

Comparative analysis of lipid levels between coronary heart disease patients and normal healthy subjects

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Abstract: Coronary Heart Disease (CHD) is the most common type of Cardiovascular Diseases (CVD). It is reported that globally more people die of CVDs than any other disease. CHD is influenced by multifactorial factors. A number of risk factors such as dyslipoproteinemia, obesity, oxidative stress, smoking, hypertension, alcohol intake, have been associated with the disease. The current study evaluates the risk factors between controls and CHD patients of Karachi. Two hundred and twenty (220) subjects were recruited in the study. 110 subjects were normal healthy controls and 110 were CHD patients. Age, BMI (kg/m²) and blood pressure (mmHg), lipid profile and relevant risk factors were evaluated. Significant differences were observed between patients and the control subjects. In BMI, blood pressure, total cholesterol (TC), triglycerides (TG) and low density lipoprotein cholesterol (LDL-C) were significantly higher in patients group ($p < 0.001$), while high density lipoprotein cholesterol (HDL-C) was higher in the control subjects ($p < 0.05$). It can be concluded that BMI, smoking and blood pressure and disturbed lipid profile are crucial determinant of atherosclerosis and may lead to the development coronary events.

Keywords: Coronary heart disease, lipid profile, risk factor, BMI, smoking.

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INTRODUCTION

Coronary Heart Disease (CHD) is the most contributing disease for deaths worldwide. It has affected west as well as the developing countries¹. World Health Organization (WHO) estimated approximately 7.3 million deaths due to CHD in 2008². There is an increase in its incidences owing to many socio-economic reasons. CHD is caused by several modifiable and non-modifiable risk factors. Modifiable factors include obesity, hypertension, smoking, physical inactivity, alcohol consumption, diabetes, stress, elevated LDL-C and decreased HDL-C³⁻⁸. The non-modifiable factors include family history of heart disease, gender and male age > 45 years and female age > 55 years⁹. Dyslipidemia contributes significantly to atherosclerosis and its manifestation includes CHD^{10,11}. The risk of development of CHD is potentiated by elevated blood pressure, obesity, smoking and aging. Hypertension may lead to mortality in CHD as it causes strain on the heart^{12,13}. Smoking is also a well-established risk factor for coronary incidence¹⁴. It is one of the preventable causes of CHD, and the prevention may reduce coronary risk substantially¹⁵. Chemicals that are present in the smoke damage the linings of the vessels which may ultimately lead to elevated blood pressure and abnormal lipid levels. High levels of total cholesterol (TC), triglycerides (TG) and low density lipoprotein cholesterol (LDL-C) and low levels of high density lipoprotein cholesterol (HDL-C) are also the hallmarks of CHD. Strong association has been suggested between CHD risk and TC, LDL-

C, TG and HDL-C levels¹⁶. Significant differences have also reported for TC, TG, LDL-C and HDL-C levels between controls and CHD patients group¹⁷. High TC, LDL-C and TG levels promote atherosclerosis and thus increasing the risk for CHD particularly in combination with age, gender and family history. The current study evaluates the risk factors between controls and CHD patients of Karachi.

MATERIALS AND METHODS

A total of 110 patients of CHD and 110 normal control subjects were included in the study. The study was approved by the institutional ethical committees of the concerned institutes. Blood samples of CHD patients were collected from local hospitals of Karachi. Informed consent was taken from each subject. Information was collected about current and past smoking habits, medical or family disease, dietary habits and general demographic data. Their BMI was calculated as kg/m². High blood pressure was defined as systolic blood pressure (SBP) greater than 120 mmHg and diastolic blood pressure (DBP) greater than 80 mmHg. Individuals having current or previous smoking habits were designated as smokers. Subjects with normal lipid profile and glucose level and without any cardiovascular event were selected as controls.

Blood samples in fasting condition were collected from normal healthy subjects and CHD patients. After separation of serum from the blood, the samples were subjected to lipid profile analysis by enzyme colorimetric method using kits (Merck

Pvt. Ltd. Pakistan); TC (Cat# 5.17500.0001) TG (Cat# 5.17510.0001) and HDL-C (Cat # 1 3540 99 66 885) were estimated. LDL-C was estimated by Friedewald formula²³. Statistical analysis was done by systat 12 software. Student's t test was employed for comparison of BMI, blood pressure and lipid profile in control and patients group. Data are presented as mean±SEM.

RESULTS AND DISCUSSION

In the current study, the mean age of controls and patients are not same as both the modifiable and non-modifiable risk factors are quite prevalent in the society. Normal healthy subjects of age matched with patients without any risk factor were hard to find. Therefore, a little flexibility was employed to recruit the controls.

Approximately 66% of the controls were males and 34% were females. In CHD patients 65% were males and 35% were females. Males are known to be more prone to develop CHD as compared to females of any age¹⁸.

