

Study of Lipid Profile, Serum Magnesium and Blood Glucose in Hypertension

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Abstract:

The aim was made to study the function of blood glucose, serum Mg⁺², and lipid profile in individuals with hypertension. Furthermore, a biochemical analysis is performed on each parameter. About 80 samples (50 cases and 30 controls) show that hypertensive people with dyslipidemia had unchanged HDL concentrations. Serum magnesium levels in hypertension patients and controls do not correlate. Serum magnesium levels in hypertension subjects have been found to be somewhat higher than in normal persons. The hypertension subjects' (101.62 mg/dl ±33.78) fasting blood glucose level is greater than the controls' (82.46 mg/dl ±10.8). There has been a statistically significant rise (p<0.001). However, 12% of the cases may be diabetic situations which could explain this increase. Even yet, hypertensive individuals have a propensity to have reduced glucose tolerance. Systolic and diastolic blood pressure readings are recorded independently for each measurement of blood pressure. As age groups increased, the systolic blood pressure became more relevant than the diastolic blood pressure.

Keywords: Hypertension, Serum Magnesium, Dyslipidemia.

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Introduction

Instead of being a particular disease, hypertension is characterized as a trait, and it deviates from the norm in terms of quantity rather than quality. Blood pressure is the force that propels blood through blood arteries to remove waste products and metabolites from the body's tissues and organs as well as to give oxygen and nutrients to them. When the diastolic blood pressure (DBP) is less than 80mm Hg and the systolic blood pressure (SBP) is less than 120mm Hg, blood pressure is considered optimum. A SBP reading of more than 140 mmHg and/or a DBP reading more than 90 mmHg are considered hypertension. Chronic hypertension, or increased arterial blood pressure, is a widespread health issue with a continuing rise in occurrence worldwide. About 25% of adult populations are impacted. Hypertension is characterized by abnormalities in cardiac output, systemic vascular resistance, and arterial compliance, despite its historical definition of "an elevation of blood pressure" alone. [6] According to the WHO Report 2002, the top 10 risks for disease burden both globally and regionally are: smoking, fuel-related smoke, high blood pressure, alcohol use, underweight, iron deficiency, unsafe water, high cholesterol, obesity, and sanitation and hygiene. [3]

When combined, these cause more than one-third of all fatalities globally. Combining data from epidemiological research revealed that in India, hypertension affects 10% (31.5 million) of people in rural areas and 25% (34 million) of persons in cities. Of these, 70% would have Stage I hypertension.

Primary or essential hypertension and secondary hypertension are the two categories into which hypertension are divided. An "unknown cause" rise in blood pressure is known as primary hypertension. The term "increase in blood pressure caused by diseases of the kidney, endocrines, or some other organs" refers to secondary hypertension. Malignant hypertension occurs in less than 5% of hypertensive people.

Based on the raised blood pressure, hypertension is further classified into three stages.1 Chronic high blood pressure can cause harm to the kidneys (benign and malignant hypertension), brain (cerebrovascular accident), and heart (hypertensive cardiomyopathy). Systolic hypertension is associated with a higher risk in individuals 50 years of age or older. [16] The differing effects of SBP

and DBP on blood pressure staging in a middle-aged Spanish sample that is indicative of the country's high-risk cardiovascular disease population.

It is commonly known that high blood pressure is linked to an increased risk of stroke and coronary heart disease. Strong correlations have also been shown between serum cholesterol levels and CHD risks. Extensive epidemiological research has shown that the prevalence of hypercholesterolemia, diabetes, hypomagnesemia, hypertriglyceridemia, and other conditions is significantly higher in individuals with hypertension. In addition to examining risk in women over a wide range of baseline BMI (kg/m²) values, the longitudinal study investigated the impact of subgroup analyses on the incidence of hypertension by measuring waist circumference (WC, cm), percent body fat, and fat mass (FM, kg). Over a mean follow-up of 16.7 years, 592 women reported having hypertension. Higher BMI was linked to a higher risk of hypertension, even when it fell under the "normal" range. [14]

This led to the current investigation of lipid profiles in individuals with hypertension. The investigation included an estimation of magnesium in light of the element's critical function in a number of metabolic processes, particularly those requiring cellular energy (ATP). Magnesium is the second most prevalent cation inside cells and the fourth most abundant cation overall in the human body.

