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EFFECT OF HBA1C CUMULATIVE SUGAR VALUES ON BMI AND LIPID IN SAUDI MALES WITH TYPE 2 DIABETES FOR PEOPLE VISITING SOME MEDICAL LABORATORIES IN SAUDI ARABIA

Amani Mahdi Al-Habib[1], Ishtyak ahmad abbas al habeeb[1], Zahra Ahmed Al_Rebh[1], Roqaiah Ali Almarhoun[1], Yasmeen Ali Almuslab[2], Suad Abdullah al-nasser[1], Fatimah Reda Alnasser[1,] Nada Abdulmohsen Al Faraj[3], Waheeda Khalil Abuqurain[1], Naeemah Hussain Al Qanbar[1], Huda Ahmed Al-Ghamdi[1], Fatimah Ali Alawami[1]

[1] Staff Nurse 1, National Guard Hospital-KSA

[2] Dental Assistant II-National Guard Hospital-KSA

[3] Staff Nurse 1 (Patient Educator)-National Guard Hospital –KSA

Abstract:

The present study was designed to evaluate the blood sugar level and the duration of diabetes mellitus and its relationship to body mass index (BMI) and lipid profile.

The current study included 90 male samples aged between 40-70 years. 60 of them were diagnosed with type 2 diabetes. The control group included 30 samples of healthy individuals who did not suffer from any chronic disease, aged between 40-65 years.

The current study included the measurement of blood glucose level (Glucose), accumulated blood sugar (HbA1c), body mass index (BMI), total cholesterol (Total Cholesterol), triglycerides (Triglyceride), high-density lipoprotein (HDL-C), low-density lipoprotein (LDL-C), very low-density lipoprotein (VLDL-C), and atherosclerosis indices (I, II, and III) in patients with type 2 diabetes.

The results of the current study showed differences that ranged between highly significant and significant when compared with the control group. The results showed highly significant differences ($P < 0.01$) between all diabetics and the control group in terms of (BMI, Glucose, and HbA1c).

When comparing the results of the lipid profile variables between the patients in general and the control group, there was a significant decrease ($P > 0.05$) in the total cholesterol level of the patient group and a highly significant increase ($P < 0.01$) in both TGVLDL and atherosclerosis indices I, II, and III. While HDL decreased significantly ($P < 0.01$), there was no significant difference in LDL level when compared with the control group.

Through the results of the current study, we conclude that type 2 diabetes, accumulated blood sugar level (HbA1c), and the duration of infection contributed to many changes in the studied biological variables and changes in the values of the lipid profile and atherosclerosis indices, which are the most important indicator of cardiovascular diseases.

Keywords: Diabetes, Lipid Profile, Accumulated Blood Sugar HbA1c, Body Mass Index BMI, Triglycerides TG.

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Introduction:

Diabetes mellitus is a group of diseases, not a single disease, characterized by high blood sugar levels (hyperglycemia) resulting from a combination of genetic and environmental factors. Diabetes is also a chronic inflammatory disease characterized by chronic hyperglycemia associated with the generation of ROS, oxidative stress, and peroxidation of biomolecules, leading to complications affecting the blood vessels. [1]

Metabolic disorders such as hyperglycemia, altered lipid profile, carbohydrate and protein metabolism, and increased risk of cardiovascular complications are associated with diabetes. Increased blood glucose levels lead to glycosylation of blood cell proteins, a process called glycation, which is used as a marker to measure the duration of the disease because it is linked to the lifespan of red blood cells.[2]

The glycosylated hemoglobin A1c (HbA1c) test is one of the diagnostic methods for diabetes and also to monitor glucose levels over the long term. The normal blood sugar level in healthy individuals is 70-110 mg/100ml. When its concentration increases, the hormone insulin is secreted, which works to convert excess sugar into glycogen, which is stored in the liver and muscles. HbA1c represents a marker of the average blood glucose concentration over 8-12 weeks before the test because the concentration of accumulated blood sugar is related to the concentration of glucose that red blood cells, which have a lifespan of four months, are exposed to. [3]

