Assessment of Serum Lipid Profile in Patients of Coronary Artery Disease: A Case-Control Study

Laltesh Kumar[1], Ajay Lal Das[2]

ABSTRACT

Introduction: Coronary Heart Disease (CHD) is widely prevalent across the globe and significantly high level of Cholesterol in circulation is a single major risk factor associated with Coronary Heart Disease. It is well established that cardiovascular disease is associated with hypertension and elevated blood levels of low-density lipoprotein (LDL), total cholesterol, and triglycerides. In disparity, a low level of high density lipoprotein (HDL) is a risk factor for mortality from cardiovascular disease. The present study was conducted with the aim to assess the changes in the lipid profile of coronary heart disease patients.

Material and Methods: The present study was done on 90 patients with acute or severe coronary heart diseases. Serum levels of total cholesterol, HDL cholesterol, LDL cholesterol, and triglycerides were examined by using biochemical kits. Fasting samples of blood were collected both from cases and healthy controls and sent to the laboratory for processing and performance of the lipid profiles in batches. Lipid profiling included Total Serum Cholesterol (TSC), Low Density Lipoprotein-Cholesterol (LDLC), High Density Lipoprotein-Cholesterol (HDLC), and Triglycerides (TG).

Results: The Total Serum Cholesterol to High density lipoprotein-Cholesterol and Low density lipoprotein-Cholesterol to High density lipoprotein-Cholesterol ratios also were significantly higher in cases than in controls, whereas the rise in Triglycerides to High Density Lipoprotein-Cholesterol ratio was not found to be significant.

Conclusion: It was found that the total cholesterol, HDL cholesterol, LDL cholesterol and triglycerides concentrations were significantly higher ($p < 0.05$) in coronary heart disease patients.

Keywords: Coronary Heart Diseases, Lipid Profile, Triglycerides, Cholesterol

INTRODUCTION

Coronary heart disease (CHD) or cardiovascular diseases are recognized to be one of the most important reasons of morbidity and mortality and imposes tremendously heavy socio-economic burden worldwide. There are varieties of risk factors in the literature which increases the incidence of CHD such as hyperlipidemia. By the year 2020, World Health Organization (WHO) is predicting more than 11.1 million deaths from CHD. It is projected that the annual number of deaths due to cardiovascular disease will increase from 17.5 million in 2012 to 25 million in 2030. Approximately out of 14 million Indians affected with coronary heart disease (CHD), 1.5 million develop Acute Myocardial Infarction (AMI), and 500,000 of these individuals die annually. This increase is due to industrialization, urbanization and related lifestyle changes which is called epidemiologic transition.[1,2]

Coronary heart disease occurs when cholesterol accumulates on the artery walls, creating plaques. Reduced blood flow occurs when one or more of these arteries become partially or completely blocked. The four primary coronary arteries are located on the surface of the heart are: right, left main coronary artery, left circumflex artery and left anterior descending artery.[3]

CHDs are the most predictable cause of sudden death. For many years, CHD prevalence was believed to be relatively low in developed countries. Recent studies have indicated a remarkably high proportion of mild to severe CHD in a number of patients. CHD is more prevalent in men than in women. However its prevalence interrelates with age. It is about 0.7% in 18 to 45 year olds. Whereas 13.3% in the 55 years and onwards. According to the Global Burden of Disease study estimate of age-standardized CVD death rate of 272 per 1,00,000 population in India is higher than the global average of 235 per 100,000 population.[4,5]

According to the guidelines of the American Heart Association, the following values are prescribed for the above-mentioned risk factors for cardiovascular disease: total cholesterol: <200 mg/dL; triglycerides: <200 mg/dL; HDL: >40 mg/dL; and LDL: <130 mg/dL.[6]

The term cardiovascular diseases are a group of disorders of the heart or blood vessels, and include mainly ischemic heart disease, rheumatic heart disease and cerebrovascular disease or strokes. The lipid profile is a group of tests that are often done together to identify the risk of heart disease. These tests are good indicators of whether someone is likely to have a heart attack or stroke caused by the blockage of blood vessels or hardening of the arteries. The lipid profile usually includes: high levels of cholesterol in blood circulation are strongly associated with progression of heart disease. For a person of about 68 kg typical total blood cholesterol synthesis is about 1g (1000mg) per day.[7,8]

[1]Assistant Professor, Department of Medicine, [2]Associate Professor, Department of Medicine, Varun Arjun Medical Coolege, Banthra, Shahjahanpur, UP, India

Corresponding author: Dr. Laltesh Kumar, Flate No -31, Varun Arjun Medical Coolege, Banthra, Shahjahanpur UP, India

How to cite this article: Laltesh Kumar, Ajay Lal Das. Assessment of serum lipid profile in patients of coronary artery disease: a case-control study. International Journal of Contemporary Medical Research 2018;5(5):E59-E62.

