IJPPR
INTERNATIONAL JOURNAL OF PHARMACY & PHARMACEUTICAL RESEARCH
An official Publication of Human Journals
ISSN 2349-7203
HUMAN

Human Journals
Research Article
December 2017 Vol.:11, Issue:1

Lipid Profile and Coronary Heart Disease

IJPPR
INTERNATIONAL JOURNAL OF PHARMACY & PHARMACEUTICAL RESEARCH
An official Publication of Human Journals
ISSN 2349-7203
HUMAN

Maha Radhi Abess[1], Majid Kadhum Hussain[2], Zuhair Mohammed Ali Jeddoa[1]

[1]University of Kerbala, Iraq

2 University of Kufa, Iraq

Submission: 25 November 2017
Accepted: 3 December 2017
Published: 30 December 2017

Keywords: Lipid profile; coronary heart disease

ABSTRACT

Background:- Coronary artery disease (CAD) remains the major cause of mortality and morbidity in the entire world population despite therapeutic advances that control many risk factors, Lipid profile is regarded as an important factor in the development of Coronary heart disease, There have been numerous studies confirming the association of hyperlipidemias with Coronary Heart Disease in most of the Western and Asian countries of the world. Many studies demonstrated the relation between coronary heart disease (CHD) prevalence and lipid levels. The aim of this study was to determine the prevalence of abnormal lipid levels and its association with coronary heart disease (CHD). **Methods and Results:-** A case-control study was conducted to find the relationship between lipid profile and coronary heart disease. This study involved 150 CHD patients and 150 controls group. The serum total cholesterol, HDL cholesterol, LDL, VLDL, Triglyceride, and body mass index were determined. The mean age of subjects was (28.71 ± 4.12) years in the CHD group and (30.14 ± 3.29) years in the control group. The lipid profile parameters were comparable between patients and control subjects. There were statistically significant in the BMI, total cholesterol, Triglyceride, low-density lipoprotein cholesterol and high-density lipoprotein cholesterol in CHD group when compared with the control group. **Conclusion:-** The changed lipid profile levels may contribute to increased risk of Coronary Heart Disease(CHD) patients.

INTRODUCTION

Coronary heart disease (CHD) is a leading cause of morbidity and mortality in many countries worldwide (WHO, 2015). CHD refers to a group of a closely related syndrome caused by the imbalance between the myocardial oxygen demand and the blood supply. Depending on the rated severity of coronary artery narrowing and the myocardial response, which is divided into angina pectoris (chest pain), acute myocardial infarction, sudden cardiac death and chronic ischemic heart disease (Kumar et al.,2015).The most common risk factors of CHD are hypertension(Jindrich.,2012), smoking, obesity (Paratz et al. 2015), diabetes (Go.,2014), stress, gender, age(Ashif.,2011) and dyslipidemia (Sarwar et al., 2007: de Campos et al., 2010) These are high levels of total cholesterol, Triacylglycerols (TAG), low-density lipoprotein cholesterol (LDL-C) and very low-density lipoprotein(VLDL) and with low levels of High density lipoprotein cholesterol (HDL-C)(Ahmed et al.,1998; Kullisaar *et al*., 2016).that considered as one of the most common modifiable risk factors for CHD(Di Angelantonio .,2009).

MATERIALS AND METHODS

Study design:-

A case-control study of 300 persons included two groups (150 CHD patients and 150 Control) randomly selected. All patients were diagnosed by specialist physicians as having CHD, were based on WHO guidelines.

Phenotype Measurements:-

We collected clinical data, such as weight, height, and other data. The Bady Mass Index was calculated as weight (in kg) divided by the square of height (in m). Blood was collected after an overnight fast (for 12-14 hours) in anticoagulant coated tubes and centrifuged after 15 minutes, the plasma was separated and lipid profile was measured according to the procedures provided with Biolabo kits. Total cholesterol (TC) was estimated using the Cholesterol Oxidase Phenol 4-Amino antipyrine peroxidase (CHOD-PAP) method.(Russel et al.,1996; Michael et al.,1996; Allain et al.,1974).Triacylglycerol's (TAG) levels were measured by GPO-PAP enzymatic method(Russel et al.,1996; Werner et al.,1981) High-density lipoprotein cholesterol (HDL-C)

and low-density lipoprotein cholesterol (LDL-C) were estimated using a two-step procedure: (i) precipitation and (ii) enzymatic determination (Russel et al.,1996).