Body Mass Index (BMI) is a measure of human body mass based on an individual's weight and height. It is considered the best way to measure overweight, which is a calculation that depends on measuring both weight and height. BMI is calculated by dividing body weight in kilograms by the square of height in meters. [4]

This calculation is less accurate in the case of athletes who have excess weight due to muscle mass rather than body fat. In this case, different methods can be used to determine body fat to find out if the weight gain is due to obesity or muscle growth only. [5]

Causes of Increased Risk of Atherosclerosis in Diabetes

The causes of increased risk of atherosclerosis in diabetes are multifactorial, but one of the important factors that contribute to the development of this disease is the high rate of synthesis and storage of triglycerides (TG) in the liver and adipose tissues. This leads to an increase in TG levels and very low-density lipoprotein (VLDL) particles, as well as small, dense LDL-C particles, while HDL-C levels are reduced. While this pattern of lipid abnormalities is very common in patients with type 2 diabetes (T2DM), it is less common in patients with type 1 diabetes (T1DM). [6]

Lipid Profile in Diabetes

Triglycerides (TG) are fats that are insoluble in water and soluble in non-polar solvents. Fats have several structures and metabolic functions. Fats are associated with proteins to form lipoproteins, which move easily in the blood serum to be transported to various organs in the body. [7]

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Total Cholesterol (TC) is a fatty molecule found in the diet and is made in the liver and intestines. The body uses cholesterol to make hormones, bile acids, vitamin D, and other substances. Cholesterol circulates in the bloodstream but cannot flow on its own. Two main types of lipoproteins carry cholesterol in the blood: LDL-C and HDL-C. [8]

The primary function of **HDL-C** is to transport cholesterol from various tissue cells to the liver for oxidation. An association has been observed between HDL concentration and the risk of atherosclerosis. [9]

Low-density lipoprotein (LDL-C) is a type of lipoprotein that carries cholesterol from the liver to the cells of the body. High LDL-C levels are associated with an increased risk of atherosclerosis, heart disease, and stroke. [10]

Very-low-density lipoprotein (VLDL) is a type of lipoprotein that carries triglycerides from the liver to the cells of the body. VLDL particles can be converted to LDL particles in the bloodstream. [11]

Atherosclerosis is a hardening and narrowing of the arteries caused by the buildup of plaque, a fatty substance that contains cholesterol, triglycerides, and other inflammatory cells. Atherosclerosis can reduce blood flow to the heart, brain, and other organs, leading to heart attack, stroke, and other serious health problems. [12]

Low-density lipoprotein (LDL-C) These particles are made in the liver and intestinal epithelial cells and are also formed by the breakdown of VLDL-C by the enzyme LPL, which converts them to intermediate-density lipoprotein (IDL) and then to LDL-C. These lipoproteins work to carry cholesterol from the liver and transport it to other tissues of the body. [13]

Very Low-density lipoprotein (VLDL), This type of lipoprotein is made primarily in the liver and may also be made in the intestines. It is characterized by its low density and contains small amounts of cholesterol and protein. It works to transport triglycerides (TG) from the liver to the rest of the tissues in the body. [14]

Aim of the study

The study aims to evaluate the effect of the level of accumulated sugar on body mass index and its effect on the variables of the lipid profile.

Importance of Research

This research is significant for several reasons. Firstly, it addresses a critical health concern, namely, Type 2 diabetes, which is increasingly prevalent in Saudi Arabia. Secondly, it focuses on the relationship between HbA1c cumulative sugar values, body mass index (BMI), and lipid levels, shedding light on potential correlations that can inform diabetes management strategies. Lastly, by targeting Saudi males, the study aims to contribute to the understanding of diabetes risk factors within a specific demographic, aiding in the development of tailored interventions.

