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To understand the reasons behind the higher occurrence of coronary heart disease in diabetes, a thorough investigation of how oxidized LDL from various population subgroups affects endothelium-dependent relaxation is necessary.

XIONGZIZENG[1], DR. SURIYAKALA[2a]

[1]PhD. Research Scholar in Medical Science, Lincoln University College, Malaysia
[2]Professor in Lincoln University College, Malaysia
Contact Details: [a]suriyakala@lincoln.edu.my

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Abstract

This study will not include Apolipoprotein B, and the possibility of a second coronary ischemia event will not be considered because it is a case control study. Additionally, the atorvastatin dosage will not be increased to the maximum effective dose. The term "low density cholesterol" refers to a metric, but the particle itself cannot be precisely measured.

INTRODUCTION

LDL cholesterol is associated with an increased risk of heart attack and stroke. Other atherogenic particles, besides low-density lipoprotein (LDL), are well-documented. Other apoprotein B-containing particles, such as VLDL and its remains, IDL, and Chylomicron remnants, are extremely important. Non-HDL cholesterol, a simple "metric," captures this. A fasting specimen is not required. Subtracting total cholesterol from HDL cholesterol. Even if people taking statins have lower LDL cholesterol, many of them still suffer a second coronary or cerebral vascular event. Patients with elevated triglycerides, low HDL cholesterol, and other remnant lipoproteins in their lipid profile are at risk. Non-HDL cholesterol, a term that encompasses all lipids other than HDL is important in determining the second level of risk for coronary or cerebral vascular events

in people taking statins. The study's goal will be to highlight the "usefulness" of other lipid profile factors in reducing ischemia episodes.

Death from atherosclerosis and its complications is currently the leading cause of death worldwide. Because of sedentary lifestyles and a fast food culture, early-onset obesity is becoming more prevalent. Public education has led to an increase in the number of people taking statins. A decrease in cardiovascular disease, however, has not occurred. The goal of this study will be to investigate lipids other than LDL.

LITERATURE REVIEW

The medical history was used to identify "CAD patients. Each participant had their height, weight, waist and hip circumferences measured, as well as their blood pressure and lipid profile. The age-standardized coronary artery disease anomalies appeared in the following order. Non-HDL cholesterol was followed by systolic blood pressure and abdominal obesity. Triglycerides, total cholesterol readings, low density lipoproteins, and HDL cholesterol were less relevant and had a decreasing correlation value in that order of importance. Non-high density lipoprotein cholesterol is used as a screening tool for people with the metabolic syndrome to determine their coronary vascular risk.

Sigedel et al. will study non-HDL cholesterol to see if it can be used as an indicator of CAD. risk. Total cholesterol measurements and low density lipoprotein cholesterol values have been used as correlation indicators for many years. Several studies have described non-HDL cholesterol as an important and simple marker because it can be calculated by subtracting "HDL cholesterol" from total cholesterol

STATEMENT OF THE PROBLEM

Lowering low-density lipoprotein cholesterol levels is widely regarded as a method of lowering the risk of coronary artery disease. There are, of course, exceptions to the rule. A large number of people do not have high levels of LDL cholesterol. Non-HDL cholesterol is becoming more widely recognized as a risk factor for cardiovascular disease. Non-HDL cholesterol, which includes the components of all atherogenic lipoprotein particles, can be obtained by subtracting HDL cholesterol from total cholesterol. People with diabetes, for example, who have dyslipidemia (a condition characterized by low HDL cholesterol levels and high triglyceride levels), are more susceptible to non HDL cholesterol. Measuring non-HDL cholesterol is expected to reveal the dangers associated with triglyceride-rich particles. Non-HDL cholesterol has been shown to correlate with the severity of coronary artery disease and can be used to predict cardiovascular disease mortality. Lifestyle changes and medication are both options for treating non-HDL cholesterol. Only rosuvastatin and simvastatin significantly lower non-HDL cholesterol among statins as a whole. Fibrates are a class of medications that lower triglycerides and non-HDL cholesterol. The FRIEDWALDS equation is used to predict LDL cholesterol, while total cholesterol and HDL are used to estimate non-HDL cholesterol. Derived from direct

measurements. Non-HDL cholesterol contains all of the lipid particles linked to cardiovascular disease. Because it has a higher predictive value, it is widely accepted.