Statistical analysis:-

Data are presented descriptively as means and standard deviation (M ± SD). Student T-test was used to compare phenotypic data between control and CHD groups using SPSS windows software (SPSS Inc., Chicago, IL). Values of P (two-tailed) less than 0.05 were considered statistically significant for all of the statistical analysis.

RESULTS:-

300 subjects aged between 40 to 69 years were included in this study (Figure 1). 54.6% were male and 45.3% female (Figure 2).

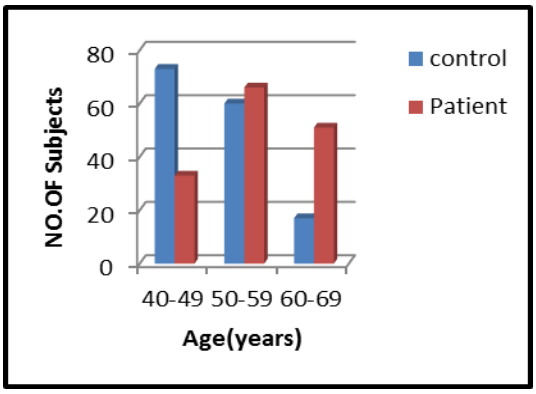

Figure 1: Age distribution of study individuals.

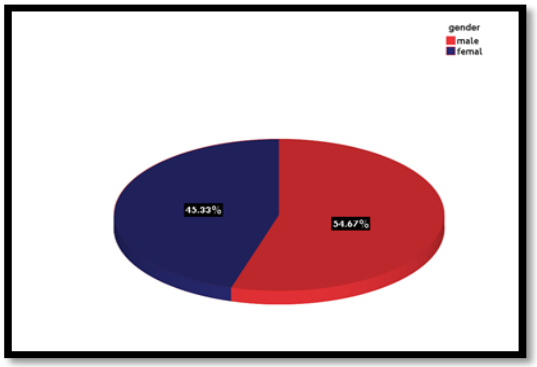

Figure 2: Gender distribution of study individuals.

Clinical and biochemical characteristics of study subjects are presented in [Table1]. 150 patients with CHD and 150 control were included in the study with a mean age of (57.41±6.38) and (49.16±4.6) years. Other basic characteristics of the study participants such as BMI of the CHD patients were statistically significantly (P = 0.001) decreased when compared with control group (22.76±2.91 vs. 28±3). There were statistically significantly of total cholesterol (P=0.001), TG (P=0.03) LDL-C (0.001) and HDL-C (P=0.013) in CHD group when compared with the control group.

Table (1): Anthropometric and biochemical characteristics of the patient and control groups

Parameters	**Control subjects (Mean± SD)**	**IHD subjects (Mean± SD)**	**P value**
No (M/F)	150(74/76)	150(90/60)	0.064
Age (y)	49.16±4.6	57.41±6.38	0.001
BMI (kg/m^2)	22.76±2.91	28±3	0.001
Cholesterol (mg/dl)	168.71±23.86	186.12±25.89	0.001
Triglycerides (mg/dl	116.78±34.73	124.39±27.82	0.033
VLDL (mg/dl)	24.00±7.54	25.00±6.00	0.231
LDL (mg/dl)	92.67±25.91	110.00±24.00	0.001
HDL (mg/dl)	57.94±15.17	54.5±7.18	0.013

Values are expressed as a mean ± standard deviation. BMI: body mass index; TG: triglyceride; LDL-C: low-density lipoprotein cholesterol; VLDL-C: very low - density lipoprotein cholesterol; HDL-C: high-density lipoprotein cholesterol.