Problems and Research Questions

1. **Lack of Understanding:** Despite the prevalence of Type 2 diabetes in Saudi Arabia, there may be insufficient awareness regarding the impact of HbA1c cumulative sugar values on BMI and lipid levels among affected individuals.

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- Research Question: What is the level of understanding among Saudi males with Type 2 diabetes regarding the relationship between HbA1c cumulative sugar values, BMI, and lipid levels?

2. **Health Outcomes:** Poorly managed diabetes can lead to various health complications, including cardiovascular diseases. Understanding the relationship between HbA1c, BMI, and lipid levels can provide insights into potential health outcomes.
    - Research Question: How do HbA1c cumulative sugar values correlate with BMI and lipid levels among Saudi males with Type 2 diabetes, and what are the potential implications for their health outcomes?
3. **Management Strategies:** Effective diabetes management requires a comprehensive understanding of the factors influencing glycemic control and lipid metabolism. Identifying the impact of HbA1c cumulative sugar values on BMI and lipid levels can inform personalized management strategies.
    - Research Question: What strategies can be recommended for improving glycemic control and lipid management based on the relationship between HbA1c cumulative sugar values, BMI, and lipid levels among Saudi males with Type 2 diabetes?

Methodology

A special questionnaire was prepared in which information was collected about people with type 2 diabetes and healthy people. Blood was drawn from the study samples using a 10 ml syringe and then the blood was divided for each sample into two parts: one part in an EDTA tube to perform the HbA1c test and the other part in a plain tube to perform the lipid profile test.

Table 1: represents the materials used in the study

no.	Materials	Company	Origin
1	Glucose analysis kit	Linear	Spain
2	Cholesterol analysis kit	Human	Germany
3	Triglycerides analysis kit	Human	Germany
4	High-density lipoprotein (HDL) analysis	Human	Germany
5	Hemoglobin A1c analysis kit	Cobas e 111 Elecsys Roche	Mannheim Germany

Study sample

The study included (120) samples, of which (80) samples were for people with type 2 diabetes (the experimental group) and (40) samples for healthy people (the control group).

Research Time Domain:

The Study period from October 2015 to February 2016. The ages of the people with diabetes ranged between 80-40 years, and the ages of the healthy people ranged between 40-65 years. Diabetes patients were diagnosed by specialists at the medical laboratories in hospitals and medical centers located in the Eastern Province of Saudi Arabia.

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Data collection

The experimental and control group was collected from the medical laboratories in hospitals and medical centers located in the Eastern Province of Saudi Arabia.

Data for this study are collected from ten medical laboratories in hospitals and medical centers located in the Eastern Province of Saudi Arabia

Research Tools:

The study employed the following research tools to collect and analyze data:

Questionnaire: A special questionnaire was prepared in which information was collected about people with type 2 diabetes and healthy people.

Blood Sampling: Blood was drawn from the study samples using a 10 ml syringe and then the blood was divided for each sample into two parts: one part in an EDTA tube to perform the HbA1c test and the other part in a plain tube to perform the lipid profile test.

Calculation of body measurements:

Body mass index (BMI) was calculated using the common method by dividing weight by height squared in units of (Kg/m2) as in the following equation:

$$\mathrm{BMI} = \frac{\mathrm{mass(kg)}}{(\mathrm{height(m)})^2}$$

Equation 1: BMI Equation [14]

Table 2: represents the classification of weight according to body mass index.

Weight classification	Obesity	BMI (Kg/m2)	Origin
Underweight	-	< 18.5	Low
Normal	-	18.5-24.9	Normal
Overweight	-	25.0-29.9	Increased
Obesity	Obesity	30.0-34.9	High
	Sever obese	35.0-39.9	Very high
	Morbidly obese	≥40.0	Extremely High

Laboratory Tests:

Estimation of fasting serum glucose concentration in serum

The concentration of glucose in the serum was estimated using the enzymatic colorimetric method "Enzymatic calorimetric method" using the ready-made kit.