THE STUDY AIMS

To examine how L-arginine and BH4 affect I/R-induced endothelial dysfunction in patients with type 2 diabetes and coronary artery disease.

Research Questions

• How do L-arginine and BH4 affect I/R-induced endothelial dysfunction in people with type 2 diabetes and coronary artery disease?

RESEARCH METHODOLOGY

This study will include atorvastatin-treated patients with coronary artery disease who had an ischemic stroke within five years of the first coronary event.
Cases in Group 1 included patients who had been taking atorvastatin 10 mg daily for more than a year, had a cerebrovascular event, such as a stroke, within five years of their first coronary event, and had an ECG or ECHO confirmation of coronary disease.
As a control group, we enrolled a group of patients with coronary artery disease who had been taking atorvastatin 10 mg for more than five years. These individuals must have a normal CT brain scan and no prior history of transient ischemic episodes. To be diagnosed.

RESEARCH DESIGN

The participants will range in age from 40 to 80, and both sexes will be represented. A committee within the institution approved the research. The study will include both modifiable and non-modifiable risk variables, such as cigarette smoking, alcohol consumption, hypertension, diabetes, and obesity (BMI). A thorough history-taking procedure will be used to determine the risk factors associated with smoking and drinking. Medical history, normal lab tests, and blood pressure measurements will be used to identify risk factors for diabetes and hypertension. After a 10-hour overnight fast, total cholesterol, HDL cholesterol, and triglycerides will be tested at 7 a.m. using a Hitachi 704 Analyzer. Low density lipoproteins Cholesterol will be calculated using the FRIEDWALD formula, which is widely accepted in the medical community. To calculate non-HDL cholesterol, divide the total cholesterol by the HDL content. The ECG and ECHO revealed the presence of coronary artery disease in both patients and "controls." Metabolic syndrome will be ruled out based on all of the patient's standard blood tests. Participants with elevated renal parameters or abnormal liver function tests will be excluded from the study.

DATA ANALYSIS

Descriptive statistics such as frequency, mean, median, and standard deviation will be calculated, and unpaired t tests will be used to detect statistically significant differences in means between

groups for any variables with a normal distribution. The chi-squared test will be used to compare the independent variables.

In contrast to Study, "data are presented as median and quartiles rather than mean and standard deviation (SEM)." Categorical information is typically presented numerically. Both sides will be considered significant if their p-values are less than 0.05. The effects of treatment on biochemical markers and FMD will be analyzed using Wilcoxon's signed rank test (within-group comparison). The Mann-Whitney rank sum test will be performed on clinical characteristics, laboratory data, and FMD as dependent variables. Compare groups. We will use the Wilcoxon signed-rank test to compare the means of the plethysmographic responses to different Ach doses in order to find statistically significant differences. In the Spearman "The correlation between the variables will be analyzed using rank." Different time points show distinct differences between the Ach and SNP dose response curves."In Study IV, a two-way ANOVA will be used to compare the effects of the two treatments on endpoints such as FBF, MAP, and P-glucose levels after reperfusion. Based on the results of Study I, we can estimate that we will need approximately 22 patients in each group to detect a 2% difference in FMD with 80% power and a two-tailed test at the 5% level. Because of the inherent speculative nature It is not possible to make reliable power estimates from studies. Here's how we intend to use historical data to make predictions. I counted 59, 145, and 157. Based on the data, there may be a significant difference between groups of 10 and 12, as evidenced by these findings.

CONCLUSION

In people with diabetes and coronary artery disease, lowering triglycerides is more important than statins' pleiotropic benefits of improving macrovascular endothelial function, microvascular function, and reducing inflammatory activity.

Diabetes patients with eosinophilic thrombocytopenia-1 (ET-1) deficiency have impaired cutaneous microcirculation. Diabetic microangiopathy may benefit from treatment that inhibits ET receptors.