DISCUSSION

Dyslipidemia was an important risk factor for atherosclerosis and Coronary heart disease (Fox, 2002). Where high levels of cholesterol, triglycerides, (LDL-C), and decreased HDL cholesterol (HDL-C) in Plasma are associated with increased risk for CHD(Molly et al., 2004 ; Sherbet *et al*., 2013;Kullisaar *et al*., 2016) Several mechanisms could explain the strong effect of lipid profile on the rate of CHD. Based on known the physiological functions of lipoproteins (Ferrari

et al., 2016). Atherogenic Lipoprotein that contains apoB (LDL- cholesterol), is known to promote cholesterol collection in macrophages as well as inflammatory responses inside the vessel wall, leading to atherosclerosis progression. Lipid deposition starts with the mobility of LDL from the blood into the wall of the vessel. Once within the media of the blood vessels it may move back into the bloodstream (a sign of lesional regression and a process that may be expedited by some lipid reducing strategies), or become oxidized (through direct activity of leukocytes or action of free radicals) or taken up by monocyte/macrophages which eventually become foam cells (Crowther.,2005).

high density lipoproteins (HDL) major apolipoprotein, apoA-I , which ordinarily carry about 20% of the total plasma cholesterol, transporting excess cholesterol from the arterial wall's foam macrophages to the liver(G.F.Lewis et al.,2005), The efflux of cholesterol from peripheral cells is largely facilitated through adenosine triphosphate-binding cassette transporters (ABC) and cholesterol esterification by lecithin-cholesterol acyltransferase (LCAT) helps modify these particles into more mature HDL2 and HDL3 particles (Huang., 2013; Bobom., 2017)**.** *Cholesterol ester transfer protein* (CETP) transfers additional cholesterol esters from apolipoprotein B-containing particles to HDL (G.F. Lewis et al.,2005; Zhang.,2012). Subsequently, these HDL particles are taken up by hepatocytes through scavenger receptor B1 (SR-B1) (G.F. Lewis et al.,2005; Zhou.,2015). So, The strong connection between low levels of HDL and the risk for atherosclerosis (Shao et al.,2009).

In this study observed that their elevated Triglyceride, Low Density Lipoprotein (LDL) ,cholesterol and decrease High Density lipoprotein (HLD) in patient of CHD that correspond with (Haddad et al.,2002) in Jordan; (Hassan et al.,2013) in Sudan;(M.Mohsen et al., 2013) in Egyptian that show the abnormal lipid profile remain strong risk factor of CHD.

CONCLUSION

In this background, it is concluded that Patients with CHD have altered lipid profile, with higher levels of TGs, total cholesterol, VLDL and LDL and low level of serum HDL; this difference may play a role in the pathophysiology found in Patients with CHD.