Estimation of the level of accumulated sugar in the blood (HbA1C)

The level of accumulated sugar in the blood (HbA1C) was estimated using the turbidimetric inhibition immunoassay (TINIA) method for whole blood.

Method of work

The method of work was carried out according to the manufacturer's instructions (111 Elecsys Roche Cobase).

Estimation of serum cholesterol level

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"The serum cholesterol level was estimated depending on the enzymatic method using the ready-made analysis kit.

$$\text{Cholesterol concentration [mg/dL]} = \frac{\text{A sample}}{\text{A standard}} \times 200$$

$$\text{Normal value} = 150 - 200 \text{ mg/dl}$$

Equation 2: Estimation of serum cholesterol level [15]

Estimation of serum triglyceride concentration

Serum triglycerides were estimated using the enzymatic method using the ready-made analysis kit.

$$\text{Cholesterol concentration [mg/dL]} = \frac{\text{A sample}}{\text{A standard}} \times 200$$

$$\text{N.V} = 65 - 150 \text{ mg/dl}$$

Equation 3: Estimation of serum triglyceride concentration [16]

Estimation of serum high-density lipoprotein (HDL-C) level

The serum HDL-C level was estimated using the enzymatic method using the ready-made analysis kit.

$$\text{HDL-C Concentration [mg/dl]} = \frac{\text{A sample}}{\text{A standard}} \times 150$$

$$\text{N.V} = 34.7 - 45 \text{mg/dl}$$

Equation 4: Estimation of serum high-density lipoprotein (HDL-C) level [17]

Estimation of serum low-density lipoprotein (LDL-C) level

The serum LDL-C level was calculated using the following equation:

$$\text{LDL-C con. [mg/dl]} = \text{Cholesterol con.} - (\text{HDL con.} + \text{VLDL con.})$$

N. V= 69-166mg/dl

Equation 5: Estimation of serum high-density lipoprotein (HDL-C) level [18]

Estimation of serum very low-density lipoprotein (VLDL-C) level

The serum VLDL-C level was calculated using the following equation:

$$\text{VLDL-C con. [mg/dl]} = \text{TG con.} / 5$$

N.V= 8-30 mg/dl.

Equation 6: Estimation of serum very low-density lipoprotein (VLDL-C) level [19]

1.10.2 Estimation of bias indicators levels

Bias indicator levels were estimated according to the following equations:

A - First bias indicator = TC | HDL

B - Second bias indicator = LDL-C | HDL

C - Third bias indicator = LDL-C+ VLDL-C / HDL

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Statistical analysis

The results obtained by the researchers will be displayed and analyzed, Data were fed to the PC and analyzed using IBM SPSS software package version 20.0. (Armonk, NY: IBM Corp). We will display the arithmetic means of the questionnaire responses obtained from the sample and present the standard deviations to identify the degree of variation in those responses by displaying the frequencies and their percentages to identify the level of responses about the variables.

Results

The results are represented in Table (3) which summarizes the values of HbA1c (glycated hemoglobin), glucose, and BMI (Body Mass Index) for two distinct groups: a control group and a group comprising patients. Each parameter is analyzed across the two groups to discern any significant differences.

Beginning with BMI, the mean BMI for the control group is recorded at 22.28 kg/m² with a standard deviation of ±0.876, while the patient group exhibits a notably higher mean BMI of 32.21 kg/m² with a standard deviation of ±7.55. Statistical analysis reveals a significant discrepancy in BMI between the two groups ($p = 0.0004$), indicating that patients possess significantly higher BMI values compared to the control subjects.

Moving on to glucose levels, the mean glucose concentration in the control group is 87.91 mg/dl with a standard deviation of ±8.26, whereas the patient group displays a substantially elevated mean glucose level of 4.86 mg/dl with a standard deviation of ±0.63. The statistical assessment underscores a significant difference in glucose levels between the control and patient groups ($p = 0.0004$), signifying markedly elevated glucose levels among the patients in contrast to the control group.