L-arginine and BH4 inhibit I/R-induced endothelial dysfunction in people with type 2 diabetes and CAD when compared to placebo. Supplements such as L-arginine and BH4 may help these patients cope with the "threat of I/R malfunction."

LIMITATIONS OF THE STUDY

Because this is a "case control study," the atorvastatin dosage will not be increased to its maximum effective dose. The phrase "low density cholesterol" refers convert to metric. The low-density cholesterol particle will not be accurately measured. There will be no Apolipoprotein B in the study. This study will not take into account the possibility of a second ischemia event within the coronary system. Despite the fact that the research population's LDL cholesterol levels will be low, they will not meet the ATP 3 threshold. Despite the fact that controls have low non HDL

cholesterol levels, they do not meet the ATP 3 criteria. There is no need for a placebo group in this study because the goal is to compare the effects of the two treatment regimens on endothelial function. Moreover, the current The inclusion of a placebo group is not supported by statin therapy recommendations in people with established cardiovascular disease (CVD) and type 2 diabetes.

REFERENCES

1. Amarenc Rosengar A, et al. "Anterior inferior cerebellar artery territory infarcts. Mechanisms and clinical features." Archieve Neurology 1993;50:154 -161
2. Andy Jones, Graham Bentham "EPIC-Norfolk prospective population"; MRC epidemiology unit: 2012
3. Badimon L, Badimon JJ, Turitto VT, Vallabhajosula S, Fuster V. mechanism of platelet aggregation using type one collagen to strengthen. A description of how trauma to vessel heals. Its impact on how blood cells move about, v WF& blood clotting and movement Circulatory physiology. 1988;78(6):1431–1442. [PMID: 3263902]
4. Badimon L, Badimon JJ. "Procedures for thrombus formation large arteries in non parallel lines. Platelets aggregate and thrombus increases in size at the tip of the wall of arteries that are maximally affected." J Clin Invest 1989; 84(4):1134–1144. [PMID: 2794050]
5. Boekholdt SM, Arsenault BJ "Mora S, relationship between LDL cholesterol, non HDL cholesterol, and apolipoprotein B levels a meta analytical study indicating increase in coronary events.." JAMA. 2012 Mar 28;307(12):1302-9. doi: 10.1001/jama.2012.366.
6. Bogousslavsky J, Regli F. "infarcts due to anterior cerebral artery in the registry by Lausanne". Archieve Neurology 1990;47:144â€"150.
7. Chapman M.J "is there relevance in the effect on low density lipoprotein cholesterol by statins?" European Heart Journal Supplements Volume 6: Issue supplC: 710 2012.
8. Cholesterol in Adults "(Adult Treatment Panel III): Final Report.US Curr Atheroscler Rep. 2012" Apr; 14(2):130-4. doi: 10.1007/s11883-011-0224-x
9. Cholesterol in Adults "(Adult Treatment Panel III): Final Report.US Curr Atheroscler Rep. 2012" Apr;14(2):130-4. doi: 10.1007/s11883-011-0224-x
10. Elizabeth Barrett-Connor, MD "Sex Differences in Coronary Heart Disease" CIRCULATION;1997 95; 252-264.
11. "Expert Panel on Detection Evaluation, &Treatment of High Blood Fuster V. Cardiovascular disease and the United nations Millennium Development Goals: a significant need for analysis." Nat Clin Pract Cardiovasc Med 2006;3:401. [PMID: 16874332
12. Garg PR, Kaabita 1 S, "a study on cholesterol"Ann Human Biol: 2012 Nov 30.
13. Havarkate F, Thomson SG JR, Pepys MB. Synthesis of CRP &increased incidence of heart problems in angina with and without enzyme elevations: "European Concerted Action on Thrombosis and Disabilities Angina Pectoris Study" [NIH Publication No. 02- 5215. September 2002.] Circulation. 2002;106:3143–3420

