

Study of Lipid Profile in Primary Hypertension

Nadera Yasmeen^{1*}, Syeda Amatul Rahman Jaweria², Imtiyaz Ali³^{1*}Associate Professor, Department of Physiology, Malla Reddy Medical College for Women, Hyderabad, India²Intern, Deccan College of Medical Sciences, Hyderabad, India³Professor, Department of Physiology, Faculty of Medical Sciences, Gulbarga, Karnataka, India

Received: 25-09-2023 / Revised: 28-10-2023 / Accepted: 30-11-2023

Corresponding author: Dr. Nadera Yasmeen

Conflict of interest: Nil

Abstract:

Hypertension is a major public health problem in India and studies have been documented a high prevalence rate of hypertension in developing countries [1]. Cardiovascular diseases account for a large proportion of all deaths and disability worldwide. The elevation of blood pressure is secondary to raised total peripheral resistance, which in turn is caused by narrowing of the lumen of the peripheral arterioles, which are the main resistance vessels. The aim of the present study is to compare and analyse the relation between blood pressure and lipid parameters in hypertensives and normotensives so that it will be a useful predictor of coronary artery disease. The study is undertaken in 50 hypertensives taken as subjects and 50 normotensives taken as controls with the age range being 40- 60 yrs. Ethical committee clearance was taken and consent obtained from all the study subjects. In the present study, it was found that the mean values of Total cholesterol and LDL cholesterol, are higher with more significance ($p < 0.01$) and VLDL cholesterol and Triglycerides are higher with less significance ($p < 0.05$) in hypertensives. The mean values of HDL Cholesterol are found to be lower with less significance ($p < 0.05$) in hypertensives. Thus, it can be concluded that in patients of primary hypertension, significant changes are seen in lipid profile which can be used for early detection of individuals prone to hypertension.

Keywords: Hypertension, Hypertriglyceridemia, Coronary Atherosclerosis.

This is an Open Access article that uses a funding model which does not charge readers or their institutions for access and distributed under the terms of the Creative Commons Attribution License (<http://creativecommons.org/licenses/by/4.0>) and the Budapest Open Access Initiative (<http://www.budapestopenaccessinitiative.org/read>), which permit unrestricted use, distribution, and reproduction in any medium, provided original work is properly credited.

Introduction

Hypertension is a major cause of both haemorrhagic and infarctive strokes and is one of the three important risk factors for coronary heart disease [2]. The elevation of blood pressure in the larger capacitance vessels is associated with an increased risk of damage to large vessels, mainly in the form of occlusive vascular disease. Hypertriglyceridemia is an important risk factor for coronary atherosclerosis as hypercholesterolemia [3].

Aims and Objectives

The aim of the present study is to compare and analyse the relation between blood pressure and lipid parameters in hypertensives and normotensives so that it will be a useful predictor of coronary artery disease through regular investigations.

Identification of these high risk patients may allow an earlier introduction of antihypertensive treatment and correction of the risk factors to prevent the progression or to induce the regression of silent vascular damage before a clinical event develops by instructing change in the lifestyle or prescribing medications or both.

Materials and Methods

The study is undertaken in 50 hypertensives taken as subjects and 50 normotensives taken as control with the age range being 40- 60 yrs. Ethical committee clearance was taken and consent obtained from all the study subjects.

Blood pressure was recorded in the sitting posture in the right arm using mercury sphygmomanometer using both palpatory and auscultatory method. Three readings are taken and the average of the three recordings is obtained. Blood samples are collected after an overnight fasting from antecubital vein by making the subject to sit comfortably on a chair. About 3 ml of blood is collected in vacutainers through disposable syringe under aseptic precautions and allowed to stand for 30 min at room temperature to allow complete clotting and clot retraction. Samples are centrifuged at 2000 g for 15 mins and readings are taken using the Semi-automated analyser. [Erba Mannheim]. Serum cholesterol, HDL Cholesterol, LDL cholesterol, VLDL cholesterol and serum triglycerides are estimated using reagent assay kits from Transasia Erba Biomedicals Ltd.

Result and analysis:

The data is analysed and all values are expressed as Mean ± standard deviation. Statistical significance of differences between control and study groups are evaluated by student “t” test .

A p- value of < 0.05 is considered to be statistically significant and p- value of < 0.01 is considered to be highly significant.

The mean values of lipid profile are presented in Table 1

Table 1:

Parameter	Normotensive (MEAN ± SD)	Hypertensive (MEAN ± SD)	P value	Significance
Total Cholesterol	194.52 ± 16.38	210.48 ± 25.56	< 0.01	HS
HDL	41.18 ± 5.49	39.32 ± 4.50	< 0.05	S
LDL	124.68 ± 15.35	138.58 ± 24.83	< 0.01	HS
VLDL	28.66 ± 4.61	31.31 ± 7.80	< 0.05	S
Triglycerides	143.28± 23.05	156.56 ± 39.01	< 0.05	S

HS- Highly significant

The mean value of Serum cholesterol in hypertensive group is found to be 210.48 mg/dl (SD ±25.56), while in normotensive controls it is found to be 194.52 mg/dl (SD ±1 6.38).