DOI: http://dx.doi.org/10.21276/ijcmr.2018.5.5.42

CHD requires an integrated approach to the reduction of its risk factors. Identification and management of risk factors are essential for preventing CHD in asymptomatic individuals mainly over 40 years of age as primary prevention, and for preventing recurrent events in patients with established disease as secondary prevention. Risk factors management should be conceived as prevention or treatment of the atherosclerotic disease process itself. CHD risk factors are modifiable and unmodifiable; the presence of unmodifiable risk factors may necessitate more intense management of modifiable risk factors.[9]

In the last twenty years, considerable advances have been achieved in the determination and improvement of CHD risk factors including diabetes and hypertension. Lipids and lipoproteins have become increasingly important in clinical practice, primarily because of their association with CHD, in case of their abnormalities known as dyslipidemia, and became the major risk factor for the development of this disease, according to epidemiological studies, especially in affluent countries where fat consumption is high.[10] Hence, this study was done to estimate the prevalence and to find out the association of lipid profile with CHD in a population of Bundelkhand region.

MATERIAL AND METHODS

The present study was conducted in Bundelkhand region from June 2017 to December 2017 at …….. The sample size was calculated by using G-power and was obtained to be 120 subjects. Out of which 90 CHD patients were selected with history of angina or surviving myocardial infarction with or without DM and HTN, admitted and diagnosed in coronary care unit. CHD cases with liver impairment, renal disease or thyroid disease were not included in the study. 90 clinically healthy subjects aged 41 to 70 years who served as population-based controls were chosen. This study was initiated after obtaining consent from the patients and healthy volunteers and after a prior approval by the .....university ethical committee.

Method of estimation

Blood sample was collected from the cubital vein of the arm of each patient by a 5cc disposable syringe, which was transferred quickly to a heparinized collecting tube and finally preserved into an ice pot. All these tubes were then transferred to a test tube, were allowed to stand overnight for the serum to separate. Then each serum sample was transferred to a separate eppendorf tube and stored at -20º C in a refrigerator; lipid profile was performed within one week for each group of samples, after running the controls for confirmation of the accuracy of each test, according to the procedures provided with Biocon kits.

Method of Biochemical estimation

Cholesterol was estimated by enzymatic colorimeter test. Estimation of HDL-C was done through phosphotungstic precipitation and LDL-C was also done through the same precipitation method. Estimation of Triglycerides (TG) was done by enzymatic colorimetric test. Serum of VHD patients were used for individual determinations of lipid profile for Cholesterol, HDL-C, LDL-C, and TG by using clinical laboratory kits.

STATISTICAL ANALYSIS

The absorbance of samples was read on spectrophotometer and the collected data was transferred to excel sheet and was then analyzed statistically by t- test.

RESULTS

In the present study, lipid profile of patients with Coronary Heart Disease (CHD) showed significant variation when compared to that of the control group. The Total Cholesterol ($p<0.05$) and Low Density Lipoprotein ($p<0.05$) were significantly higher in the cases, whereas the High Density Lipoprotein-Cholesterol had significantly lowered ($p<0.05$) from that of the controls. Serum Triglycerides were increased among cases than that of the controls but was not found to be significant statistically.