Lastly, the analysis of HbA1c levels reveals a mean value of 169.2 with a standard deviation of ±54.7 for the control group, while the patient group demonstrates a mean HbA1c level of 8.92 with a standard deviation of ±1.45. Once again, the statistical evaluation indicates a significant contrast in HbA1c levels between the control and patient groups ($p = 0.0005$), highlighting substantially higher HbA1c values among patients compared to the control subjects.

Table 3: Values of (HBA1C, Glucose, BMI) for the studied samples

Group Parameter	Control	Patient	P≤	P-value
No.	40	80	***	***
BMI(kg/m2)	22.28±0.876	32.21±7.55	0.01	0.0004
Glucose(mg/dl)	87.91±8.26	4.86±0.63	0.01	0.0004
HBA1C	169.2±54.7	8.92±1.45	0.01	0.0005

Lipid profile and bias indicator concentrations for the studied samples

Table 4 delineates the lipid profile concentrations and indicators of radicalization observed within both the control and patient groups in the studied samples. The table offers crucial insights into various lipid parameters and their potential implications for health outcomes among the studied cohorts.

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Beginning with cholesterol levels, the control group exhibits a mean cholesterol concentration of 164.6 mg/dl with a standard deviation of ±22.8, while the patient group demonstrates a slightly lower mean cholesterol level of 150.5 mg/dl with a standard deviation of ±29.9. Although the difference in cholesterol levels between the two groups approaches statistical significance ($p = 0.056$), further investigation may be warranted to ascertain its clinical relevance.

Moving on to triglyceride levels, the control group displays a mean triglyceride concentration of 95.9 mg/dl with a standard deviation of ±31.9, contrasting with the patient group, which manifests a substantially higher mean triglyceride level of 130.6 mg/dl with a standard deviation of ±76.6. Statistical analysis reveals a significant discrepancy in triglyceride levels between the control and patient groups ($p = 0.009$), indicating elevated triglyceride concentrations among patients compared to the control subjects.

The analysis of HDL levels unveils a notable contrast between the two groups, with the control group showcasing a mean HDL level of 50.1 mg/dl and the patient group exhibiting a markedly lower mean HDL level of 27.81 mg/dl. This discrepancy in HDL levels between the control and patient groups is highly statistically significant ($p = 0.0002$), underscoring substantially reduced HDL concentrations among patients compared to the control cohort.

Similarly, the examination of LDL levels elucidates a significant difference between the control and patient groups. While the control group demonstrates a mean LDL level of 104.4 mg/dl, the patient group displays a lower mean LDL level of 91.8 mg/dl. This variance in LDL levels between the two groups reaches statistical significance ($p = 0.009$), indicating comparatively diminished LDL concentrations among patients relative to the control subjects.

Lastly, the evaluation of VLDL levels underscores another noteworthy difference between the control and patient groups. The control group presents a mean VLDL level of 19.93 mg/dl, whereas the patient group exhibits a higher mean VLDL level of 26.8 mg/dl. Statistical analysis indicates a significant disparity in VLDL levels between the two groups ($p = 0.009$), suggesting elevated VLDL concentrations among patients in contrast to the control individuals.

Table 4: Lipid profile concentrations and indicators of radicalization for the studied samples

Group Parameter	Control	Patient	P≤	P-value
No.	40	80	***	***
cholesterol	164.6 ± 22.8	150.5 ± 29.9	0.05	0.056
Triglycerides	95.9 ± 31.9	130.6 ± 76.6	0.01	0.009
HDL	50.1 ±11.5	27.81 ±6.81	0.01	0.0002
LDL	104.4 ±22.3	91.8 ±24.4	0.05	0.009
VLDL	19.93 ±6.43	26.8 ±15.1	0.01	0.009

Table 5 provides a detailed breakdown of biases observed within the studied samples, comparing data between the Control and Patient groups.

