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ASSESSMENT OF LIPID AND OTHER RISK FACTORS IN CVD

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ABSTRACT

This research reveals that individuals with cardiovascular disease have severe postprandial hypertriglyceridemia and a notable delay in the clearance of postprandial triglycerides. In this investigation, individuals with cardiovascular disease also exhibited increased values of an estimated variable. Data imply that measures linked to postprandial lipemia should be included in the evaluation and treatment of atherosclerosis, and the current study may lead to more focus on the monitoring and management of dyslipidemia in cardiovascular patients.

KEYWORDS: cardiovascular disease, postprandial lipemia, blood glucose levels, evaluation

INTRODUCTION

Postprandial lipemia adds to the inflammatory condition in the endothelium milieu, which in turn promotes atherogenesis and endothelial dysfunction. Both in vitro and in vivo investigations have shown that postprandial lipemia activates leukocytes, which in turn promote atherosclerosis by adhering to endothelial walls and migrating to the subendothelial area. Endothelial inflammation and an increase of pro-inflammatory cells inside the vascular walls may be caused by VLDL, IDL, and chylomicron remnants. The production of pro-inflammatory cytokines by TG and TGRLs leads to the adherence of monocytes and the expression of the vascular cell adhesion molecule (VCAM)-1 in endothelial cells. Endothelial cells and the lipoprotein lipase (LPL) enzyme break down trans glyceride receptor ligands (TGRLs), releasing inflammatory and atherogenic by-products. Oxidized free fatty acids, which are byproducts of lipolysis, increase ROS, endothelial inflammation, and vascular apoptosis. Endothelial inflammation enhances VDL absorption and permeability in the vascular wall.

Endothelial dysfunction, as assessed by flow-mediated dilatation (FMD) of the brachial artery, is a consequence of postprandial levels of remnant lipoproteins (RLP) and TG, according to Maggi et al. They proved a correlation between lower FMD and higher postprandial RLP and TG levels. The most endothelial dysfunction also occurred around 6 hours following a meal, when RLP levels were at their highest. Their results are in line with those of research by Caringal et al. that used FMD as a surrogate measure to examine the connection between endothelial dysfunction and postprandial lipid levels. A typical low-fat diet was administered to five high-risk individuals who had normal fasting lipid levels. Curiously, although the fasting lipid levels were within normal range, the peak of total cholesterol and very low-density lipoprotein 6 hours after the injection and the subsequent decline in HDL levels seem to correspond with the reduction in femoral artery flow-mediated dilation (FMD).

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LITREATURE REVIEW

Chahal (2021) In type 2 diabetes mellitus (DM), postprandial dyslipidemia is a key player in the development of atherosclerosis and potential macrovascular consequences. The purpose of this study is to analyze and contrast the lipid profiles of type 2 diabetic individuals both before and after meals. The medical division of a large academic medical center served as the site and investigator for this case-control research. Procedures and ingredients: Fifty people with type 2 diabetes and fifty healthy controls, matched for age and gender, made up the study's 100 participants. Lipid levels were measured in all participants both before and after food consumption and compared. The statistical analysis that was used included the student's t-test for normally distributed data and the analysis of variance (ANOVA) test for data that was to be compared between more than two groups. The outcomes are: Mean total cholesterol (TC), triglyceride (TG), low density lipoprotein (LDL), and very low density lipoprotein (VLDL) levels were significantly higher and high density lipoprotein (HDL) level was significantly lower in the diabetics in comparison to the controls in both fasting (200.82, 172.59, 126.20, 37.63, and 40.74 mg/dL in diabetics versus 179.90, 98.03, 109.54, 19.60, and 50.46 mg/dL in controls) and postprandial states (223.75, 232.99, 139.19, 46.52, and 40.54 mg/dL in diabetics versus 185.36, 102.20, 110.36, 20.24, and 48.96 mg/dL in controls). Fasting TC and TG levels in diabetic individuals were 200.82 and 172.59 mg/dL, respectively; postprandial TC and TG levels were 223.75 and 232.99 mg/dL, respectively. In summary: Significant postprandial lipid abnormalities, especially postprandial hypertriglyceridemia, are seen in individuals with type 2 diabetes. In type 2 diabetes mellitus, elevated postprandial lipid parameters emphasize the importance of postprandial lipid assessment in comparison to fasting lipid measurement.