As compared with the lowest value of normal individual, the serum cholesterol value of VHD patients is three-

Age group	Normal Serum Cholesterol	Serum Cholesterol among CHD Patients	P value
41-50 years	169.21±24.07	196.24±12.56	0.022
51-60 years	119.25±16.08	204.81±19.15	0.049
61-70 years	194.45±21.58	267.78±24.32	0.005

Table-1: Showing age-wise distribution of serum cholesterol (SC) concentration among normal and CHD subjects

Age groups	Normal Triglyceride	Triglyceride Concentration among CHD Patients	P value
41-50 years	102.6±22.34	166.53±42.41	0.064
51-60 years	127.31±34.61	210.48±56.32	0.0034
61-70 years	164.56±21.49	124.61±36.09	0.889

Table-2: showing age- wise distribution of triglyceride (TG) concentration among normal and CHD subjects

Age groups	Normal HDL Concentration	HDL Concentration among CHD Patients	P value
41-50 years	49.51±6.89	41.52±4.39	0.016
51-60 years	77.2±8.03	43.09±6.63	0.001
61-70 years	87.4±11.61	43.26±6.84	0.456

Table-3: Showing age wise distribution of HDL concentration among normal and CHD patients

Age groups	Normal LDL Concentration	LDL Concentration among CHD Patients	P value
41-50 years	74.7±11.04	100.49±21.39	0.0246
51-60 years	89.01±12.34	106.33±22.46	0.004
61-70 years	98.23±16.94	109.07±24.06	0.005

Table-4: Showing age-wise distribution of LDL concentration among normal and CHD patients

times higher and the highest VHD value of 210.18 mg/dl is again 34.16 mg/dl higher than the serum cholesterol of normal subjects. The statistical analysis showed a significant difference (P<0.05). The acceptable maximum amount of cholesterol for healthy persons is 200 mg/dl; while 180 mg/dl is desirable for healthy persons. Therefore normal individuals with 170 mg/dl to 180 mg/dl can be regarded under the risk.

Lipid profile of patients in CHD was significantly different from that of the normal; though within the desirable levels. TC (p<0.05) and LDL-C (p<0.05) were significantly higher while the HDL-C was significantly lower (p<0.05) in cases in comparison with that of controls (Fig. 2). TAG was raised among patients with CHD than that of the controls but not significant statistically. The TC to HDL-C and LDL-C to HDL-C ratios were significantly higher in CHD patients than controls, whereas the rise in TAG to HDL-C ratio was not significant (Fig. 2). The results of total cases and controls are summarized in Table.1 and the age-variable lipid profiles described in Table no.3 and 4.

DISCUSSION

In developed countries like the United States, although there has been a very significant decrease in the incidence of vascular diseases like CHD1 cerebrovascular disease, and peripheral vascular diseases, yet CHD remains to be the major cause of death. The major risk factors are elevated LDL-C, reduced HDL-C, smoking, hypertension, insulin resistance with or without overt diabetes mellitus, age, and family history of premature CHD. Modifiable risk factors account for 85% of the elevated CHD risk, of which the most important is plasma cholesterol. TC levels of <160 mg/dl is able to decrease CHD risk, even if other risk factors are present.[11]

The key role of cholesterol in CHD has given rise to the universally accepted cholesterol-diet-CHD hypothesis. According to this hypothesis, increased plasma cholesterol concentrations increase the risk of CHD and decreasing plasma cholesterol levels decreases the risk of CHD. The Multiple Risk Factor Intervention Trial (MRFIT) showed that there is an increased risk at levels >200 mg/dL. The Seven Countries Study also demonstrated that elevated plasma cholesterol levels increased the incidence of CHD. The Framingham study clearly demonstrated the association of elevated cholesterol with CHD.[12,13]

Epidemiologic studies have linked the intake of high levels of dietary fat rich in cholesterol and saturated fats, with increased plasma cholesterol levels. Therefore, restriction of saturated fat and cholesterol is the cornerstone of dietary therapy to lower down the elevated blood cholesterol levels.[14]

In the present study, CHD incidence was more especially those in the age group 51- 60 years. Despite the wide literature on the relationship between lipid and lipoprotein particles to CHD incidence, there has been controversial evidence on the specific association of TAG with CHD. The Framingham study demonstrated that TAGs are independently related in women at all ages but missing statistical significance in the multivariate studies in men. According to two meta-analyses, TAGs were independent risk factors for CHD, even after adjustment with HDL-C, which is strongly and inversely correlated with TG.[15]

In the present study, although there was an increase in the triglycerides levels in the cases compared to the controls; yet the increase was not statistically significant. Clinical studies based on extensive literature supports the inverse relationship between HDL-C levels and atherosclerosis. HDL enhances the reverse cholesterol transport and has antioxidative, anti-inflammatory, antithrombotic, and vasoprotective effects.[16]

