eISSN: 09748369, www.biolmedonline.com

Study of lipid profile, serum magnesium and blood glucose in hypertension

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Abstract
An attempt was made to study the role of lipid profile serum Mg^{+2}, and blood glucose in hypertension individuals. Moreover, all the parameters are analyzed biochemically. In about 80 samples (50cases and 30 controls) and it is observed that dyslipidemia is seen in Hypertensive individuals with no change in HDL concentration. There is no correlation of serum magnesium in hypertensive cases with controls. It has been observed that serum magnesium of hypertensive cases is slightly higher than that of normal individuals. Fasting blood glucose of hypertensive cases (101.62mg/dl ±33.78) is higher than that of Controls (82.46 mg/dl±10.8). This increase is statistically significant ($p<0.001$). But this increase may be due to the presence 12% diabetic cases present in the cases. Even then, there is a tendency of developing impaired glucose tolerance in hypertensive subjects. The blood pressure is noted separately as systolic blood pressure and diastolic blood pressures. The systolic blood pressure was more significant than the diastolic blood pressure with increasing age groups.

Keywords: Hypertension, Serum Magnesium**,** Dyslipidemia.

Introduction

Hypertension is defined as a trait as opposed to a specific disease and represents a quantitative rather than a qualitative deviation from the normal. Blood pressure is the force that drives blood through blood vessels to supply oxygen and nutrients to the body's organs and tissues and carry away metabolites and waste materials. Blood pressure is optimal if the systolic blood pressure (SBP) is lower than 120mmHg and diastolic blood pressure (DBP) is less than 80mm Hg. Hypertension is defined as SBP level higher than 140mmHg and/or a DBP higher than 90mmHg. An elevated arterial blood pressure (chronic hypertension) is a common health problem worldwide and with ongoing global increase in the incidence. Approximately 25% of the adult populations are affected. Although historically defined as "an elevation of blood pressure" alone, hypertension is characterized by abnormalities of cardiac output, systemic vascular resistance, and arterial compliance (Giles *et al*., 2005).

As per the WHO Report 2002 in terms of burden of disease top 10 risks globally and regionally are alcohol consumption, high blood pressure, tobacco consumption, under weight, iron deficiency, unsafe water, high cholesterol and obesity, smoke from fuels, sanitation and hygiene (Chockalingam *et al*., 2006).Together these account for more than 1/3rd of all deaths worldwide. Pooling of epidemiological studies showed that hypertension is present in 25% (34 million) urban and 10% (31.5 million) rural subjects in India. 70% of these would be Stage-I hypertension.

Hypertension is classified into two groups – primary or essential hypertension and secondary hypertension. Primary hypertension is defined as a 'rise of blood pressure of unknown cause'. Secondary hypertension is the 'increase in blood pressure caused by diseases of kidney, endocrines, or some other organs'. Less than 5% of hypertensive patients develop malignant hypertension.

Hypertension is further graded into 3 stages based on the elevated blood pressures (Chalmer *et al*., 1999). A persistent and sustained high blood pressure has damaging effects on the heart (hypertensive cardiomyopathy), brain (cerebrovascular accident) and kidneys (benign and malignant hypertension).In people 50yrs or older systolic hypertension represents a greater risk (Whitworth *et al*., 2003). The differential impact of SBP and DBP on blood pressure staging in a representative sample of the middle-aged population in Spain, a population with a high incidence of cardiovascular disease.

The associations between blood pressure and the risks of stroke and CHD are well established. Similarly, there are also strong associations between serum cholesterol and risks of CHD. Large epidemiologic studies have demonstrated that subjects with hypertension have a marked increase in the prevalence of hypercholesterolemia, diabetes, hypomagnesemia, hypertriglyceridemia etc. The longitudinal study, examined risk in women across a wide spectrum of baseline BMI (kg/m2) values and studied waist circumference (WC, cm), percent body fat, fat mass (FM, kg) on incidence of hypertension in subgroup analyses. 592 women reported hypertension during a mean 16.7 years of follow-up. Higher BMI, even within the 'normal' range, was associated with greater risk of hypertension (Shugeri *et al*., 2008).

This prompted the present study of lipid profile in hypertension patients. The study included the estimation of magnesium considering the pivotal role of Mg in various metabolic reactions especially those involving cellular energy ATP. Mg is the fourth most abundant cation in the human body and the second most abundant intracellular cation. The average amount of body Mg in an adult weighing 70Kg is about 2,000 mEq. Of this approximately 50-70% is in bones, 1% in ECF and the remaining is intracellular, where it is concentrated mainly in the mitochondria (Sanders *et al*., 1999). Blood glucose levels in hypertensive cases are also included to find out the association of hyperglycemia with hypertension. The reasons for increased rate of hypertension include life style changes, sugar rich diet, high fat processed foods and sedentary behavior (Kearney *et al*., 2005). 'Dyslipidemia' is seen among the common metabolic diseases. Lipoprotein disorders or hyperlipidaemia may result from a primary abnormality in lipid metabolism or is a secondary manifestation of some other condition. The prevalence of dyslipidemia in a population of youth (7-17yrs) with type2DM and examined the relationship between lipid parameters and other known cardiovascular risk factors. Elevated apoB levels with normal LDL-C levels highlight the importance of full lipid panel including apo-B in defining potential modifiable cardiovascular risk in population having high rates of obesity, smoking, and poor glycemic control (Sellers *et al*., 2007).

