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Assessing the Effects of Age, Sex, and Body Mass Index on Glycemic and Lipid Levels in Newly Diagnosed Type 2 Diabetic Patients: A Cross-Sectional Study

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Abstract

Objective: Newly diagnosed type 2 diabetic patients show varied metabolic profiles influenced by factors like age, sex, and body mass index, which affect disease progression and risk of other health problems. To evaluate how demographic factors such as age, sex, and body mass index (BMI) influence glycemic control and lipid profile parameters in patients newly diagnosed with type 2 diabetes mellitus. The study included 60 newly diagnosed type 2 diabetic patients aged 30 to 75 years. Blood samples were collected after overnight fasting; they were divided into two parts, with the EDTA tube used immediately for HbA1c measurement, while the other parts were separated to obtain serum for biochemical parameters. Biochemical measurements included glucose and lipid profile by enzymatic methods, HbA1c by ion exchange HPLC, and insulin by sandwich electrochemiluminescence immunoassay. Insulin resistance was calculated using the HOMA-IR formula. Biochemical parameters were not varied significantly by age or sex except for higher total cholesterol and HDL cholesterol in females. Obesity was strongly associated with increased insulin resistance and an adverse lipid profile characterized by elevated TG, VLDL cholesterol, LDL cholesterol, and reduced HDL cholesterol. Glycemic control markers (glucose, HbA1c) were not significantly linked to lipid abnormalities. This study concluded that age and sex do not affect glycemic control and lipid profile, except that women have higher total cholesterol and HDL cholesterol. However, obesity is strongly associated with increased insulin resistance and an adverse lipid profile.

Keywords: type 2 diabetes mellitus, insulin resistance, glucose, obesity, lipid

Introduction

Type 2 diabetes mellitus (T2DM) is a chronic metabolic disorder marked by persistent hyperglycemia due to insulin resistance and impaired insulin secretion. It represents a major global public health challenge owing to its increasing prevalence and associated complications, particularly cardiovascular diseases linked to dysregulated glycemic and lipid metabolism [1;2]. Newly diagnosed T2DM patients often present with heterogeneous metabolic profiles influenced by demographic and physiological factors such as age, sex, and body mass index (BMI), which modulate disease progression and risk of comorbidities [3]. Age is a critical determinant in the pathophysiology and clinical manifestation of T2DM, Younger individuals with T2DM tend to experience more severe hyperglycemia and poorer glycemic control compared to older patients, potentially due to differences in insulin sensitivity and lifestyle factors [4]. Moreover, age-related changes in lipid metabolism contribute to varying patterns of dyslipidemia, a common complication that exacerbates cardiovascular risk in diabetic patients [3]. Sex differences also play a significant role; males and females exhibit distinct glycemic and lipid profiles at diagnosis, males under 35 years show lower high-density lipoprotein cholesterol (HDL-C) compared to females, whereas these differences tend to diminish or alter with advancing age [4;5]. BMI, a surrogate marker of adiposity, is strongly implicated in the development and metabolic control of T2DM. Elevated BMI, particularly obesity, exacerbates insulin resistance and disrupts lipid metabolism, leading to poorer glycemic control and dyslipidemia [6;7]. Cross-sectional studies have demonstrated a positive correlation between BMI and glycated hemoglobin (HbA1c) levels, with obese patients exhibiting significantly higher HbA1c compared to overweight and normal-weight individuals [6]. This relationship underscores the importance of weight management in improving glycemic outcomes and reducing cardiovascular risk in T2DM patients [8]. However, some studies in newly diagnosed populations have reported no significant association between BMI and HbA1c, possibly reflecting the variable asymptomatic phase of T2DM and the independent roles of these parameters in disease management [9]. The complex interplay of age, sex, and BMI on glycemic and lipid parameters, a comprehensive assessment of these factors in newly

diagnosed T2DM patients is essential, such evaluation can elucidate demographic-specific metabolic patterns, inform personalized therapeutic strategies, and optimize risk stratification for diabetes-related complications [10;11]. This cross-sectional study aims to assess the effects of age, sex, and BMI on glycemic indices (HbA1c, fasting serum glucose, insulin, HOMA-IR) and lipid profiles (total cholesterol, triglycerides, VLDL, HDL-C, and LDL-C) in newly diagnosed T2DM patients. By analyzing these relationships, the study seeks to contribute to a nuanced understanding of metabolic heterogeneity at T2DM onset and support targeted interventions to improve clinical outcomes.