The mean value of Serum HDL Cholesterol in hypertensive group is found to be 39.32 mg/dl (SD ± 4.50), while in normotensive controls it is found to be 41.18 mg/dl (SD ± 5.49).

The mean value of Serum LDL cholesterol in hypertensive group is found to be 138.58 mg/dl (SD ± 24.83), while in normotensive controls it is found to be 124.68 mg/dl (SD ± 15.35).

The mean value of Serum VLDL cholesterol in hypertensive group is found to be 31.31 mg/dl (SD ± 7.80), while in normotensive controls it is found to be 28.66 mg/dl (SD ± 4.61).

The mean value of Triglyceride in hypertensive group is found to be 156.56 mg/dl (SD ± 39.01),

while in normotensive controls it is found to be 143.28 mg/dl (SD ± 23.05).

The mean values of Total cholesterol and LDL cholesterol are higher with high significance. (p < 0.01).

The mean values of VLDL cholesterol and triglycerides are higher with less significance. (p < 0.05) while the mean value of HDL cholesterol is lower with less significance (p <0.05) in hypertensive group.

Discussion

In the present study, the mean values of Total Cholesterol, LDL Cholesterol, VLDL Cholesterol and Trigylcerides are found to be significantly higher while HDL Cholesterol is lower in hypertensive group.

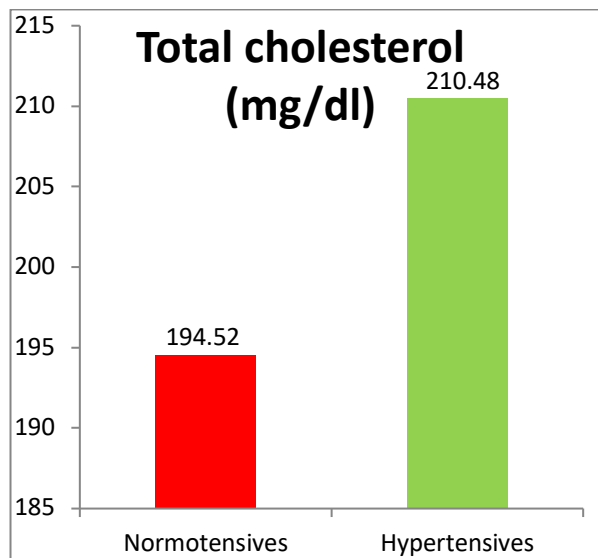


Figure 1: Total Cholesterol (mg/dl)

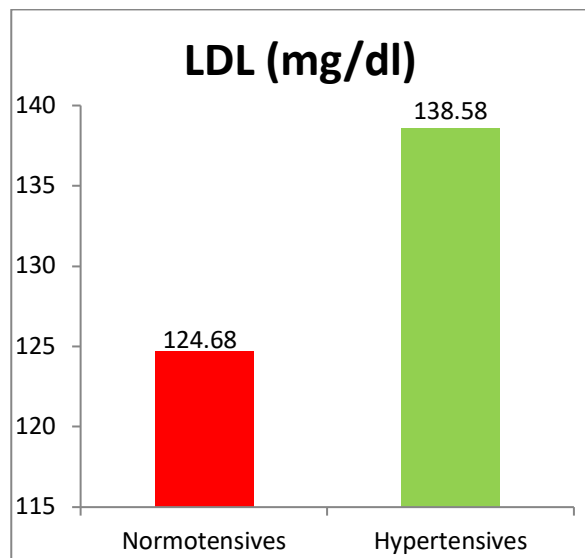


Figure 2: LDL (mg/dl)

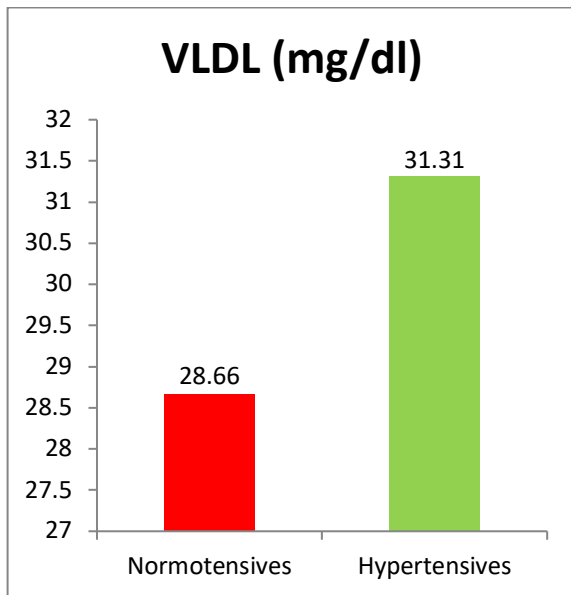


Figure 3: VLDL (mg/dl)