14. Hoseini K, Saedeghian S, Mamoudian M, Hamiidian R, Abasi A. "Family history of cardiovascular disease as a risk factor for coronary artery disease in adult offspring." Monaaldi Archieve Chest Disease. 2008 Jun;70(2):84-7.
15. Jeremy A. Kelley, MSN, RN, CRNP1 "CORONARY ARTERY DISEASE AND SMOKING CESSATION INTERVENTION" Online Journal of Health Care, vol. 9, no.2, Fall 2009
16. John C. M. Brust , Merritt's Neurology, "Cerebral Infarction"11th Edition, Lippincott Williams & [NIH Publication No. 02-5215. September 2002.] Circulation. 2002;106:3143–3420
17. KruthHS, "Sequestration of aggregated low density lipoproteins because of macrophages":CurrOpinLipidol: 2002; 13:483
18. Langile BL, "part in generation and progression of thrombus formation the circulatory system and atherothrombosis of heart". In: Fuuster V, "Atherothrombosis and Coronary Artery Disease", 2d edition Philadelphia: Lippincott Williams & Wilkins, 2005:561–568 : Lancet 1997:349:462–466.
19. Liby P, "Act local, act global: Inflammation and the multiplicity of "vulnerable" coronary plaques." J Am. ColCardiology 2005; 45:1600
20. Meenakshi Sharma, "Premature Coronary Artery Disease in Indians and its Associated Risk Factors" Vascular Health Risk Management. 2005 September; 1(3): 217–225.
21. Michal G Marrmota, "alcohol and heart disease" Int. J. Epidemiology. (2001) 30 (4): 724-729.
22. Michel Miler, MD, "What Are the Effects of Statins on Triglycerides and What Are the Results of Major Outcomes Studies?" JAMA. 2012 Mar 28;307(12):1302-9.
23. Mulvihil ER, Jager J, :" smooth muscle cells in atheothrombosis plaques have a distinct phenotype". Arteriosclerosis Thrombosis Vascular Biology 2004; 24:1283
24. Napoli C, D'Armiento FP, Mancini FP, et al. "mechanism of formation of streak of fat in embryonic aorta & which causes severe increase because of increase in cholesterol levels in the mother increase in concentration of LDL and how its metabolism by oxidation cause influx of monocyte at the site of very early atherogenic plaques", J Clin Invest 1997;100:2680–2690. [PMID: 9389731]
25. Nilson J,"Atherogenesis regulated by immune mechanisms: prospects for the development of preventive medicines". Arteriosclerosis Thromb Vascular Biol 2005;25:18–28. [PMID: 15514204.
26. Pasterrkamp G, "Expansive arterial remodeling: Location, location, location". Arteriosclerosis Thromb Vascular Biol 2004; 24:650.Pp. C58-C63.
27. Rana JS, "The role of non-HDL cholesterol in risk stratification for coronary artery disease" Elsevier Ireland Ltd Nov 16:2012.
28. Sigdel M, "study on cholesterol" B.BMC Res Notes. 2012 Nov 17;5(1):640.

29. Stamler, J, "do we have a grading between cholesterol levels and risk of dying prematurely? "Findings in 356,222 primary screenees of the "Multiple Risk Factor Intervention Trial (MRFIT)": JAMA, 1986, 256:2823–2828. [PMID: 3773199]
30. Susan A, "Use of Statin" Lipid-Lowering Drugs Compared With Guidelines" JAMA January 2001:Vol 161, No.1234.
31. The Expert Panel. Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, & Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). The end report." Circulation, 2002, 106:3143–3421.
32. "Third report of the National Cholesterol Education Program (NCEP) :Tuzcu EM, "High prevalence of coronary atherosclerosis in asymptomatic teenagers and young adults: Evidence from intravascular ultrasound."Circulation 2001; 103:2705.
33. Vaccaro JA KELLEY, New England journal of medicine 2012;2012:916816. doi: 0.1155/2012/916816.
34. Robert C. Byrd ,Sniderman AD ,"Discordance analysis of Apolipoprotein B and non-high density lipoprotein cholesterol as markers of cardiovascular risk in the INTERHEART study Atherosclerosis." 2012 Dec;225(2):444-9.