

# Effect of Hypertension on Lipid Profile of Individuals of Bihar State

Kumari Rekha<sup>1</sup>, Rajiv Ranjan Prasad<sup>2</sup>

<sup>1</sup>Junior Resident, Department of Physiology, Patna Medical College and Hospital, Patna, Bihar, India, <sup>2</sup>Professor, Department of Physiology, Patna Medical College and Hospital, Patna, Bihar, India

## Abstract

**Introduction:** Hypertension and dyslipidemia are major risk factors for coronary artery disease (CAD) and account for >80% of death and disability in the low- and middle-income countries.

**Materials and Methods:** The study was conducted in the Department of Physiology, Patna Medical College and Hospital (PMCH), Patna. 60 patients of hypertension aged 40-50 years attending Outpatient Department of PMCH and 40 healthy volunteers were recruited in the present study to see the effect of hypertension on the lipid profile.

**Results:** Hypertensives showed a highly significant upper range of triglyceride (TG) with  $P < 0.01$ . Total cholesterol (TC) and very-low-density lipoprotein (VLDL) showed a partially significant upper range in hypertensives with  $P < 0.1$ , whereas high-density lipoprotein (HDL) and LDL showed no variations between these two groups with  $P > 0.4$ .

**Conclusion:** It was concluded that patients with hypertension are more likely to exhibit dyslipidemia, including elevated TC, TG, VLDL, and reduced HDL levels. So, they need a measurement of blood pressure and lipid profile at regular intervals to prevent the risk of CAD and stroke.

**Key words:** Blood pressure, Coronary artery disease, Low-density cholesterol, Triglyceride, Total cholesterol

## INTRODUCTION

Hypertension and dyslipidemia are major risk factors for coronary artery disease (CAD) and account for >80% of death and disability in the low- and middle-income countries.<sup>1,2</sup> The prevalence of hypertension is projected to increase globally, especially in the developing countries. The co-existence of the two risk factors has more than an additive adverse impact on the vascular endothelium, which results in enhanced atherosclerosis, leading CAD.<sup>3</sup>

In the recent years, rapid urbanization, increased life expectancy, unhealthy diet, and lifestyle changes have led to an increased rate of CAD. It is widely accepted that CAD is associated with hypertension and increased blood levels

of triglyceride (TG), total cholesterol (TC), and low-density lipoprotein (LDL).<sup>4</sup>

The Framingham Heart Study data on the hypertensive population reported that more than 80% had at least on additional cardiovascular disease (CVD) risk factors such as obesity, glucose intolerance, and dyslipidemia.<sup>5</sup> The risk of concomitant hypertension and dyslipidemia is more multiplicative than the sum of the individual risk factors.<sup>6</sup>

Dyslipidemia, one of the strong predictors of CVD, causes endothelial damage and loss of physiological vasomotor activity. The damage may manifest as elevated systemic blood pressure (BP).<sup>7</sup>

Hypertension, damages the endothelium through altered shear stress and oxidative stress, resulting in increased endothelial cell synthesis of collagen and fibronectin, reduced nitric oxide-dependent vascular relaxation, and increased permeability to lipoprotein. It is also associated with an upregulation of lipid oxidation enzymes, especially oxidized LDL contributing to atherosclerosis.<sup>8,9</sup>

Access this article online



www.ijss-sn.com

**Month of Submission :** 06-2016  
**Month of Peer Review :** 07-2016  
**Month of Acceptance :** 08-2016  
**Month of Publishing :** 08-2016

**Corresponding Author:** Dr. Kumari Rekha, C-126, Harmu Housing Colony, Near Sahjanand Chawk, P.O. Argora, Ranchi - 834 012, Jharkhand, India. Phone: +91-9631070033. E-mail: zingipal@gmail.com

## MATERIALS AND METHODS

Blood samples were obtained after an overnight fast. About 5 ml of blood was collected from the left antecubital vein. Of which, about 2 ml is transferred into an overfast vial and mixed well and centrifuged at a speed of 3000 revolutions per minute for 10 min to separate the plasma, which was used for biochemical analysis. Rest 3 ml of blood is transferred to the test tube, and this blood was allowed to clot to get serum. This serum was separated in a centrifuge tube at 3000 revolutions per min to get a clear sample of serum. This clear supernatant serum was used for biochemical investigation.

### Estimation of Serum TC

Method: Enzymatic - (colorimetric trinder end point).

The reagents were allowed to attain room temperature before use.

	Blank	Standard	Sample
Reagent R	1000 µl	1000 µl	1000 µl
Standard	-	10 µl	-
Sample	-	-	10 µl

They were incubated for 5 min at 37°C and reading was done against the blank at 500 nm and calculation was made. The concentration of cholesterol in the sample is directly proportional to the intensity of red complex (red quinone), which was measured at 500 nm.

Calculation:

Cholesterol = Absorbance of sample/absorbance of standard × concentration of standard.

### Estimation of Serum TG Method

Enzymatic: Colorimetric method contents were mixed and incubated for 5 min at 37°C. The reading was done against blank at 546 nm.

	Blank	Standard	Sample
Reagent R	1000 µl	1000 µl	1000 µl
Standard	-	10 µl	-
Sample	-	-	10 µl

Calculation:

Serum TG = Absorbance of sample/absorbance of standard × n

n = Standard concentration.

Reference values: >150 mg/dl.

### Estimation of High-density Lipoprotein (HDL)-cholesterol

Method: Phosphotungstate method.