For an adult weighing 70 kg, the average body magnesium content is approximately 2,000 mEq. Of this, between 50 and 70 percent are found in bones, 1 percent in ECF, and the remainder is found inside cells, primarily in the mitochondria. [12] Blood glucose levels are also included in cases of hypertension in order to determine whether hyperglycemia and hypertension are related. Changes in lifestyle, sugar-rich diets, high-fat processed foods, and sedentary behavior are among the factors contributing to the rising rate of hypertension. [8] Among the common metabolic illnesses is "dyslipidemia." Hyperlipidemia or lipoprotein abnormalities might be secondary symptoms of another ailment or the fundamental cause of an anomaly in lipid metabolism. The prevalence of dyslipidemia in young people (7–17 years old) with type 2 diabetes and looked studied how lipid parameters related to other established cardiovascular risk factors. The significance of a

complete lipid panel, which includes apo-B, in identifying potentially modifiable cardiovascular risk in a population with high rates of obesity, smoking, and inadequate glycemic control is highlighted by elevated apo B levels with normal LDL-C levels. [13]

Materials and Methods

From July 2022 to June 2023, the study was conducted in the biochemistry department of Patna Medical College in Patna, Bihar. The study had two groups: Cases and Controls. 50 hypertension patients between the ages of 30 and 71 who were seen at the General Medicine department were recruited for the study (27 men and 23 women). Cases of secondary hypertension were not included in the study. A total of thirty participants who were matched for age and sex and who had no prior medical history of diabetes, hypertension, heart disease, or kidney disease were present as attendees.

All subjects had their blood pressure monitored in accordance with JNC-VIII (2014) guidelines. All blood samples are taken from the subject while they are fasting and without the use of an anticoagulant. The blood is then centrifuged for five minutes at 1500 rpm to extract the serum, which is then collected in a fresh vial for biochemical analysis using the following standard procedures.

The data was analyzed by SPSS software version 20.

- Serum magnesium: Calmagite method
- Serum total cholesterol: Cholesterol oxidase Method
- Triglycerides: Glycerolkinase, Peroxidase, method
- HDL: Precipitation method
- LDL: $LDL-C (MG/DL) = Total\ cholesterol - (HDL-C + VLDL-C)$
- VLDL: $VLDL-C (mg/dl) = triglycerides/5$
- Blood Glucose: Glucose oxidase and Peroxidase method

Results

The study included 80 patients, 30 of whom were controls and 50 of whom were hypertension cases. Serum magnesium, fasting blood glucose, lipid profile, and blood pressure were measured for each group. The study's outcome is provided below.

Table 1: Age-wise distribution in cases and controls

Sl. No.	Age Group (years)	Case	Controls
1	30-39	04	25
2	40-49	11	13
3	50-59	13	07
4	60-69	20	05
5	70-79	02	-

The age range of the hypertensive cases is 30-71 years old. The average age is 55.53 ± 10.61 years; while the controls in the 31–69 age range are 48.53 ± 9.58 years old (table 1).

Table 2: Sex-wise distribution in cases and controls

Group	Males		Females		Total
	Number	%	Number	%	
Cases	27	54	23	46	50
Controls	18	36	12	24	30

Out of 50 cases, 27 are males and 23 are females. In case of 30 controls, 18 are males and 12 are females (table-2).

Table 3: Group status in cases and controls

Group Status		SBP (mmHg)	DBP (mmHg)	FBS (mg/dl)	Mg (mg/dl)
Cases (n=50)	Mean	163.7	87.64	101.62	1.94
	SD	16.72	17.85	33.78	0.05
Control (n=30)	Mean	114.66	73.33	82.46	1.92
	SD	6.62	0.53	10.8	0.08

The diastolic and systolic components of blood pressure are computed independently. The hypertensive sufferers' mean SBP is 163.7 ± 16.72 mmHg, while the controls' mean SBP is 114.66 ± 6.62 mmHg. Subjects with hypertension have a higher mean SBP than controls ($p < 0.001$). The hypertensive cases' mean DBP is 87.64 ± 17.85 mmHg, while the controls' mean DBP is 73.33 ± 0.53 mmHg. In Table 3, the mean for cases is greater than that of controls ($p < 0.05$). Hypertensive patients have an average fasting blood glucose of 101.62 ± 33.78 mg/dl. The average

blood glucose level of controls after fasting is 82.46 ± 10.8 mg/dl. Subjects with hypertension had a mean that is greater than controls ($p < 0.05$). Nonetheless, 10% of hypertensive people may have diabetes, which could account for the increase. (Table: 3) The hypertension participants' mean serum magnesium level is 1.94 ± 0.05 mEq/L, while the control group's mean is 1.92 ± 0.08 mEq/L. Table 3 shows that although the mean of hypertensive cases is greater than that of controls, the difference is not statistically significant ($p > 0.05$).