Materials and Methods

Subjects

The 60 patients with newly identified T2DM who participated in this study. Their ages ranged from 30 to 75 years, divided according to age into three groups: 30-45 years, 46-60 years, and 61-75 years. Twenty participants for each group. There were 30 participants for each group, both male and female. In addition, three groups of BMI were normal weight (18.5 – 24.9 kg/m^2), overweight (25.0 – 29.9 kg/m^2), and obese (≥ 30 kg/m^2) for each group of twenty participants.

The patients visited specialist clinics at Basra city's Al-Fayhaa Teaching Hospital. The collection of blood samples occurred between the start of April 2024 and the last day of August 2024. Name, age, sex, height, and weight were among the details gathered from each participant. They were between thirty and seventy-five years old. Each participant had four milliliters of venous blood drawn in the morning after fasting for the whole night, and the blood was split into two tubes using a disposable syringe.

Initially, two milliliters of blood were moved into a vacutainer tube that contained EDTA K3 in order to estimate the HbA1c right away. Next, two milliliters of blood were put into an evacuated tube with a gel and clot activator. The serum was separated, and the insulin and glucose levels were immediately determined by centrifuging the blood for 15 minutes at 3500 revolutions per minute (RPM) after it had stood for half an hour.

Methods

The application of a balance for evaluating weight and a stadiometer to determine standing height. This formula was utilized to determine the body mass index (BMI): This is the BMI: weight (kg) divided by height (m^2).

Serum glucose and lipid profile have been determined by the enzymatic methods in a fully automated biochemistry analyzer (Spin

200 analyzer, Spinreact Company, Spain). Whole blood was tested for glycosylated hemoglobin A1c (HbA1c) by the ion exchange high-performance liquid chromatography (HPLC) technique in a fully automated analyzer (D-10 analyzer, Bio-Rad Company, USA). The sandwich-based electrochemiluminescence immunoassay (ECLIA) technique was applied in a fully automated immunoassay instrument (Cobas e 411 analyzer, Roche Business, Germany) to determine the serum insulin level. Insulin resistance was assessed using the homeostatic model assessment of insulin resistance (HOMA-IR) formula: HOMA-IR is equivalent to fasting insulin (mIU/L) × fasting glucose (mg/dl)/405.

Statistical analysis

All data analysis was done using version 26.0 of SPSS. The use of one-way ANOVA allowed for comparison of means between multiple groups (age and BMI categories), while the use of the t-test allowed for comparison of means between sex groups, identifying statistically significant differences. Pearson's correlation coefficient assessed the relationships between continuous variables. A significance threshold of $P < 0.05$ was used, meaning any observed differences or associations with p-values below this threshold are unlikely to be due to chance.

Results

The results in Table 1 revealed that a comparison of mean ± standard deviation (SD) values for various biochemical parameters across three age groups were group 1 (30-45 years), group 2 (46-60 years), and group 3 (61-75 years), each with 20 participants found no statistically significant differences in glucose, HbA1c, insulin, HOMA-IR, TC, TG, VLDL, and LDL among the groups ($P > 0.05$).

Table 1: Mean ± standard deviation of circulating biochemical parameters levels in T2DM according to the physiological age groups

Parameters	Group 1 (30-45 years) (n=20)	Group 2 (46-60 years) (n=20)	Group 3 (61-75 years) (n=20)	F-value	P-value
Glucose (mg/dL)	209.05 ± 79.75	209.80 ± 74.55	205.95 ± 70.28	0.015	0.985
HbA1c (%)	9.025 ± 1.68	9.420 ± 1.54	8.825 ± 1.62	0.705	0.498
Insulin (µlU/mL)	29.16 ± 8.45	27.92 ± 7.68	27.88 ± 3.75	0.220	0.803
HOMA-IR	14.06 ± 4.48	13.66 ± 4.06	14.07 ± 4.86	0.054	0.948
TC (mg/dL)	233.55 ± 8.89	235.65 ± 8.64	238.40 ± 8.86	1.529	0.225
TG (mg/dL)	389.95 ± 48.94	375.55 ± 47.87	379.85 ± 41.18	0.514	0.601
VLDL (mg/dL)	77.99 ± 9.79	75.10 ± 9.58	75.97 ± 8.24	0.516	0.599
LDL (mg/dL)	144.25 ± 8.84	141.45 ± 7.28	141.35 ± 8.25	0.816	0.447
HDL (mg/dL)	35.55 ± 4.31	36.90 ± 4.17	36.45 ± 4.05	0.542	0.585