Bias One: Bias one, a critical parameter, exhibits notable discrepancies between the Control and Patient groups. In the Control group, bias one is reported at 3.482 with a standard deviation of ±0.928, while in the Patient group, it significantly rises to 5.89 with a standard

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deviation of ±1.73. Statistical analysis reveals a significant difference (p = 0.0003) between the two groups, indicating distinct biases that warrant further investigation.

Bias Two: Similarly, bias two displays significant differences between the Control and Patient groups. In the Control group, bias two is documented as 2.105 with a standard deviation of ±0.881, whereas in the Patient group, it elevates to 3.83 with a standard deviation of ±1.24. Statistical evaluation underscores a significant contrast (p = 0.0002) between the two groups, suggesting diverse biases that merit thorough scrutiny.

Bias Three: The analysis of bias three further accentuates disparities between the Control and Patient groups. In the Control group, bias three is recorded at 2.495 with a standard deviation of ±0.919, while in the Patient group, it markedly increases to 4.935 with a standard deviation of ±1.70. Statistical significance (p = 0.0003) is once again observed, underscoring substantial differences in biases between the two groups.

Overall Implications: The findings underscore significant variations in biases between the Control and Patient groups across all three parameters. These disparities highlight the necessity of considering biases when interpreting study results, particularly in comparative analyses involving different groups or populations. Understanding and addressing biases are crucial for ensuring the accuracy and reliability of study findings, ultimately enhancing the validity of conclusions drawn from the data. Further research and meticulous attention to biases are warranted to elucidate their impact on study outcomes and refine methodologies for future investigations.

Table 5: Values of Bais for the studied samples

Group Parameter	Control	Patient	P≤	P-value
No.	40	80	***	***
bias one	3.482±0.928	5.89±1.73	0.05	0.0003
bias Two	2.105±0.881	3.83±1.24	0.01	0.0002
bias Three	2.495±0.919	4.935±1.70	0.01	0.0003

Discussion

Body Mass Index (BMI) and Blood Sugar Level (%BMI, Glucose, HBA1C, and cumulative sugar (for the study samples):

The results in Table 3 showed significant differences ($P \leq 0.01$) between all diabetic patients and the control group regarding Body Mass Index (BMI). The reason may be the patients' failure to adhere to a healthy diet, consistent with the findings of the researcher (4), concerning fasting blood glucose concentration, indicating elevated blood glucose levels in T2DM patients due to increased insulin resistance, reducing insulin's ability to carry glucose into cells. Consequently, glucose remains accumulated in the blood. [20]

Regarding cumulative sugar, the results showed significant differences between the experimental and control groups, consistent with the findings of researchers, where the cumulative sugar ratio increased due to elevated VLDL-C and decreased protective HDL-C. The HbA1c test has now become a better diagnostic criterion in the United States with more biological stability within individuals compared to Fasting Plasma Glucose (FPG) levels. [21]

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Another study indicated an increase in HbA1c levels in patients with type 2 diabetes compared to the control group. It also indicated a positive correlation between HbA1C and total cholesterol, HbA1c & LDL, HbA1c, and triglycerides, with an inverse correlation between HbA1c & HDL. [22]

Lipid profile concentrations and indicators of atherosclerosis for the study samples:

The results revealed significant differences between the two groups concerning lipid profile concentrations, albeit within the normal range. This was attributed to the patient's use of Atorvastatin tablets, which proved more effective in reducing lipid parameters and consequently lowering lipid levels, as indicated by researchers. [23]

However, the current study indicated a decrease in HDL-C levels in the experimental group compared to the control group, contrasting with the findings of researchers, who did not find significant differences in their study on diabetic patients without comorbidities compared to a group of healthy individuals. Other studies have linked high levels of coronary heart disease, resulting from arterial sclerosis, to elevated total cholesterol levels in diabetic patients. The increased consumption of saturated fats, coupled with an inconsistent calorie intake among diabetic patients, has been implicated in raising blood cholesterol levels, particularly LDL-C. This was highlighted by researchers. [24]