Yanai (2023) Atherogenic postprandial hyperlipidemia, defined as a rise in serum triglyceride (TG) levels after a meal, is linked to the onset of coronary artery disease (CAD). Although the oral fat loading test (OFLT) is necessary for the diagnosis of postprandial hyperlipidemia, it is a laborious and tedious procedure. Cholesterol-rich lipoproteins (TRL) such chylomicrons (CM), very low-density lipoproteins (VLDL), and remnants of these lipoproteins (CM remnant [CMR] and VLDLR) are more abundant in the blood when serum TG levels are high. If we know that CMR and/or VLDLR levels are elevated, we may deduce that postprandial hyperlipidemia is present. One way to suspect the presence of postprandial hyperlipidemia is to measure apo B48, a component of CM and CMR, non-fasting TG, which includes TG content in all lipoproteins (including CM and CMR), non-HDL-C, which includes TRL and low-density lipoprotein, and remnant cholesterol. Patients with metabolic syndrome, type 2 diabetes, chronic renal disease, familial combination hyperlipidemia, family type III hyperlipoproteinemia, or chronic kidney disease are more likely to have postprandial hyperlipidemia. There is strong evidence that insulin resistance is a risk factor for both postprandial hyperlipidemia and postprandial hyperglycemia, and that the two conditions are closely associated to one another. Postprandial hyperlipidemia and its metabolic abnormalities, including remnant lipoproteins, induce inflammation and endothelial dysfunction, two of its atherogenic features. In order to improve postprandial hyperlipidemia, one should eat healthily, limit calories, lose weight, and exercise regularly. Pemafibrate, bezafibrate, ezetimibe, and eicosapentaenoic acid are

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anti-hyperlipidemic medications that have been shown to treat postprandial hyperlipidemia. Metformin, pioglitazone, dipeptidyl-peptidase-4 inhibitors, alpha-glucosidase inhibitors, and glu-cagon like peptide 1 analogues are anti-diabetic medications that have been shown to improve postprandial hyperlipidemia. While there is little evidence that sodium glucose cotransporter-2 inhibitors may lower postprandial hyperlipidemia, they did lower residual lipoprotein cholesterol and fasting apo B48. Last but not least, knowing the signs of postprandial hyperlipidemia and how to manage it effectively are crucial.

Huggins (2022) Delays or impairments in the breakdown of triglyceride (TG) from chylomicrons (TG derived from the diet) and very low-density lipoprotein (TG derived from the liver) following a meal led to an increase in the production of cholesterol-rich remnants, an increase in the production of small dense LDL particles, and a decrease in HDL. This trait has the potential to cause cardiovascular disease and is called a pro-atherogenic phenotype. Dietary variables, meal timing, and the possibility of interesterification of dietary lipids during food processing are some of the known and postulated modulators of postmeal lipid responses covered in this chapter. After that, we'll go into the important, immutable aspects of lipid reactions after meals, including hereditary variables. This chapter discusses several methodological ways to assessing plasma TG, which has limited the data base thus far. These techniques may be used to guide future research.

Jambhulkar (2023) Glucose intolerance, high blood pressure, abnormal lipid profiles, a tendency to clot, and an upsurge in macrovascular complications are all symptoms of type 2 diabetes mellitus (Type 2 DM), which is defined by insulin resistance or relative insulin deficiency. Purposes and Goals: In order to predict the risk of atherosclerosis in T2DM participants, the current research aimed to link fasting and postprandial apolipoprotein B with total cholesterol/high-density lipoprotein-cholesterol (TC/HDL-C). We used sixty healthy participants who were age-and sex-matched as controls and sixty clinically diagnosed patients of type 2 diabetes mellitus (aged 35–65 with a diabetes duration of more than 5 years). We assessed the lipid profiles of both the study groups before and after meals, including blood TC, triglycerides (TGs), HDL-C, LDL-C, and apolipoprotein B. Both the fasting and postprandial states may be used to compute the ratio, which is TC/HDL-C. The students' unpaired t-test was used for the statistical analysis. The current study's findings demonstrated that postprandial serum TC, TGs, LDL-C, and apo B levels were considerably higher than those in the fasting state ($P<0.05$). Compared to the fasting state, the postprandial state had a substantially reduced blood HDL-C level ($P<0.05$). Subjects with type 2 diabetes have a substantial rise in postprandial TC/HDL-C and apo B as compared to the fasting state. Conclusion: In the postprandial state, apolipoprotein B, TC/HDL-C ratio, and postprandial lipid profile are all considerably higher than preprandial levels, suggesting that they may be used as simple markers for cardiovascular risk factors.This means that in the postprandial state, individuals with type 2 diabetes may frequently have their TC/HDL-C ratio and Apolipoprotein B, a measure of the quantity of LDL particles in the blood (for atherosclerosis), assessed.