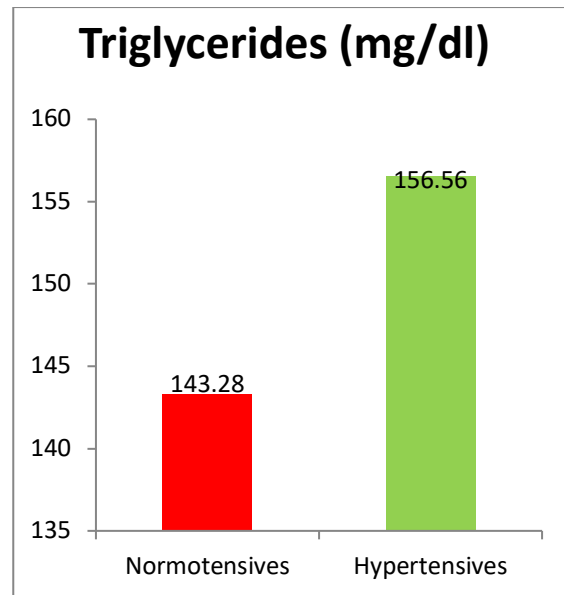


Figure 4: Triglycerides (mg/dl)

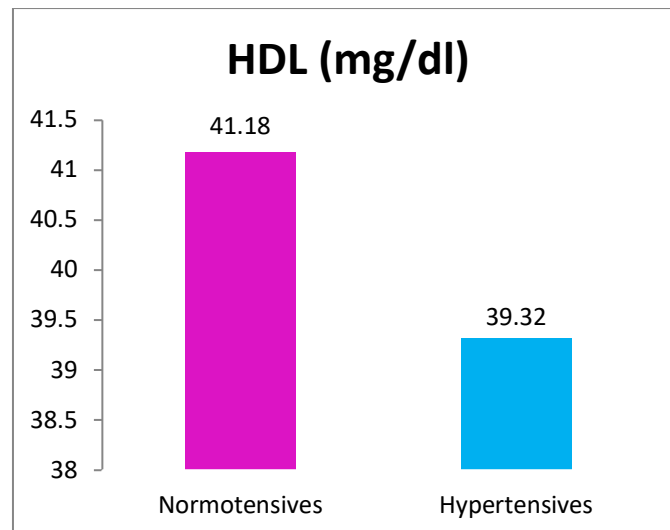


Figure 5: HDL (mg/dl)

These findings are similar to the findings reported by Joseph L et al [3], J.H. Fuller [4], Steven C. Hunt [5] M.S.Saha et al [6], Jose Tunon et al [7], David E. L. et al [8], Dong-Jun et al [9], Cristina Sierra [10], Alan Morrison [11] and Al- Muhana et al [12].

Jose Tunon et al studied on common pathways of hypercholesterolemia and hypertension leading to atherothrombosis. They stated dyslipidaemia is responsible for hypertension by causing the atherosclerotic and atherothrombotic changes. All major atherothrombotic risk factors induce endothelial dysfunction. The key feature in this disorder is a reduced availability of nitric oxide (NO) due to both a decrease in its synthesis and to an enhanced degradation. Oxidised LDL diminishes the expression of endothelial NO synthase (eNOS). Furthermore, in hypercholesterolemia there is an increase of asymmetric dimethyl arginine levels, an eNOS endogenous inhibitor. Angiotensin II may

also down regulate eNOS expression via protein – kinase C, thus leading to a decrease in NO production. Due to endothelial dysfunction, there is an increase in vascular permeability to LDL, which becomes oxidised in the arterial wall where the macrophages uptake them evolving into foam cells. In addition, ACE is present in greater amounts in atherosclerotic plaques.