The results in Table 2 revealed that mean ± standard deviation (SD) values for various biochemical parameters measured in males (n=30) and females (n=30) of newly diagnosed T2DM, along with the p-values indicating the statistical significance of differences between the two groups. No significant differences ($P > 0.05$) in glucose, HbA1c, insulin, HOMA-IR, TG, VLDL, and LDL between males and females of newly diagnosed T2DM. However, TC and HDL levels in females were higher than those of males with a statistical change ($P < 0.001$).

Table 2: Mean ± standard deviation of circulating biochemical parameters levels in T2DM according to the sex

Parameters	Male (n=30)	Female (n=30)	P-value
Glucose (mg/dL)	208.57 ± 69.20	207.97 ± 79.13	0.975
HbA1c (%)	9.01 ± 1.66	9.17 ± 1.57	0.691
Insulin (µlU/mL)	28.76 ± 6.62	27.88 ± 7.15	0.623
HOMA-IR	14.36 ± 4.91	13.49 ± 3.88	0.449
TC (mg/dL)	227.90 ± 3.67	243.83 ± 3.92	< 0.001
TG (mg/dL)	381.83 ± 46.96	381.73 ± 45.29	0.993
VLDL (mg/dL)	76.37 ± 9.39	76.34 ± 9.07	0.991
LDL (mg/dL)	141.17 ± 7.28	143.53 ± 8.85	0.263
HDL (mg/dL)	32.93 ± 2.38	39.67 ± 2.41	< 0.001

Table 3 represents mean ± standard deviation (SD) values of various biochemical parameters measured in three groups of individuals: Normal weight (n=20), Overweight (n=20), and Obese (n=20). Despite differences in weight status, fasting glucose and HbA1c levels were not significantly different ($P > 0.05$). While insulin levels increase progressively from normal weight (24.83 ± 5.36, µlU/mL) to overweight (26.64 ± 5.20, µlU/mL) to obese (33.48 ± 6.78, µlU/mL), indicating hyperinsulinemia associated with increased adiposity. Significant ($P <0.05$) increase in insulin resistance with weight gain. Obese individuals exhibited markedly higher HOMA-IR (16.75 ± 4.67) compared to normal weight (12.27 ± 2.18) and overweight (12.76 ± 4.59), reflecting greater insulin resistance. No statistically significant difference ($P > 0.05$), though a trend toward higher TC in overweight and obese groups was noted. However, there was a highly significant ($P < 0.001$) increase in TG levels with increasing weight status. Obese individuals have markedly elevated TG (439.25 ± 10.99, mg/dL) compared to normal weight (333.30 ± 10.57, mg/dL). VLDL cholesterol, which correlates with TG levels, shows a significant increase with obesity, consistent with hypertriglyceridemia. LDL cholesterol was significantly ($P < 0.01$)

higher in the obese group (150.75 ± 8.23, mg/dL) compared to the normal weight (138.10 ± 3.16, mg/dL) and overweight groups (138.20 ± 3.98, mg/dL), indicating increased atherogenic risk. Conversely, HDL cholesterol significantly (P <0.001) decreases with increasing weight. Obese individuals have the lowest HDL (33.80 ± 3.56, mg/dL), suggesting an impaired cardioprotective lipid profile. Obesity is associated with atherogenic dyslipidemia characterized by elevated triglycerides, VLDL cholesterol, LDL cholesterol, and decreased HDL cholesterol, which collectively increase cardiovascular risk.