It is crucial to maintain a balance between deposition and elimination processes, as an imbalance, such as an excessive intake of dietary cholesterol exceeding daily requirements, can disrupt this equilibrium. High levels of cholesterol increase the production of free radicals, which may impair the activity of nitric oxide synthase, leading to vasoconstriction. Moreover, a significant portion of cholesterol converts to LDL-C due to oxidation and glycation processes in diabetic individuals, leading to increased total cholesterol levels. However, lipid-lowering therapies, particularly Atorvastatin, restore the physiological availability of nitric oxide, a key mediator of vasodilation. [25]

As for triglycerides, researchers reported an increase in their levels in diabetic and hypertensive patients compared to healthy individuals, attributed to metabolic disturbances in diabetes where the body relies on fat breakdown from adipose tissues for energy due to the body's inability to utilize blood glucose. This leads to elevated triglyceride levels in the blood. Interestingly, our study found no significant differences in LDL-C levels, possibly due to patients using Atorvastatin, which is highly effective in reducing LDL-C levels and moderately effective in lowering TG and raising HDL-C levels. [26]

Our findings align with those of researchers regarding increased atherosclerosis indicators in the experimental group compared to the control group, attributed to oxidative stress induced by hydrogen peroxide. Additionally, the elevated levels of atherosclerosis indicators (first, second, and third) were attributed to the large amounts of free radicals produced by foam cells, contributing to lipid oxidation, particularly LDL-C, thereby enhancing atherosclerosis. [27]

These results were consistent with those of researchers, who found a significant increase in atherosclerosis indicators due to oxidative stress. Phospholipids play a crucial role in atherosclerosis development, accumulating in the intima layer of arteries, thereby promoting

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plaque formation. This process, in turn, stimulates foam cell formation and free radical production, both of which contribute significantly to atherosclerotic plaque formation. [10, 11]

Conclusions:

Our study findings underscore significant associations between type 2 diabetes incidence, HbA1c levels, and the duration of illness with various biological variables, lipid profile values, and indicators of atherosclerosis. These findings highlight the intricate interplay between diabetes-related factors and cardiovascular health, emphasizing the importance of comprehensive management strategies in diabetes care.

Recommendations:

1. Further Research: Conduct longitudinal studies to explore the long-term effects of type 2 diabetes management strategies on liver and kidney function, as well as on physiological variables related to diabetes disorders and complications. Additionally, investigates the impact of novel therapeutic interventions on mitigating cardiovascular risks associated with diabetes.
2. Public Health Initiatives: Implement public health programs aimed at raising awareness about the importance of glycemic control and regular monitoring of HbA1c levels among individuals with type 2 diabetes. Promote lifestyle modifications, including healthy dietary habits and regular physical activity, to improve overall cardiovascular health in diabetic populations.
3. Healthcare Provider Education: Offer training and educational resources to healthcare providers on the significance of comprehensive diabetes management, including the monitoring of lipid profiles and atherosclerosis indicators. Encourage multidisciplinary approaches to diabetes care that address both glycemic control and cardiovascular risk reduction.
4. Patient Empowerment: Empower individuals with type 2 diabetes to actively engage in their care by providing them with access to resources and support networks for managing their condition. Encourage regular follow-up appointments and adherence to prescribed medications to optimize health outcomes and reduce the risk of cardiovascular complications.
5. Policy Implications: Advocate for policies that prioritize diabetes prevention and management efforts, including increased access to affordable healthcare services, medications, and preventive screenings for at-risk populations. Collaborate with policymakers to develop initiatives aimed at reducing the burden of diabetes-related cardiovascular disease on healthcare systems and society as a whole.

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