Studies have also consistently demonstrated that HDL-C is inversely associated with the risk of CHD. Thus, an increase in HDL-C is linearly associated with a reduction in cardiovascular risk. In line with these findings, present study demonstrated a significant decrease in the HDL-C levels in patients with CHD when compared to controls.[17,18]

Another recent prospective cohort study of apparently healthy male physicians demonstrated that an increase in HDL-C of ≥12.5 mg/dl over 14 years was associated with a 57% lesser risk of developing CHD. However, according to major clinical guidelines, HDL-C is a secondary target in CHD prevention. Current guidelines from the Adult Treatment Panel III emphasize on targeting primarily LDL-C, secondarily non–HDL-C, and then HDL-C. According to the American Diabetes Association, HDL-C should be considered a "secondary target" along with TAG, with a goal of HDL-C levels >40 mg/dL. The recent American Heart Association / National Heart, Lung, and Blood Institute scientific statement proposes that HDL-C should be a "tertiary target", following LDL-C and TAG, with goals of HDL-C levels >40 mg/dl in men and >50 mg/dl in women.[19,20]

The importance of LDL-C in the pathogenesis of CHD is well documented, and so is the benefit of lowering LDL in high-risk patients. This study demonstrated a significant increase in LDL-C in the CHD group. The National Cholesterol Education Program (NCEP) recommends an LDL-C goal of <100 mg/dl in patients with established CHD and in those who are CHD risk-equivalent. Aggressive LDL-C reduction is associated with less atherosclerosis progression, lower rates of revascularization, and fewer ischemic events compared with moderate LDL-C reduction or conventional treatment.[21]

In this study, there was an overall significant increase in the TC/HDL-C; it was extremely significant (p<0.0001) in men of 51-60 age group and in women between 51-70 years of age. In another study of patients with heterozygous familial hypercholesterolemia subjects, plasma HDL cholesterol values and TC/HDL ratios were found to be two important coronary risk factors. The LDL-C/HDL-C ratio is a valuable and a standard tool to evaluate CVD risk in all populations.[22]

In a study evaluating the prognostic significance of several risk factors on the outcome of CHD in 639 cardiovascular disease-free subjects with heterozygous familial hypercholesterolemia (FH), it was found that a one-unit difference in LDL-C/HDL-C ratio was associated with a

17% higher risk.[23] This study shows that LDL-C levels eight-times more than HDL-C predicts an adverse CHD event, in patients with FH.

CONCLUSION

The findings from this study revalidate this scientific observation that there was a significant rise in the levels of TC and LDL-C even among patients who had recovered from CHD when compared to the controls. Triglyceride level was also increased among the CHD group from that of the control group, but was not statistically significant. It is therefore important to focus on reduction of cholesterol levels. Lifestyle measures like consumption of a proper diet along with regular exercise; and if required, cholesterol-lowering therapy (e.g. statins) should be initiated in susceptible populations even though the levels may be well within the internationally desired levels.