Table 3: Mean ± standard deviation of circulating biochemical parameters levels in T2DM according to the body mass index

Parameters	Normal weight (n=20)	Overweight (n=20)	Obese (n=20)	F-value	P-value
Glucose (mg/dL)	215.70 ± 84.07	200.30 ± 74.39	208.80 ± 64.33	0.213	0.809
HbA1c (%)	9.05 ± 1.72	9.36 ± 1.65	8.87 ± 1.48	0.467	0.629
Insulin (µlU/mL)	24.83 ± 5.36	26.64 ± 5.20	33.48 ± 6.78	12.264	< 0.001
HOMA-IR	12.27 ± 2.18	12.76 ± 4.59	16.75 ± 4.67	7.595	0.001
TC (mg/dL)	232.20 ± 8.07	237.00 ± 8.90	238.40 ± 8.82	2.855	0.066
TG (mg/dL)	333.30 ± 10.57	372.80 ± 15.14	439.25 ± 10.99	372.442	< 0.001
VLDL (mg/dL)	66.65 ± 2.11	74.56 ± 3.03	87.85 ± 2.20	373.458	< 0.001
LDL (mg/dL)	138.10 ± 3.16	138.20 ± 3.98	150.75 ± 8.23	33.956	< 0.001
HDL (mg/dL)	39.15 ± 3.51	35.95 ± 3.61	33.80 ± 3.56	11.432	< 0.001

Table 4 shows the correlation between various glycemic parameters and lipid profile components in newly diagnosed T2DM. There was a moderate positive correlation between BMI and TC (r = 0.324), significantly at p < 0.05. Very strong positive correlations exist between BMI and both triglycerides (r = 0.903) and VLDL (r = 0.903), highly significant (P < 0.001). A strong positive correlation between BMI and LDL cholesterol (r = 0.688) was observed, also highly significant (P < 0.001). A moderate negative correlation between BMI and HDL cholesterol (r = -0.450) was found, significant at P < 0.001. HbA1c and glucose were not correlated with lipid parameters and

were statistically nonsignificant (P > 0.05). Insulin had moderate positive significant correlations (P < 0.001) with TG (r = 0.557) and VLDL cholesterol (r = 0.557). Additionally, insulin had a moderate positive and significant correlation (P < 0.05) with LDL cholesterol (r = 0.369). On the contrary, insulin had a weak negative correlation with HDL cholesterol (r = -0.288), significant at p < 0.05. No significant correlation (P > 0.05) between insulin and TC. HOMA-IR had moderate positive and significant correlations (P < 0.001) with TG (r = 0.454) and VLDL cholesterol (r = 0.454). There was a moderate, positive, and significant correlation (P < 0.05) between HOMA-IR and LDL cholesterol (r = 0.340). However, there is a moderately significant negative correlation between HOMA-IR and HDL cholesterol (r = -0.307). No significant correlation (P > 0.05) between HOMA-IR and TC.

Table 4: Correlation between glycemic parameters and lipid profile in newly diagnosed type 2 diabetes mellitus

parameters	correlation	TC	TG	VLDL	LDL	HDL
BMI	r	0.324	0.903	0.903	0.688	-0.450
	P value	0.012	0.000	0.000	0.000	0.000
HbA1c	r	0.041	-0.041	-0.041	0.010	0.038
	P value	0.754	0.756	0.754	0.939	0.772
Glucose	r	-0.036	-0.048	-0.048	0.030	-0.017
	P value	0.784	0.715	0.713	0.820	0.896
Insulin	r	0.094	0.557	0.557	0.369	-0.288
	P value	0.477	0.000	0.000	0.004	0.026
HOMA-IR	r	0.042	0.454	0.454	0.340	-0.307
	P value	0.752	0.000	0.000	0.008	0.017

Discussion

The primary cause of T2DM is insulin resistance, where muscle, fat, and liver cells do not respond properly to insulin, leading to reduced glucose uptake and increased blood glucose levels [12;13]. To compensate, the pancreas produces more insulin (hyperinsulinemia), but over time, pancreatic beta cells become exhausted and cannot maintain adequate insulin production, resulting in sustained hyperglycemia [14;15]. Several studies support the notion that these biomarkers may not differ significantly solely due to age within certain adult