Table 4: Group status in cases and controls

Group Status		TC (mg/dl)	HDL (mg/dl)	LDL (mg/dl)	VLDL (mg/dl)	TGL (mg/dl)
Cases (n=50)	Mean	209	41.1	134.31	35.77	180.88
	SD	31.63	5.92	29.24	13.43	68.5
Control (n=30)	Mean	172.8	42.94	107	22.87	114.7
	SD	13.43	3.56	13.1	3.46	17.62

In hypertension situations, the mean total cholesterol is 209 ± 31.63 mg/dl. The controls have a mean total cholesterol of 172.8 ± 13.43 mg/dl. Compared to controls, the mean of hypertension patients is greater ($p < 0.001$).

The average HDL of individuals with hypertension is 41.1 ± 5.92 mg/dl, while controls had a mean of 42.94 ± 3.56 mg/dl. There is no statistically significant difference in the mean HDL rise between controls and patients ($p > 0.05$). In hypertension situations, the mean LDL is 134.31 ± 29.24 mg/dl. The controls have an average LDL of 107 ± 13.1 mg/dl. Cases have a mean lower LDL than controls ($p < 0.001$). Hypertensive patients have a mean VLDL of 35.77 ± 13.43 mg/dl. The controls have a mean VLDL of 22.87 ± 3.46 mg/dl. Cases have a higher mean VLDL than controls ($p < 0.001$). The hypertension participants' mean TGL is 180.88 ± 68.5 mg/dl, while the control group's mean is 114.7 ± 17.62 mg/dl. In Table 4, the mean TGL of patients is larger than that of controls ($p < 0.001$). (Table - 4)

Discussion

Comorbid conditions such obesity, glucose intolerance, hyperinsulinemia, decreased HDL cholesterol; raised LDL cholesterol, elevated triglycerides, etc. affect more than 80% of individuals with hypertension. Two or more comorbidities are present in over 50% of hypertensive individuals.

The goal of the current study is to compare the lipid profile pattern of hypertension patients to that of controls. Serum magnesium and fasting blood sugar were also evaluated in this investigation to see if there was any difference between the patients and controls. Thirty control and fifty hypertensive cases made up the 80 cases that were examined. The two groups' blood pressure readings were noted. Serum magnesium, lipid profile, and blood glucose were measured in fasting blood samples.

Table 1 shows that, among 50 cases ranging in age from 30 to 71, the greatest proportion of cases fell into the 60–69 age range. Table 2 shows that of the

50 cases, there were 27 men and 23 females. Table 3 shows that SBP ($163.7 \text{ mmHg} \pm 16.72$) is greater than Controls' SBP ($114.66 \text{ mmHg} \pm 6.62$). There has been a considerable rise ($p < 0.05$). In many circumstances, the SBP rises with increasing age. Additionally, the DBP of hypertensive individuals is greater ($87.64 \text{ mmHg} \pm 17.85$) than that of the control group ($73.33 \pm 0.53 \text{ mmHg}$) ($p < 0.05$). The DBP stabilizes as age increases rather than rising. These findings imply that SBP, as opposed to DBP, is the better predictor in the elderly. It is advised that the DBP be decreased to 90 mmHg and the SBP to less than 140 mmHg. [10] Table 3 shows that the hypertension cases' Fasting Blood Glucose (FBS) is greater ($101.62 \text{ mg/dl} \pm 33.78$) than the controls' FBS ($82.46 \text{ mg/dl} \pm 10.8$). There has been a statistically significant rise ($p < 0.001$). However, 10% of the cases may have been diabetic patients, which could account for this increase. Even yet, hypertensive individuals have a propensity to have reduced glucose tolerance.

Table 3 shows that the hypertension cases' serum magnesium level ($1.94 \text{ mEq/L} \pm 0.05$) is marginally higher than the controls' ($1.92 \text{ mEq/L} \pm 0.08$). The higher incidence of hypomagnesaemia in women than in males is correlated with this rise, but it is not statistically significant ($p > 0.05$). Intra-erythrocyte magnesium concentrations were higher in the essential hypertension patients than in the healthy controls. Only serum magnesium and serum albumin concentration showed a positive correlation.

According to recent research, there is a significant rise in the prevalence of diabetes, insulin resistance, hypo magnesemia, hypercholesterolemia, and obesity among people with hypertension. These metabolic abnormalities may be inherited due to a genetic predisposition. A possible explanation for the higher occurrence of hypomagnesemia in women than in males could be mitochondrial inheritance via the maternal line.