Mortensen (2012) An elevated risk of cardiovascular disease is linked to worsened postprandial lipid responses. Whey protein has a more pronounced impact on reducing

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lipids than other dietary proteins on postprandial lipemia. After a high-fat meal, we tested how various whey protein fractions affected the hormone responses and postprandial lipid profiles of people with type 2 diabetes. In a random sequence, twelve individuals with type 2 diabetes consumed four isocaloric test meals. Each of the test meals included 100 g of butter, 45 g of carbohydrates, and 45 g of either whey hydrolysate, α-lactalbumin enhanced whey, caseinoglycomacropeptide enhanced whey, or iso-meal. There were pre- and post-prandially assessed quantities of triglycerides, retinyl palmitate, free fatty acids, insulin, glucose, glucagon, glucagon-like peptide 1, and glucose-dependent insulinotropic peptide in the plasma.Our major variable, triglyceride, did not show any statistically significant variations among meals. The hydro-meal resulted in a greater retinyl palmitate response in the chylomicron-rich fraction compared to the iso- and lac-meal ($P=0.008$), however no such changes were seen in the chylomicron-poor fraction. In comparison to the lac- and CGMP-meal, the insulin response was greater in the hydro- and iso-meal ($P<0.001$). Over the course of the 480-minute period, there were no discernible variations in the hormone responses as measured by the incremental area under the curve. In type 2 diabetic patients, the effects on postprandial triglyceride responses were comparable when four different whey protein fractions were added to a fat-rich meal. The insulin response was elevated when whey hydrolysate and whey isolate were utilized.

RESEARCH METHODOLOGY

For the in vitro biochemical study, only blood samples that were taken at the Clinical Biochemistry laboratory were used. The samples were obtained using established protocols in an aseptic environment. Prior to analysis, samples were preserved and stored according to standard protocols.

A complete systemic evaluation would involve measuring the patient's weight, height, and waist circumference in addition to taking their blood pressure and body mass index (both systolic and diastolic). BMI is determined by dividing weight in kilograms by height in meters squared.

Fasting and postprandial lipid profiles, as well as blood glucose levels, were part of the screening process. In vitro biochemical experiments were performed using standard procedures provided below to assess serum concentrations of the specified indicators for each instance.

DATA NALYSIS

All of the data acquired on the chosen instances was used to create a master chart. by use of electronic means Sigma Stat 3.5 version (2012) and SPSS software were used for data analysis. A p-value less than 0.05 was considered significant after using this program to determine percentages, means, standard deviations, and 'p' values using the student 't' test, one way ANOVA, Pearson Correlation, and Chi square test.

individuals with type 2 diabetes mellitus had their fasting and postprandial lipid levels compared, and the relevance of postprandial dyslipidemia relative to fasting dyslipidemia as a cardiovascular risk factor in these individuals was evaluated.

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The XLSTAT 2014.1.09 software was used for data analysis, and a value of p<O.OOOl was deemed very significant, while p<O.Ol and p<0.05 were judged significant, respectively. To determine whether there were any variations in the means of the variables, one-way analysis of variance (ANOVA) was used for repeated measurements within a group or for entirely randomized measures across groups, respectively. The student's paired or unpaired t-test, as well as the test for parametric or non-parametric data, were used to compare two groups. The results are presented as the average plus or minus some standard deviation. Whether the LDL sub-fractions were big or tiny was compared in terms of frequency. By using the trapezoid method, the area under the curve (AUC) for consecutive measurements of TG concentrations at baseline and after the fat load was computed. In order to identify any important factors that may influence postprandial lipaemia, we used linear regression analysis and calculated correlation coefficients. (i.e. high AUC).