For normal healthy individual, BMI is expected to be between 18.5-25.0 kg/m². BMI less than 18.5 has been considered as underweight, above 25.0 as overweight and greater than 30.0 kg/m² as obese¹⁹. The BMI for patients was 27.42±0.37 and for controls it was 22.90±0.27 kg/m². BMI of the patients group was significantly higher than the control group (p<0.01). Obesity plays a crucial role in the development of atherosclerosis and ultimately to the development of CHD. As BMI increases the prevalence and severity of CHD also increases and it has been reported that BMI and blood pressure are significantly higher in CHD patients group as compared to controls^{17,21}.

The normal SBP ranges between 100-140 mmHg and DBP ranges between 60-90 mmHg, if blood pressure consistently remains ≥140/90mmHg then it is assumed to be hypertensive²⁰. It has been reported that increased blood pressure has a causal relationship with CHD. Polymorphisms of the genes that are involved in the regulation of blood pressure primarily contribute to elevated blood pressure²². Significant difference was observed between means of SBP (P<0.001) and DBP (p<0.05) of controls and patients group.

The total cholesterol levels for control group was 120.91±3.41 mg/dl and for patients 216.76±6.73 mg/dl. TC levels were observed significantly higher in the patients (p<0.001). The mean of triglycerides levels in control group was 113.34±6.96 mg/dl and for patients 166.49±7.78 mg/dl and that of LDL-C were 88.63±3.42 mg/dl and patients 166.71±6.15

mg/dl, respectively. Significantly high levels of triglycerides (p<0.001) and LDL-C (p<0.001) were found in patients. The mean HDL-C levels for control group was 40.76±1.18 mg/dl and for patients 33.66±0.99 mg/dl. HDL-C levels were significantly higher in control group as compared to patients group (p<0.05). Significant differences have been reported for TC, TG, LDL-C and HDL-C levels between controls and CHD patients group in other populations¹⁷. Elevated TC, TG and LDL-C levels and decreased HDL-C level in patients group signify the importance of lipid profile in the prognosis of the disease.

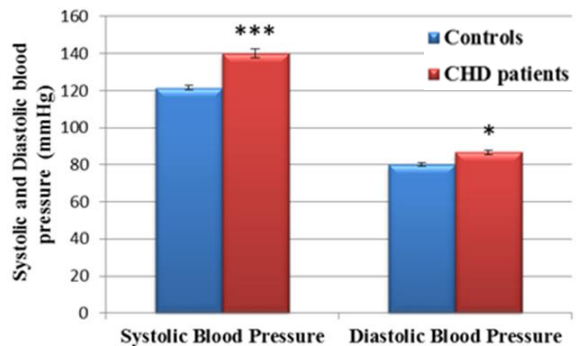


Figure 1: Systolic and diastolic blood pressure in control and patients groups.

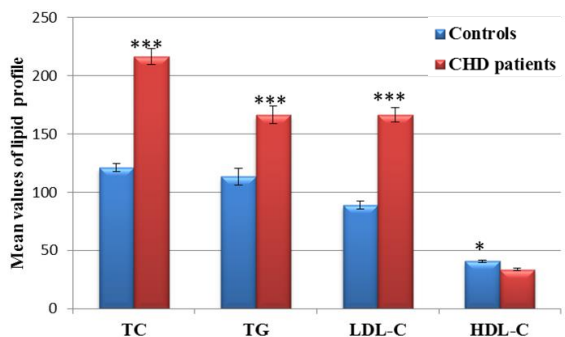


Figure 2: Lipid profiles of controls and patients.

In conclusion, lipid profile parameters are important constituents in the outcome of the disease. Disturbed lipid profile may predispose an individual to atherosclerosis, that eventually leads to CHD if diabetic and therapeutic interventions are not applied

REFERENCES

1. Roger VL, Go AS, Lloyd-Jones DM, Benjamin EJ, Berry JD, Borden WB, Bravata DM, Dai S, Ford ES, Fox CS, Fullerton HJ, Gillespie C, Hailpern SM, Heit JA, Howard VJ, Kissela BM, Kittner SJ, Lackland DT, Lichtman JH, Lisabeth LD, Makuc DM, Marcus GM, Marelli A, Matchar DB, Moy CS, Mozaffarian

