 %	0.154 0.732 0.065
Waist circumference (cm)	79.4±6.3	84.8±8.1	95.5±7.8	109.9±10.4	6.7 % 20.3% 38.4%	0.028 0.001 0.001
BMI (Kg/m^2)	22.6±2.2	23. 5±1.8	28.0±1.4	36.7±5.4	3.7 % 23.8 % 62.2 %	0.202 0.001 0.001
Systolic B P (mmHg)	111.5±11.6	127.5±18.7	126.2±15.4	130±17.4	14.4% 13.2 % 16.2 %	0.003 0.001 0.001
Diastolic B P (mmHg)	73.0±8.6	81.4±11.4	80.7±12.5	82.3±9.1	11.5 % 10.5 % 12.7%	0.014 0.017 0.001
Pulse (beats/min)	78.2±7.4	81.4±13.5	83.9±12.13	84.3±10.25	4.9 % 7.3 % 7.8 %	0.370 0.061 0.019

Each reading represents Mean ±SD of 50 subjects.

The significance of difference was checked by One Way ANOVA, significant at P <0.05, compare all vs control.

Table 3: Omentin, Insulin, Hemoglobin A1C, and Glucose determination among study population.

Parameters	Control Mean ± SD	Diabetic patients Mean ± SD				
		Normal weight	Overweight	Obese	% Change	P-value
Omentin (ng/mL)	24.3±11.3	26.3±14.7	36.3±8.9	36.4±10.0	8.2 % 49.6 % 50 %	0.708 0.001 0.003
Insulin (µ IU/ml)	11.6±1.39	13±1.56	15.9±1.51	15.6±1.45	11.4 % 36.2 % 34.1%	0.781 0.297 0.297
HBA1C (%)	5.2±0.3	7.6±1.9	7.8±1.7	7.6±1.6	45.9 % 48.6 % 45.1%	0.001 0.001 0.001
Glucose (mg/dl)	81.8±10.6	161.6±84.2	154.7±55.9	146.2±63.1	97.6 % 89.1 % 78.7 %	0.001 0.001 0.001

Each reading represents Mean ±SD of 50 subjects. The significance of difference was checked by One-Way ANOVA, significant at *P* <0.05, compare all vs control.

Table 4: Determination of lipid profile among study population

Parameters	Control Mean ± SD	Diabetic patients (Mean ± SD)				
		Normal weight	Overweight	Obese	% Change	P-value
Cholesterol (mg/dl)	175.0±29.4	199.9±44	207.6±46.9	200.5±47.6	14.2 % 18.6 % 14.6 %	0.046 0.007 0.031
Triglyceride (mg/dl)	100.8±45.1	164.2±87.9	215.8±64.1	180.6±70.9	62.9 % 114.1 % 79.2 %	0.008 0.004 0.003
LDL (mg/dl)	115.1±30.1	133.5±34.8	135.5 ± 39.8	124.7±37.2	15.9 % 17.7 % 8.3 %	0.087 0.049 0.309
HDL (mg/dl)	40.7±12.9	33 ±13.3	32.2±13	38.9±15.4	19.00 % 21 % 4.5 %	0.074 0.018 0.636

Each reading represents Mean ±SD of 50 subjects.

The significance of difference was checked by One-Way ANOVA, significant at *P* < 0.05, compare all vs control.

Table 5: Correlation between BMI and the biochemical parameters among the study populations.

Parameters	BMI							
	Control		Normal weight		Overweight		Obese	
	r	P	r	P	r	P	r	P
Weight	0.813**	0.001	0.866**	0.001	0.614**	0.001	0.895**	0.001
Waist circumference	0.727**	0.001	0.716**	0.001	0.362*	0.012	0.804**	0.001
Omentin	-0.479	0.162	0.515*	0.020	0.054	0.777	0.285	0.127
Insulin	0.059	0.811	0.110	0.644	0.131	0.374	0.004	0.974
HBA1C (%)	0.143	0.559	0.227	0.335	-0.049	0.743	-0.155	0.230
Glucose	0.084	0.732	0.089	0.708	-0.048	0.747	-0.016	0.904
Cholesterol	0.607**	0.006	0.209	0.377	0.184	0.210	0.013	0.922
Triglyceride	0.057	0.817	0.177	0.456	0.040	0.789	0.133	0.302
LDL	0.139	0.570	0.239	0.311	0.123	0.404	-0.053	0.681
HDL	0.295	0.220	- 0.217	0.358	-0.029	0.846	0.052	0.686

.Correlation is significant at the 0.01 level (2-tailed) **

Correlation is significant at the 0.05 level (2-tailed *

significant positive correlation with cholesterol concentrations (P<0.01).