populations. One study indicates that insulin resistance and lipid profiles are more strongly influenced by factors such as obesity and lifestyle rather than age [16]. Age was not significantly correlated with HOMA-IR in these groups, aligning with the lack of age-related differences found in this study [16]. Thus, the absence of significant differences in HbA1c and lipid markers across age groups in a presumably non-diabetic or controlled population is consistent with a study that emphasizes disease status over age as a determinant [17]. Elevated free fatty acids and triglycerides are often linked to reduced insulin sensitivity, which tends to complications with age in the general population [18]. Some studies report no significant differences between males and females in key metabolic parameters such as glucose, HbA1c, insulin, HOMA-IR, TG, VLDL, and LDL at the time of T2DM diagnosis, a study examining insulin sensitivity and beta-cell function found no significant sex differences in insulin sensitivity, although females had higher HOMA-β and early insulin secretion indices than males, suggesting similar overall glucose metabolism between genders in newly diagnosed T2DM [19]. A study of 404 T2DM patients found significantly higher BMI, TC, LDL, and HDL levels in females than males, with statistical significance ($p < 0.05$) [20]. This aligns with findings that females generally have higher HDL levels, which is considered protective against cardiovascular disease [21]. The same study noted that despite higher HDL and TC in females, the TC: HDL ratio was significantly higher in males, indicating a potentially higher cardiovascular risk in males with T2DM [20]. Another large cross-sectional study in elderly Chinese adults found that HDL-c and triglycerides (TG) were significantly associated with T2DM prevalence in females, whereas LDL-c, HDL-c, and TC were significant in males. Moreover, lipid ratios such as LDL-c/HDL-c and TC/HDL-c were associated with T2DM prevalence only in females, highlighting gender-specific lipid profile patterns in T2DM [22]. Conversely, some studies report that higher LDL-c and TC levels are inversely associated with T2DM prevalence in men but not in women, suggesting a complex and possibly paradoxical relationship between cholesterol fractions and diabetes risk that differs by gender [22]. While females tend to have higher HDL and TC levels, the clinical significance of these differences in newly diagnosed T2DM patients is debated. Higher HDL in females does not always translate into lower cardiovascular risk, especially if other lipid ratios or glycemic control are poor [23]. The

discrepancies may stem from differences in study populations, age groups, and methodologies. Hormonal factors, such as estrogen, likely modulate lipid metabolism and cardiovascular risk differently in males and females with T2DM, contributing to observed variations [5;24].

One cross-sectional study found no correlation between BMI and HbA1c levels in newly diagnosed T2DM patients, indicating that fasting glucose and HbA1c did not significantly differ according to BMI groups [9]. This indicates that at the time of diagnosis, glycemic control markers may be independent of BMI, possibly reflecting early disease heterogeneity or other metabolic factors not captured by BMI alone. Conversely, another study highlights a significant positive correlation between BMI and HbA1c in T2DM patients, with higher BMI associated with poorer glycemic control and elevated HbA1c levels [6]. These findings emphasize the importance of weight management in diabetes care, as obesity can exacerbate insulin resistance and worsen glycemic outcomes. However, these correlations may be more pronounced in established T2DM rather than at initial diagnosis. The discrepancy between studies may be due to differences in study design, population characteristics, or the timing of measurements relative to disease progression. Additionally, fasting glucose and HbA1c reflect different aspects of glucose metabolism, fasting glucose captures momentary glycemia, while HbA1c reflects average glucose over 6-8 weeks [25]. This could contribute to variability in their relationship with BMI.

Insulin levels increase progressively from normal weight to overweight to obese, indicating hyperinsulinemia associated with increased adiposity. Significant increase in insulin resistance with weight gain. Obese individuals exhibited markedly higher HOMA-IR compared to normal weight and overweight, reflecting greater insulin resistance in the patients with newly diagnosed T2DM. Increased adipose tissue, especially visceral fat, contributes to insulin resistance by secreting pro-inflammatory cytokines and adipokines that impair insulin signaling [26]. This chronic, low-grade inflammation in adipose tissue leads to metabolic disturbances that reduce the sensitivity of muscle, liver, and fat cells to insulin. Consequently, the pancreas produces more insulin to maintain normal blood glucose levels, resulting in hyperinsulinemia [27;28]. Insulin resistance is characterized by the diminished ability of cells to respond to insulin, causing glucose to accumulate in the blood. To compensate, the pancreas secretes more insulin. However, excess insulin

promotes fat storage, particularly in adipose tissue, making weight loss difficult and often leading to further weight gain. This creates a vicious cycle where increased adiposity exacerbates insulin resistance, which in turn promotes more fat accumulation [28]. HOMA-IR is a widely used measure of insulin resistance. Studies show that obese individuals, especially those newly diagnosed with T2DM, exhibit significantly higher HOMA-IR values compared to normal-weight and overweight individuals. This indicates a greater degree of insulin resistance in obese patients, reflecting the severity of metabolic dysfunction associated with excess adiposity [27].