Table: 1 Distribution of study groups

Groups	No. of subjects
Healthy control	120
Known history of CVD	120
Male with CVD	79 (out of 120 of CVD)
Female with CVD	41 (out of 120 of CVD)
CVD with Mets (ATP III criteria)	66 (out of 120 of CVD)
CVD without Mets (ATP HI criteria)	54 (out of 120 of CVD)

Table: 2 CVD patients' history

Patients' history	Percentage (%
Family history	54
Smoking • Non-smokers • Ex-smokers • Occasionally smokers • <10 cigarettes/day • > I0cigarettes/day	 15 46 17 16 6

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Drink/alcoholic • Alcoholic • Occasionally alcoholic • Ex-alcoholic	 08 24 37
Diet type • Vegetarian • Non-vegetarian • Mix type	 18 24 58
Taking hypertensive lowering medication	92
Taking cholesterol lowering medication	89
Obese (based on BMI Kg/M") • BMI<25 • BMI<25.30 • BMI<30	 27 63 10

DISTRIBUTION OF STUDY GROUPS

The distribution of the study groups is shown in Table 1. Males made up 67 and females 53 of the 120 CVD patients. The ATP III panel criteria were used to classify all CVD patients; sixty-six of these patients had metabolic syndrome, whereas the remaining fifty-four did not. The current research included a total of 240 participants, 120 of whom were healthy and included in the control group.

Table 2 displays the patient's medical history derived from the patient questionnaires administered during blood sample collection.

Table: 3 Anthropometric measurements, fasting biochemical variables and clinical characteristics of the main study group

Variables	Control (n=120)	Range	CVD (n=120)	Range	t-value

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Age (Yrs.)	45.87+11.28	25-58	54.62+9.28	32-67	5.95
BMI (Kg/M2)	22.02+2.77	16.8-25.5	26.72+2.43	20.7-31.7	1.75
Waist (cm)	82.65+6.62	55-93	93.01+6.31	76-106	11.32
BPS mm/Hg	118.11+2.831	110-124	136.72+20.70	100-240	8.90
BPD mm/Hg	78.97 +2.53	72-85	86.11+7.89	70-100	8.52
Glu (mmol/L)	5.09+0.62	3.73-6.39	8.34+1.79	5.8-13.7	17.13
TC (mmol/L)	3.61+0.44	2.85-4.74	5.27+0.88	2.9-S.4	16.49
TG (mmol/L)	1.24+0.17	0.79-1.61	2.13+0.43	1.4-4.0	18.85
Free gly (pmoI/L)	90.79+6.74	72-119.5	168.30+42.40	112-372	18.05
HDL-C (mmol/L)	1.21+0.18	0.83-1.70	0.78+0.11	0.50-1.09	-19.50
LDL-C (mmol/L)	1.85+0.45	1.03-2.85	3.50+0.89	1.2-6.8	16.60
VLDL-C (mmol/L)	0.57+0.07	0.36-0.74	0.97+0.20	0.67-1.86	18.85
TC: HDL-C	3.05+0.51	2.09-4.42	6.87+1.73	3.20-13.2	21.13
LDL-C: HDL-C	1.57+0.48	0.70-2.84	4.59+1.53	1.36-10.66	18.74
sd-LDL-C (mmoi/L)	0.80+0.19	0.39-1.26	2.21+0.80	0.77-5.23	21.51
LB-LDL-C (mmol/L)	1.04+0.19	0.47-2.00	0.99+0.24	0.49-1.88	-3.01
N-HDL-C (mmol/L)	2.42+0.44	1.62-3.44	4.48+0.90	2.05-7.79	20.46

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Insulin (pU/mL)	8.55+0.80	7.01-11.00	12.53+1.51	9.34-15.34	23.12
HOMA IR	1.94+0.32	1.23-3.06	4.71+1.37	2.66-8.11	19.63

Table: 4 Fasting statuses of associated markers in main study group

Variables	Control (n=120)	Range	CVD (n=120)	Range	t-value
hs-CRP (mg/mL)	1.17+0.25	0.81-1.80	3.32+0.57	2.16-4.65	34.30
Adiponectin (Mg/mL)	12.78+0.45	11.82-13.87	7.13+1.80	4.21-10.61	-30.37
Leptin (ng/mL)	5.08+0.55	4.01-6.01	7.42+1.11	5.61-10.20	18.76
EL-6 (pg/mL)	4.00+0.65	2,68-9.34	5.59+0.65	4.01-7.21	17.10
LAR	0.39+0.04	0,30-0.47	1.13+0.40	0.58-2,16	17.89

CONCLUSION

The results showed that after a standardized fat load, individuals with cardiovascular disease had markedly increased postprandial triglyceride levels and a markedly delayed rate of clearance of these levels. These findings lend credence to the idea that TG levels measured after fasting are a better indicator of VLDL-C or residual lipoprotein concentrations than TG levels measured during fasting.

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