Materials and Methods

The study was carried out in Department of Biochemistry, Central Laboratories, GSL Medical College and General Hospital, Rajahmundry. Two groups were included in the study – Cases and Controls. 50 hypertensive subjects (27 males and 23 females) were taken up for the study who attended the department of General Medicine with the age group 30 – 71 years. Secondary hypertensive cases were excluded in the study. A total of 30 age and sex matched subjects who attended as attendants with no history of diabetes, hypertension, cardiac or renal diseases were included. Blood pressure was measured in all subjects as per the recommendations of JNC –VII (Chobanian *et al*., 2003). All the blood samples are collected form the individual in fasting blood samples with out anti coagulant and centrifuge at 1500 rpm for 5 min and serum is collected in fresh vial for biochemical studies by using standard methods as follows. The data was analyzed by SPSS software version 1.4.

Serum magnesium : Calmagite method (Gindler *et al*., 1971)
Serum total cholesterol : Cholesterol oxidase Method (Richmond, 1973)
Triglycerides : Glycerolkinase, Peroxidase, method (Foosati *et al*., 1982)
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

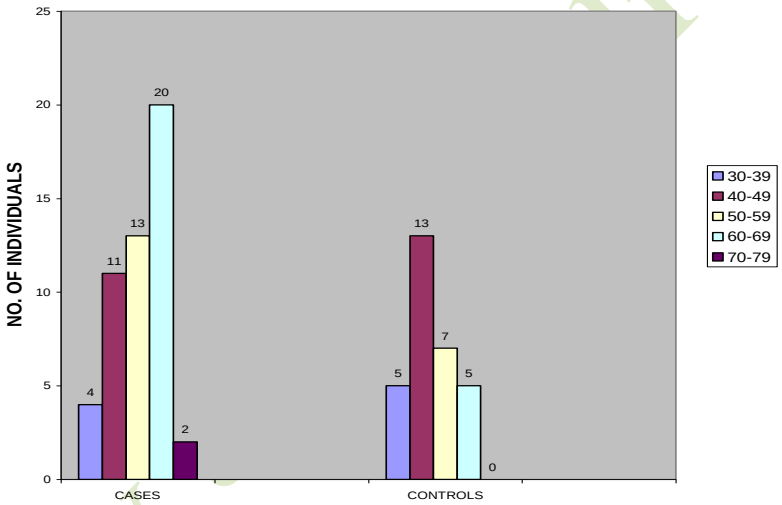
Results

80 subjects comprising of 50 hypertensive cases and 30 controls were included in the present study. Measurement of blood pressure, lipid profile, fasting blood glucose and serum magnesium were done in both the groups. The result of the study is given below.

Table 1

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

FIG-1: AGE-WISE DISTRIBUTION IN CASES AND CONTROLS

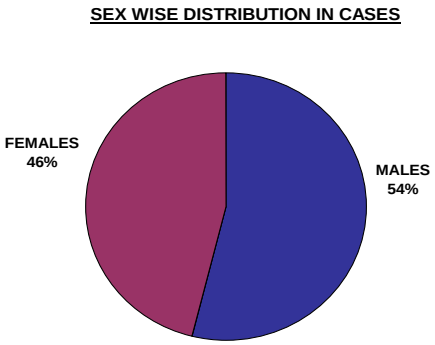


The hypertensive cases are in the age group of 30-71 years. The mean age is 55±10.61 years whereas the controls in age group of 31- 69 years is 48.53±9.58 years (table-1; fig: 1).

Table 2

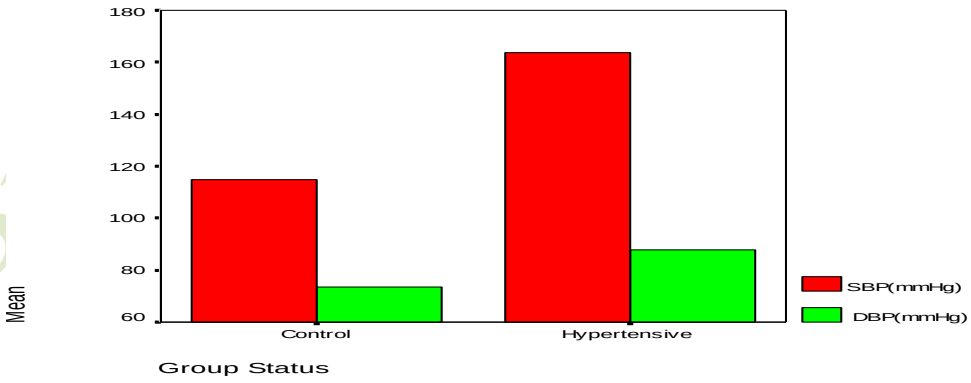
Group	Males		Females		Total
	Number	%	Number	%	
Cases	27	54	23	46	50
Controls	18	36	12	24	30