REFERENCES

1. Amit Daphale, Sourya Acharya, Samarth Shukla. Detection of asymptomatic coronary artery disease (CAD) in newly detected type 2 diabetes mellitus (DM) by exercise treadmill test. International Journal of Contemporary Medical Research 2017;4:2269-2275.
2. Husan Pal, Savita Kapila, Ashish Bhagat, Harharpreet Kaur, Kiranjit. Risk Factors molding the direction of ischemic heart disease and the most prevalent ishcemic heart disease amongst males and females of Punjab. International Journal of Contemporary Medical Research 2017;4:127-129.
3. Saumya Gupta, Krishna K. Lakhani, Hirava Munshi. A study of risk factors in young patients of acute coronarysyndrome. International Journal of Contemporary Medical Research 2017;4:2144-2147.
4. National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). Executive Summary of The Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). JAMA. 2001; 285:2486-2497.
5. John VC, William AB, Dorman T, Sharon GO. Valvular heart disease. In: The Johns Hopkins manual of cardiac surgical care. 2nd ed. Marry land, Baltimore, Elsevier publisher, 2007, pp 96.
6. Bonow RO, Carabello BA, Kanu C. Guidelines for the management of patients with valvular heart disease: a report of the American Heart Association Task Force on Practice Guidelines. Circulation. 2006; 114: 84 – 231.
7. Vuyisile T N, Julius MG, Thomas NS, John SG, Christopher GS, Maurice ES. Burden of valvular heart diseases: a population based study. The lancet. 2006; 368: 969 - 971.
8. Rahilly-Tierney C, Bowman TS, Djoussé L, Sesso HD, Gaziano JM. Change in High-Density Lipoprotein Cholesterol and Incident Coronary Heart Disease in Apparently Healthy Male Physicians. Am J Cardiol. 2008 December 15; 102: 1663–1667.
9. Singh IM, Shishehbor MH, Ansell BJ. High-Density Lipoprotein as a Therapeutic Target A Systematic Review. JAMA. 2007; 298:786-798.
10. Thomas A. Gaziano, MD, MSc, Asaf Bitton, MD, Shuchi Anand, MD, Shafika Abrahams-Gessel, MS, and Adrianna Murphy. Growing Epidemic of Coronary Heart Disease in Low- and Middle-Income Countries. Curr Probl Cardiol. 2010; 35: 72–115.
11. Friedewald WT, Lewy RJ, Fredrickson DS. Estimation of concentration of low density lipoprotein cholesterol in plasma, without use of the preparative ultracentrifuge. Clin. Chem. 1972; 18: 499 – 502. Canada, Alive books publishers, Green press, 1993, pp 56.
12. Werner M, Garberielson DG, Estman J. Ultramicro determination of serum triacylglycerols by bioluminescent assay. Clin Chem. 1981; 27:268-271.
13. Rhonda BeLue, Titilayo A Okoror, Juliet Iwelunmor, Kelly D Taylor, Arnold N Degboe, Charles Agyemang. An overview of cardiovascular risk factor burden in sub-Saharan African countries: a socio-cultural perspective. Globalization and Health 2009.
14. Yusuf S, Hawken S, Ounpuu S, Dans T, Avezum A, Lanas F, McQueen M, Budaj A, Pais P, Varigos J, Lisheng L et al.,Effect of potentially modifiable risk factors associated with myocardial infarction in 52 countries (the INTERHEART study): case-control study. Lancet. 2004; 364:937-52.
15. Wilson PW, Cupples LA, Kannel WB. Is hyperglycemia associated with CVD? The Framingham Study. American Heart Journal. 1991; 121:586-590.
16. Castelli WP, Anderson K, Wilson PW, Levy D. Lipids and risk of coronary heart disease. The Framingham Study. Ann Epidemiol. 1992; 2:23-8.
17. Gotto AM. Cholesterol intake and serum cholesterol level. N Engl J Med 1991; 324:912-913.
18. Grundy SM, Denke MA.Dietary influences on serum lipids and lipoproteins. J Lipid Res 1990; 31:1149-1172.
19. Hokanson JE, Austin MA. Plasma triglyceride level is a risk factor for cardiovascular disease independent of high-density lipoprotein cholesterol level: a meta-analysis of population-based prospective studies. J Cardiovasc Risk. 1996; 3:213-9.
20. Sarwar N, Danesh J, Eiriksdottir G, Sigurdsson G, Wareham N, Bingham S, et al. TG and the risk of coronary heart disease: 10,158 incident cases among 262,525 participants in 29 Western prospective studies. Circulation. 2007; 115:450-8.
21. Panagiotakos DB, Pitsavos C, Skoumas J, Chrysohoou C, Toutouza M, Stefanadis CI, Toutouzas PK. Importance of LDL/HDL cholesterol ratio as a predictor for coronary heart disease events in patients with heterozygous familial hypercholesterolaemia a 15-year follow-up (1987-2002). Curr Med Res Opin. 2003; 19:89-94. pp. 802–809.
22. The Multiple Risk Factor Intervention Trial Research Group: Mortality rates after 10.5 years for participants in the Multiple Risk Factor Intervention Trial. Findings related to a priori hypotheses of the trial. JAMA 1990; 263:1795-1801.
23. Greenland P, Knoll MD, Stamler J, et al. Major risk factors as antecedents of fatal and nonfatal coronary heart disease events. JAMA 2003; 290:891-897.

Source of Support: Nil; **Conflict of Interest:** None

Submitted: 10-05-2018; **Accepted:** 05-06-2018; **Published:** 23-06-2018