Principle: Chylomicrons, LDL, and very-LDL (VLDL) are precipitated by addition of phosphotungstic acid and magnesium chloride. After centrifugation, the HDL fraction remains in the supernatant is determined with cholesterol oxidase/peroxidase aminophena method.

Reference value: >40 mg/dl.

Calculation of LDL and VLDL by Friedewald's formula:

$$LDL = TC - (HDL + VLDL)$$

$$VLDL = TG/5$$

Reference value:

$$LDL = \text{Up to } 190 \text{ mg/dl.}$$

$$VLDL = 14\text{-}31.8 \text{ mg/dl.}$$

### BP

It was measured using standard BP measurement protocol after the patient had rested for 10 min. Two measurements were taken by a mercury sphygmomanometer, with at least a 5-min interval between successive measurements. Hypertension was defined as an average systolic BP ≥140 mm of Hg and diastolic BP ≥90 mm of Hg without antihypertensive medication.

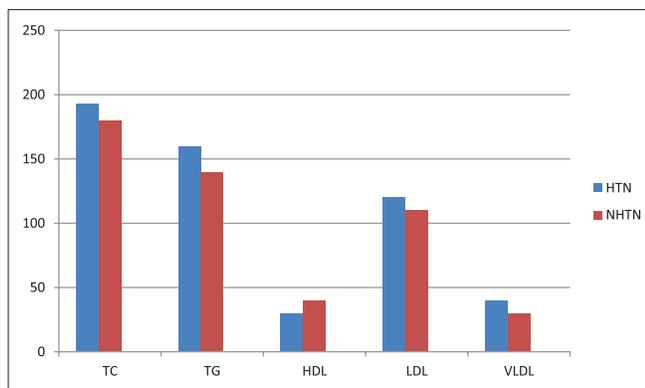
## RESULTS

Hypertensive showed a highly significant upper range of TG. Levels of TC and VLDL were also in partially significant higher range in hypertensive while HDL and LDL were of the same range in both groups (Table 1 and Figure 1).

**Table 1: Lipid level variations among hypertensives and normotensives (mean±SD) in mg/dl**

Lipids type	Hypertensive n=60	Normotensive n=40	t	P	Significance
TC	193.93±20.46	180.17±17.08	1.83	<0.1	PS
TG	164.02±17.87	144.09±15.33	2.44	<0.01	HS
HDL	40.27±5.03	44.77±7.81	0.79	>0.4	NS
LDL	112.28±19.78	106.21±18.81	0.80	>0.4	NS
VLDL	32.80±3.57	28.80±3.07	1.42	<0.1	PS

TG: Triglyceride, TC: Total cholesterol, VLDL: Very-low-density lipoprotein, HDL: High-density lipoprotein



**Figure 1: Lipid level variations among hypertensives and normotensives (mean in mg/dl)**

## DISCUSSION

In this study, we investigated the relationship between serum lipid profile and hypertension among the individuals of Bihar state. Results of this study revealed that the mean value of serum TG was significantly higher and statistically significant. Levels of TC and VLDL were also in partially significant upper range in hypertensive than normotensive population. Various workers such as MacMohan *et al.*, Coffin *et al.* (1990), and Samuelsson *et al.* had established a significant correlation between baseline BP and subsequent development of CAD. The management of these disorders, particularly in high-risk patients, requires multiple interventions, including dietary

and pharmacological. There is a need to increase the awareness, both in the medical and patient communities, for early detection and treatment of these two conditions to decrease the incidence of future CAD.<sup>10</sup>

## REFERENCES

1. Reddy KS. Cardiovascular disease in non-Western countries. *N Engl J Med* 2004;350:2438-40.
2. Yusuf S, Reddy S, Ounpuu S, Anand S. Global burden of cardiovascular diseases: Part II: Variations in cardiovascular disease by specific ethnic groups and geographic regions and prevention strategies. *Circulation* 2001;104:2855-64.
3. Murray CJ, Lopez AD. Global mortality, disability, and the contribution of risk factors: Global Burden of Disease Study. *Lancet* 1997;349:1436-42.
4. Goyal A, Yusuf S. The burden of cardiovascular disease in the Indian subcontinent. *Indian J Med Res* 2006;124:235-44.
5. Hansson L, Zanchetti A. The Hypertension Optimal Treatment (HOT) Study - patient characteristics: Randomization, risk profiles, and early blood pressure results. *Blood Press* 1994;3:322-7.
6. National Heart, Lung and Blood Institute. Third Report of the National Cholesterol Education Program (NCEP). Expert Panel Detection, Evaluation and Treatment of High Cholesterol in Adults (ATP III). Bethesda: NIH Publication No. 01-3670; 2001.
7. Wong ND, Lopez V, Tang S, Williams GR. Prevalence, treatment, and control of combined hypertension and hypercholesterolemia in the United States. *Am J Cardiol* 2006;98:204-8.
8. O'Donnell VB. Free radicals and lipid signaling in endothelial cells. *Antioxid Redox Signal* 2003;5:195-203.
9. Kaplan M, Aviram M. Oxidized low density lipoprotein: Atherogenic and proinflammatory characteristics during macrophage foam cell formation. An inhibitory role for nutritional antioxidants and serum paraoxonase. *Clin Chem Lab Med* 1999;37:777-87.
10. Kannel WB, Carter BL. Initial drug therapy for hypertensive patients with hyperlipidemia. *Am Heart J* 1989;118:1012-21.

**How to cite this article:** Rekha K, Prasad RR. Effect of Hypertension on Lipid Profile of Individuals of Bihar State. *Int J Sci Stud* 2016;4(5):197-199.

**Source of Support:** Nil, **Conflict of Interest:** None declared.