Thrombosis leads to acute coronary syndromes and ischaemic stroke. It begins with platelet adhesion and aggregation, followed by activation of the coagulation cascade. Tissue factor activation is the first step of the coagulation cascade in vascular thrombosis and it is present in the lipid core component of the atherosclerotic plaques. Angiotensin II induces tissue factor expression in human monocytes via the protein kinase C pathway, and ARBs decrease tissue factor activity in hypertensive patients. Also Renin Angiotensin

system is involved in the regulation of the endogenous fibrinolytic system. Moreover ACE inhibitors block bradykinin degradation, and this peptide induces the expression of t-PA (tissue plasminogen activator) [7]. David E.L. et al assessed the association of dyslipidemia with incident hypertension. In their study, abnormal triglyceride and LDL metabolism seemed to be strongly associated with the development of hypertension. In multiple regression analysis of the lipid, lipoprotein variables separately, 1-SD increments in the concentrations of triglycerides and triglyceride content of HDL cholesterol were associated with a 1.4-1.8 fold higher risk of developing hypertension. Daugherty et al have shown in mice that hypercholesterolemia is associated with increased levels of circulating angiotensinogen and angiotensin peptides and that all the components of the renin- angiotensin- aldosterone system (RAAS) , including renin, are overexpressed within atherosclerotic lesions. Furthermore, these epidemiological associations may fuel studies on the biological mechanisms linking lipid metabolism to blood pressure regulation [8].

Summary and conclusion

In the present study, the variables found significant can be suggested as predictors of hypertension. Hyperlipidemia is seen in hypertensives. In the setting of endothelial dysfunction, there is an increase in vascular permeability to LDL, which becomes oxidised in the arterial wall where the macrophages uptake them evolving into foam cells. Triglyceride – rich lipoproteins and LDL cholesterol have been shown to be toxic to endothelial cells, whereas HDL cholesterol may be protective. Therefore long term damage to the endothelium may lead to increased peripheral resistance and thus to arterial hypertension. Dyslipidemia may also cause hypertension by increasing arterial stiffness.

From the above study, it can be concluded that that in patients of primary hypertension, significant changes are seen in lipid profile which can be used for early detection of hypertensive prone individuals.

References

1. Shyamlal Kumar Das, Kalyan Sanyal, Arindam Basu. Study of urban community survey in India: growing trend of high prevalence of hypertension in a developing country. *Int. J. Med. Sci.* 2005;2: 70-78.
2. G.W.K. Ching, D.G. Beavers. Hypertension. *Postgrad Med J.* 1991;67: 230-246.
3. Joseph I. Goldstein, William R. Hazzman, Helmut G. Scinott, Edwin I. Bierman, and Arno G. Motulsky. Hyperlipidemia in Coronary Heart Disease. The Association of American Physicians, Atlantic City, N.J., 2-3 May 1972.
4. J.H.Fuller, S. Pinney, R.J. Jarrett, K. Kilbourn and H. Keen. Plasma lipids in a London population and their relation to other risk factors for coronary heart disease. *British Heart Journal*, 1978; 40: 170-176.
5. Steven C. Hunt, Susan H. Stephenson, Paul N. Hopkins and Roger R. Williams. Predictors of an Increased risk of future hypertension in Utah (1991).
6. M.S. Saha, N K Sana and Ranajit Kumar Shaha. Serum Lipid Profile of hypertensive patients in the northern region of Bangladesh. *J. bio-sci.* 2006; 14: 93-98.
7. Jose Tunon, Jose Luis Martin -Ventura, Luis Miguel Blanco- Colio, Nieves Tarin, Jesus Egido. Common pathways of hypercholesterolemia and hypertension leading to atherothrombosis: the need for a global approach in the management of cardiovascular risk factors. *Vascular Health and Risk Management.* 2007; 3(4):521-526.
8. David E. Laaksonen, Leo Niskanen, Kristiina Nyyssönen, Timo A. Lakka, Jari A. Laukkanen and Jukka T. Salonen. Dyslipidaemia as a predictor of hypertension in middle - aged men. *European Heart Journal.* 2008; 29: 2561-2568.
9. Dong-Jun Kim, Jung- Hyun Noh, Byung-Wan Lee, Yoon-Ho Choi, Jae-Hoon Chung, Yong-Ki Min, Myung-Shik Lee, Moon- Kyu Lee and Kwang- Won Kim. The association of total and differential white blood cell counts with obesity, hypertension, dyslipidemia and Glucose intolerance in a Korean population. *J. Korean Med Sci.* 2008; 23: 193-8.
10. Cristina Sierra, Alejandro de la Sierra. Early detection and management of the high- risk patient with elevated blood pressure. *Vascular Health and Risk Management.* 2008; 4(2): 289-296.
11. Alan Morrison, John E. Hokanson. The independent relationship between triglycerides and coronary heart disease. *Vascular health and risk management.* 2009; 5: 89-95.
12. F.A. Al- Muhana, E.B. Larbi, A.K. Al-Ali, A. Al- Sultan, S. Al- Ateeq, I. Soweilem, I. Goa, A.A. Bahnassy, A. Al- Rubaish and M.F. Abdul mohsen. Hematological, lipid profile and other biochemical parameters in normal and hypertensive subjects among the population of the eastern province of Saudi arabia. *East African medical journal.* 2006; 83: 1.