In patients with newly diagnosed T2DM, the relationship between obesity and lipid profile abnormalities reveals important trends and significant differences that have implications for cardiovascular risk. Studies indicate no significant difference in total cholesterol levels across normal weight, overweight, and obese T2DM patients, although there is a noted trend toward higher TC in overweight and obese groups [29]. This suggests that while TC may increase with weight status, the variability or sample size may limit statistical significance in some patients [30]. There is a highly significant increase in triglyceride levels with increasing weight status. Obese individuals exhibit markedly elevated TG compared to those of normal weight. This hypertriglyceridemia is a consistent finding and is strongly associated with obesity in T2DM patients [7]. The elevation in TG is also reflected in increased VLDL-C, which correlates with TG levels and rises significantly with obesity, reinforcing the link between obesity and atherogenic dyslipidemia [7]. LDL cholesterol is significantly higher in obese T2DM patients compared to normal weight and overweight groups, with reported mean values around 138 mg/dL or higher in obese individuals. This elevation in LDL-C contributes to increased atherogenic risk and cardiovascular disease potential in obese diabetic patients [31;32]. Conversely, HDL cholesterol levels significantly decrease with increasing weight. Obese individuals have the lowest HDL-C levels, which is concerning given HDL's protective role against cardiovascular disease. This inverse relationship between BMI and HDL-C is well documented in T2DM populations [7]. Reduced HDL-C is a common feature of diabetic dyslipidemia and contributes to impaired reverse cholesterol transport and increased atherosclerotic risk [31;33]. Diabetic dyslipidemia which involves increased TG, VLDL, LDL-C, and lowered HDL-C. Obesity-induced IR is a central mechanism driving these lipid changes,

stimulating increased hepatic lipogenesis, impaired lipoprotein clearance, and altered lipid transport. These metabolic derangements underscore the importance of weight management and lipid control in newly diagnosed T2DM patients to mitigate long-term cardiovascular complications. HbA1c alone may not be a reliable dual marker for dyslipidemia in newly diagnosed patients, unlike in those with longer disease duration [34;35].

Both insulin and HOMA-IR exhibit moderate positive and significant correlations with TG and VLDL cholesterol. This relationship is consistent with the pathophysiology of insulin resistance, where hyperinsulinemia promotes increased hepatic production of TG-rich lipoproteins and impaired clearance of TG, leading to elevated plasma TG and VLDL levels [36;37]. Similarly, insulin and HOMA-IR also show a moderate positive and significant correlation with low-density lipoprotein (LDL) cholesterol. Insulin resistance is associated with increased levels of small dense LDL particles, which are more atherogenic and commonly elevated in T2DM [36]. This dyslipidemic pattern contributes to the heightened cardiovascular risk observed in these patients. Insulin resistance and hyperinsulinemia are linked to decreased HDL cholesterol levels, which further exacerbates cardiovascular risk. The decrease in HDL is partly due to altered lipoprotein metabolism and reduced activity of enzymes such as lecithin-cholesterol acyltransferase (LCAT), which is regulated by insulin and plays a role in HDL maturation [36;37]. Interestingly, insulin and HOMA-IR did not show a significant correlation with TC levels in newly diagnosed T2DM patients. This may be because TC includes all cholesterol fractions, and the opposing changes in LDL (increased) and HDL (decreased) may offset each other, resulting in a net neutral effect on TC [36;38].