- D, Mussolino ME, Nichol G, Paynter NP, Soliman EZ, Sorlie PD, Sotoodehnia N, Turan TN, Virani SS, Wong ND, Woo D, Turner MB, American Heart Association Statistics Committee and Stroke Statistics Subcommittee. Heart disease and stroke statistics—2012 update: a report from the American Heart Association. *Circulation.*, 2012; 125: e2–e220.
2. Global atlas on cardiovascular disease prevention and control. Geneva, World Health Organization, 2011.
3. Freedman DS, Srinivasan SR., Shear CL, Hunter SM, Croft JB, Webber LS and Berenson GS. Cigarette smoking initiation and longitudinal changes in serum lipids and lipoproteins in early adulthood: the Bogalusa Heart Study. *Am. J. Epidemiol.*, 1986; 124: 207–219.
4. Craig WY, Palomaki GE, and Haddow JE. Cigarette smoking and serum lipid and lipoprotein concentrations: an analysis of published data. *BMJ.*, 1989; 298: 784–788.
5. Bønaa KH, and Thelle DS. Association between blood pressure and serum lipids in a population: the Tromsø Study. *Circulation.*, 1991; 83: 1305–1314.
6. Neuhauser ML, Miller DL, Kristal AR, Barnett MJ, and Cheskin LJ. Diet and exercise habits of patients with diabetes, dyslipidemia, cardiovascular disease or hypertension. *J. Am. Coll. Nutr.*, 2002; 21: 394–401.
7. Mohanna S, Baracco R, and Seclén S. Lipid profile, waist circumference, and body mass index in a high altitude population. *High Alt. Med. Biol.*, 2006; 7: 245–255.
8. de Campos W, Stabelini Neto A, Bozza R, Ulbrich AZ, Bertin RL, Mascarenhas LP, da Silva SG, and Sasaki J. Physical activity, lipid consumption and risk factors for atherosclerosis in adolescents. *Arq. Bras. Cardiol.*, 2010; 94: 601–607.
9. Bachorik PS, Levy RI and Refkind BM. Lipids and dyslipoproteinemia, In: *Clinical Diagnosis and Management by Laboratory Methods*, Eighteenth edition, Editor: Henry JB, WB Saunders, Philadelphia PA. 1991; 188-214.
10. Sarwar N, Danesh J, Eiriksdottir G, Sigurdsson G, Wareham N, Bingham S, Boekholdt SM, Khaw KT and Gudnason V. Triglycerides and the risk of coronary heart disease: 10,158 incident cases among 262,525 participants in 29 Western prospective studies. *Circulation.*, 2007; 115: 450–458.
11. National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). 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) Final Report. *Circulation.*, 2002; 106: 3143–3421.
12. Stamler J, Stamler R and Neaton JD. Blood pressure, systolic and diastolic, and cardiovascular risks: US population data. *Arch. Intern. Med.*, 1993; 153: 598-615.
13. Stamler J, Dyer AR, Shekelle RB, Neaton JD and Stamler R. Relationship of baseline major risk factors to coronary and all-cause mortality, and to longevity: findings from long-term follow-up of Chicago cohorts. *Cardiology*, 1993; 82: 191-222.
14. Inoue T. Cigarette Smoking as a Risk Factor of Coronary Artery Disease and its Effects on Platelet Function. *Tob. Induc. Dis.*, 2004; 2: 2.
15. van Berkel TF, Boersma H, Roos-Hesselink JW, Erdman RA and Simoons ML. Impact of smoking cessation and smoking interventions in patients with coronary heart disease. *Eur. Heart J.*, 1999; 20: 1773-1782.
16. Sharrett AR, Ballantyne CM, Coady SA, Heiss G, Sorlie PD, Catellier D and Patsch W. Coronary Heart Disease Prediction From Lipoprotein Cholesterol Levels, Triglycerides, Lipoprotein(a), Apolipoproteins A-I and B, and HDL Density Subfractions. The Atherosclerosis Risk in Communities (ARIC) Study. *Circulation.*, 2001; 104: 1108-1113.
17. Frikke-Schmidt R, Nordestgaard BG, Schnohr P, Tybjaerg-Hansen A. and Schmidt R. Single nucleotide polymorphism in the low-density lipoprotein receptor is associated with a threefold risk of stroke. A case-control and prospective study. *Eur. Heart J.*, 2004; 25: 943–951.
18. Khaw KT. Epidemiology of coronary heart disease in women. *Heart*. 2006; 92: 2-4.
19. WHO expert consultation. Appropriate body-mass index for Asian populations and its implications for policy and intervention strategies. *The Lancet*, 2004; 363: 157-163.
20. Carretero OA and Oparil S. Essential hypertension. Part I: definition and etiology. *Circulation.*, 2000; 101: 329–35
21. Labounty TM, Gomez MJ, Achenbach S, Al-Mallah M, Berman DS, Budoff MJ, Cademartiri F, Callister TQ, Chang HJ, Cheng V, Chinnaiyan KM, Chow B, Cury R, Delago A, Dunning A, Feuchtner G, Hadamitzky M, Hausleiter J, Kaufmann P, Kim YJ, Leipsic J, Lin FY, Maffei E, Raff G, Shaw LJ, Villines TC and Min JK. Body mass index and the prevalence, severity, and risk of coronary artery disease: an international multicentre study of 13,874 patients. *Eur. Heart J. Cardiovasc. Imaging.*, 2013; 14: 456-463.
22. Lieb W, Jansen H, Loley C, Pencina MJ, Nelson CP, Newton-Cheh C, Kathiresan S, Reilly MP, Assimes TL, Boerwinkle E, Hall AS, Hengstenberg C, Laaksonen R, McPherson R, Thorsteinsdottir U, Ziegler A, Peters A, Thompson JR, König IR, Erdmann J, Samani NJ, Vasan RS and Schunkert H; CARDIoGRAM. *Hypertension.*, 2013; 61: 995-1001
23. Friedewald WT, Levy RI and Fredrickson DS. Estimation of the concentration of low-density lipoprotein cholesterol in plasma, without use of the preparative ultracentrifuge. *Clin. Chem.*, 1972; 18: 499–502.