REFERENCES

1. Ahmed SM, Clasen ME, Donnelly JE.(1998). Management of dyslipidemia in adults. Am Fam Physician.57:2192.
2. Allain CL, Poon LS, Chan CSG, Richmond W, Fu PC.(1974).Enzymatic determination of total serum cholesterol. Clin Chem. 20:470-475.
3. Ashif M. (2011).Role of fruit, vegetables, and spice in diabetes. International Journal Nutrition Pharmacology Neurological Diseases.1:27–35.
4. Crowther MA. (2005).Pathogenesis of atherosclerosis. Hematology. 1(1):436–441.
5. Campos Wd, Stabelini Neto A, Bozza R, Ulbrich AZ, Bertin RL, Mascarenhas LP, Silva SG, Sasaki JE.(2010).[Physical activity, lipid consumption and risk factors for atherosclerosis in adolescents].Arq Bras Cardiol. 94(5):601-7.
6. Di Angelantonio E, Sarwar N, Perry P, Kaptoge S, Ray KK, Thompson A, et al.(2009). Major lipids, apolipoproteins, and risk of vascular disease. Jama. 302(18):1993–2000.
7. Ferrari, R., Aguiar, C., Alegria, E., Bonadonna, R.C., Cosentino, F., Elisaf, M., Farnier, M., Ferrières, J., Filardi, P.P., Hancu, N. and Kayikcioglu, M.(2016). Current practice in identifying and treating cardiovascular risk, with a focus on the residual risk associated with atherogenic dyslipidemia. European Heart Journal Supplements. 18.2-12.
8. Fox S.I. (2002) "Human Physiology",7th in. Eds. McGraw-Hill Higher Education. New York. 29 (5):396-398.
9. G.F. Lewis, D.J. Rader.(2005).New insights into the regulation of HDL metabolism and reverse cholesterol transport. Circulation Research.96:1221-1232.
10. Go AS, Mozaffarian D, Roger VL, Benjamin EJ, Berry JD, Blaha MJ, Dai S et al.(2014). Heart Disease and Stroke Statistics—2014 Update: A Report from the American Heart Association. Circulation. 129(3):228–292.
11. Haddad FH, Omari AA, Shamailah QM, Malkawi OM, Shehab AI, Mudabber HK, Shubaki MK. (2002).Lipid profile in patients with coronary artery disease. Saudi Med J. 23(9):1054-8.
12. Hassan Hussein Musa, Etayeb Mohamed Ahmed Tyrab, Muzamil Mahdi Abdel Hamid, Elbagire Abdel Rahman Elbashir, Lemya Mohammed Yahia and Nihad Mohammed Salih.(2013). Characterization of lipid profile in coronary heart disease patients in Sudan.Indian Heart J.; 65(2): 232–233.
13. JindrichSpinar.(2012). Hypertension and ischemic heart disease. Sciverse ScienceDirect .54: 433-438.
14. Jousilahti P, Vartiainen E, Tuomilehto J, Puska P.(1996). Twenty-year dynamics of serum cholesterol in the middle-aged population of eastern Finland. Ann Intern Med .125:713–722.
15. Kullisaar T, Zilmer K, Salum T, Rehem A and Zilmer M. (2016). The use of probiotics. Fermentum ME-3 containing Reg'Activ Cholesterol supplement for 4 weeks has a positive influence on blood lipoprotein profiles and inflammatory cytokines: an open-label preliminary study. Nutrition Journal.15:93.
16. Kumar V, Abbas AK, Aster JC.(2015). Robbins & Cotran Pathologic Basis of Disease, 9th Edition, Elsevier Saunders, Philadelphia PA 19103 - 2899, USA.
17. Molly, C., John, D.(2004). The Journal of Clinical Endocrinology and Metabolism.89: 2601-2607.
18. Michael L; Bishop; Janet L; Duben-Engelkirk; Edward P; Fody.(1996). Clinical Chemistry. Principles, Procedures, Correlations, 3rd edition. 313-318.
19. M. Mohsen Ibrahim, Ahmed Ibrahim, Khaldoon Shaheen, Mona A. Nour.(2013). Lipid profile in Egyptian patients with coronary artery disease. The Egyptian Heart Journal.65(2);79-85.
20. Paratz Z, Sonny Palmer, Justin Mariani, Fracp, Fcsanz.(2015).The cardiac complications of obesity. Medicine Today. 16(11):211–232.
21. Russel Warenick G, Judith R, McNamara, Lily L.(1996). Lipids and lipoproteins. Bishop ML (ed), Clinical Chemistry. Principles, Procedures, Correlations. 3rd edition. Lippincott. 313-314.
22. Sherbet DP, Garg P, Brilakis ES, Banerjee S. (2013). Low-density lipoprotein cholesterol: how low can we go?. American Journal *of* Cardiovascular Drugs .13 (4):225–32.
23. Shao B, Heinecke JW. (2009).HDL lipid peroxidation, and atherosclerosis. Journal of Lipid Research.50:599–601.

24. Sarwar N, Danesh J, Eiriksdottir G, Sigurdsson G, Wareham N, Bingham S, Boekholdt SM, Khaw KT, Gudnason V.(2007).Triglycerides and the risk of coronary heart disease: 10,158 incident cases among 262,525 participants in 29 Western prospective studies.Circulation. 115(4):450-8.

25. Werner M, Garberielson DG, Estman J.(1981). Ultramicro determination of serum triacylglycerols by the bioluminescent assay. Clin Chem. 27:268-271.

26. World Health Organization - 2015.

27. Zhang H, Mo X, Hao Y, Gu D. (2012). Association between polymorphisms in the adiponectin gene and cardiovascular disease: a meta-analysis. Bio-Medical Center Medical Genetics.13:40.

28. Zhou L, Li C, Gao L, Wang A. (2015). High-density lipoprotein synthesis and metabolism (Review). Molecular Medicine Reports.12 (3):4015-4021.