These correlations underscore the role of insulin resistance in the dyslipidemia commonly observed in early T2DM. Elevated TG and VLDL reflect increased hepatic lipogenesis and impaired clearance, while raised LDL and lowered HDL levels indicate qualitative changes in lipoproteins that contribute to atherogenesis [39]. The lack of significant correlation with TC suggests that focusing on individual lipid fractions rather than TC is more informative in assessing cardiovascular risk in these patients. The lack of significant differences in this study might be due to the relatively small sample size (20 participants per group), which may limit statistical power to detect subtle age-related changes. Additionally, factors such as physical activity, diet, medication use, and the presence of

comorbidities were not specified but could influence these metabolic markers independently of age or sex [40].

Conclusion

This study revealed that age and sex generally do not significantly affect glycemic control or lipid profiles, except that females tend to have higher TC and HDL cholesterol than males. In contrast, obese patients show markedly higher insulin levels and insulin resistance (HOMA-IR), indicating hyperinsulinemia with increasing obesity. Obesity is also linked to an atherogenic lipid profile, characterized by elevated TG, VLDL, LDL cholesterol, and reduced cardioprotective HDL cholesterol. These findings underscore obesity's critical role in exacerbating insulin resistance and dyslipidemia in early T2DM, independent of age and sex, highlighting the importance of early weight and lipid management to reduce cardiovascular risk and improve long-term outcomes in this population.

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تقييم تأثير العمر والجنس ومؤشر كتلة الجسم على مستويات السكر والدهون لدى المرضى حديثي التشخيص بمرض السكري من النوع الثاني: دراسة مقطعية

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الخلاصة

الهدف: يُظهر المرضى المشخّصون حديثًا بداء السكري من النوع الثاني ملفات أيضية متنوعة تتأثر بعوامل مثل العمر، الجنس، ومؤشر كتلة الجسم، والتي تؤثر على تقدم المرض وخطر الإصابة بمشاكل صحية أخرى. يهدف هذا البحث إلى تقييم تأثير العوامل الديموغرافية مثل العمر، الجنس، ومؤشر كتلة الجسم على التحكم في نسبة الكلوكوز في الدم ومؤشرات ملف الدهون لدى المرضى المشخّصين حديثًا بداء السكري من النوع الثاني.

المواد وطُرُق العمل: شملت الدراسة 60 مريضًا مشخّصًا حديثًا بداء السكري من النوع الثاني، تراوحت أعمارهم بين 30 و75 سنة. جمعت عينات الدم بعد صيام طوال الليل؛ حيث قسمت إلى جزئين، الجزء الاول استخدم أنبوب يحتوي مانع تخثر (EDTA) وقياس السكر التراكمي مباشرة، بينما في الجزء الاخر تم فصل مصل الدم لتحليل المؤشرات الكيميائية الحيوية. شملت القياسات الكيميائية الحيوية كلوكوز الدم وملف الدهون باستخدام الطرق الإنزيمية، والسكر التراكمي باستخدام تقنية الكروماتوغرافيا السائلة عالية الأداء بتبادل الأيونات، والإنسولين بطريقة الكشف المناعي الكيميائي الكهروضوئي. تم حساب مقاومة الإنسولين باستخدام معادلة HOMA-IR.

النتائج: لم تُظهر المؤشرات البيوكيميائية اختلافاً معنوياً وفقًا للعمر أو الجنس باستثناء ارتفاع الكوليسترول الكلي والكوليسترول الجيد لدى الإناث. كما ارتبطت السمنة بشكل قوي بزيادة مقاومة الإنسولين وتدهور ملف الدهون، والذي تميز بارتفاع الدهون الثلاثية، كوليسترول البروتين الدهني منخفض الكثافة جدًا ، الكوليسترول السيء ، وانخفاض الكوليسترول الجيد. لم ترتبط مؤشرات ضبط السكر في الدم مثل الكلوكوز والسكر التراكمي بشكل معنوي باضطرابات الدهون.

الاستنتاج: توصلت الدراسة إلى أن العمر والجنس لا يؤثران على ضبط سكر الدم وملف الدهون، باستثناء أن الإناث لديهن مستويات أعلى من الكوليسترول الكلي والكوليسترول الجيد لدى النساء. ومع ذلك، ترتبط السمنة ارتباطًا وثيقًا بزيادة مقاومة الإنسولين وتدهور ملف الدهون.

الكلمات المفتاحية: داء السكري من النوع الثاني، مقاومة الإنسولين، الكلوكوز، السمنة، الدهون.