Hypertension and dyslipidemia are associated with hypomagnesemia, which is caused by a mutation in mitochondrial tRNA. Maternal lineage members demonstrated a significant increase in urine fractional excretion of Mg^{+2} , which was primarily observed in hypomagnesaemia participants, indicating that poor renal Mg^{+2} reabsorption is the etiology of hypomagnesaemia in kindred syndrome. Despite normal serum calcium levels, examination of other urine electrolyte values revealed decreased urinary calcium on maternal lineage. Furthermore, there was no difference in the 24-hour urine sodium excretion between maternal and nonmaternal lineages, and hypokalemia was noted as a result of incorrect renal loss.

There is no link between the hypertension cases and controls' serum magnesium levels in the current investigation. Since serum proteins were

not estimated in this investigation, we were unable to determine any correlation with serum magnesium levels. Regarding Mg, there was also no difference in age or gender. Table 4 shows that the blood total cholesterol levels of hypertension subjects ($209 \text{ mg/dl} \pm 5.92$) are greater than those of the control group ($172.8 \text{ mg/dl} \pm 13.43$). A statistically substantial rise has occurred ($p < 0.001$). Despite being lower than controls ($42.94 \text{ mg/dl} \pm 3.56$), the serum HDL of hypertension subjects ($41.1 \text{ mg/dl} \pm 5.92$) is not statistically significant ($p > 0.05$). Compared to controls ($107 \text{ mg/dl} \pm 13.1$), hypertension subjects have higher serum LDL levels ($134.31 \text{ mg/dl} \pm 29.24$). A statistically substantial rise has occurred ($p < 0.001$). Hypertensive subjects have greater serum VLDL levels ($35.77 \text{ mg/dl} \pm 13.43$) compared to controls ($22.87 \text{ mg/dl} \pm 3.46$). A statistically substantial rise has occurred ($p < 0.001$). Hypertensive subjects have a greater serum TGL ($180.88 \text{ mg/dl} \pm 68.5$) than controls ($114.7 \text{ mg/dl} \pm 17.62$). A statistically substantial rise has occurred ($p < 0.001$). As a result, it has been noted that dyslipidemia occurs in hypertensive individuals while HDL concentration remains unchanged.

Essential hypertension is frequently accompanied by several metabolic disorders. Many hypertension patients had decreased HDL along with elevated plasma levels of LDL and VLDL, hypercholesterolemia, hypertriglyceridemia, and insulin resistance. A negative lipid and hemostatic profile is seen in men with hypertension who are between the ages of 50 and 59. Furthermore, β -blocker anti-hypertensive therapy is linked to decreased levels of HDL-related measures, while ACE inhibitor therapy appears to have a minor positive impact on total cholesterol and LDL-related metrics. Studies showing that dyslipidemia and hypertension are frequently linked to one another and that dyslipidaemic hypertension increases mortality when compared to either hypertension or dyslipidemia alone point to a significant clinical entity. The frequency of dyslipidaemic hypertension and its possible insulin resistance were compared with those of two other groups (hypertension only, dyslipidemia only). Research on the prevalence of dyslipidaemic hypertension in people with type 2 diabetes aged 7 to 17. The findings of the associations between serum lipid levels, ApoE genotypes and alleles, and stroke risk factors (diabetes, hypertension, etc.) indicated that ApoE4 is a separate risk factor linked to a changed lipid profile.

In those with cardiovascular and related illnesses, elevated GGT activities are independently linked to a more atherogenic lipid profile. [9] In order to prevent secondary stroke, it is vital to take into account two modifiable vascular risk factors: blood pressure and serum lipids. Furthermore, it is important to consider that the apoB, apoB/apoA-I

ratio, and apoA-I are highly predictive when assessing cardiac risk. The genetic locus connected to dyslipidemia that coexists with hypertension or diabetes may be the cause of the aberrant lipid metabolism in hypertension. This locus appears to be strongly linked to the insulin receptor and LDL receptor loci. The conventional units for hypertension and dyslipidemia ought to transform into worldwide cardiovascular risk management units in the future. [15]

Conclusion

As age groups increased, the systolic blood pressure became more relevant than the diastolic blood pressure. There are elevated levels of triglycerides, LDL, VLDL, and cholesterol; there is no discernible change in HDL or magnesium levels. When comparing hypertension cases to controls, fasting blood glucose is statistically significant; however, the significance may stem from the fact that 12% of the hypertensive patients also had diabetes.

According to the aforementioned study, dyslipidemia and hypertension are linked. This association may be caused by a genetic predisposition, secondary lifestyle choices, consumption of fatty foods, saturated fat, and cholesterol in food, which raise blood cholesterol levels. Saturated fat is the main culprit in this relationship. Drinking more booze and smoking.

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