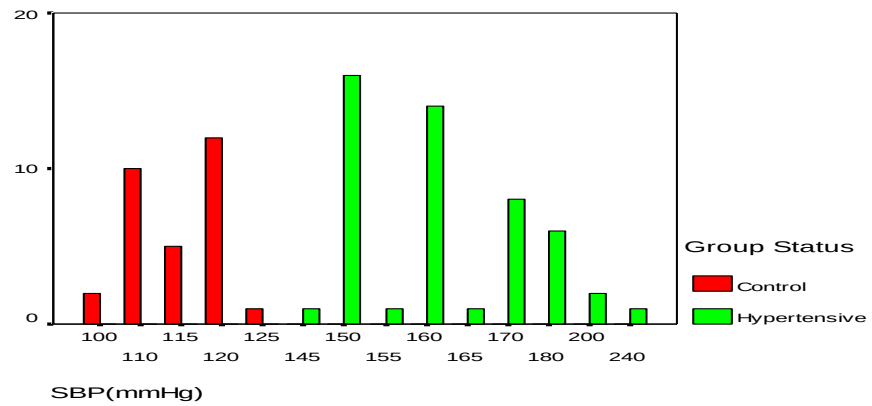
Fig 2



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; fig: 2).

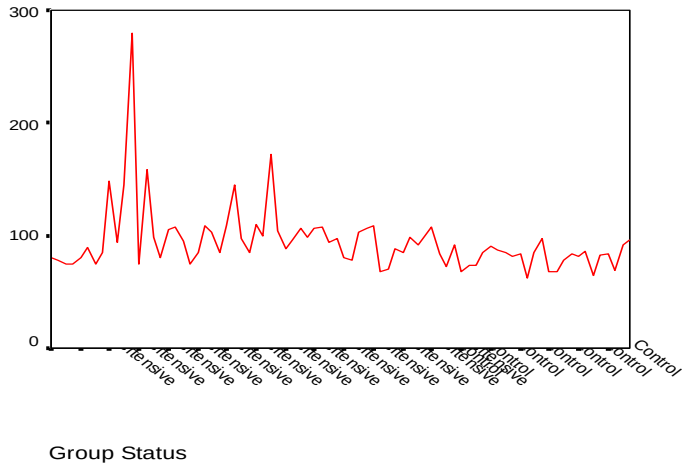
Group Status		SBP (mmHg)	DBP (mmHg)	FBS (mg/dl)	Mg (mg/dl)
Case (n = 50)	Mean	163.7	87.64	101.62	1.94
	SD	16.72	17.85	33.78	0.05
Control (n = 30)	Mean	114.66	73.33	82.46	1.92
	SD	6.62	0.53	10.8	0.08





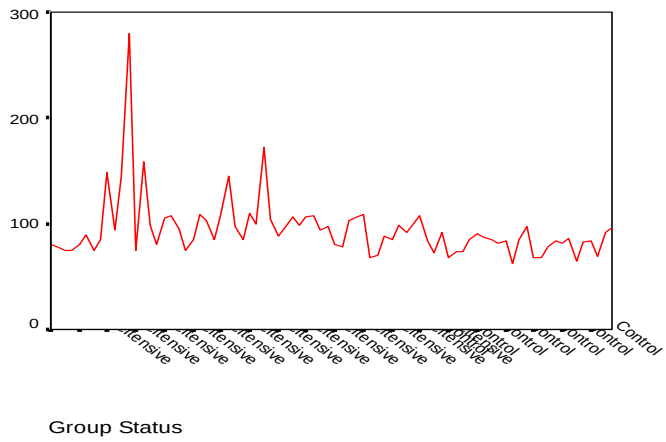
The blood pressure is calculated separately as systolic blood pressure and diastolic blood pressure. The mean SBP of the hypertensive subjects is 163.7±16.72 mmHg and that of the controls is 114.66±6.62 mmHg. The mean of the SBP is higher in hypertensives subjects than controls ($p<0.001$). The mean DBP of hypertensive cases is 87.64±17.85 mmHg and that of the controls is 73.33±0.53 mmHg. The mean of cases is higher than controls ($p<0.05$) (table-3; fig: 3).

Fig 4



The mean fasting blood glucose of hypertensive subjects is 101.62±33.78 mg/dl. The mean fasting blood glucose of controls is 82.46±10.8 mg/dl. The mean of hypertensive subjects is higher than controls ($p<0.05$). However, the increase may be due to the diabetic cases (10%) present in hypertensive subjects. (Table-3; fig: 4)

Fig 5

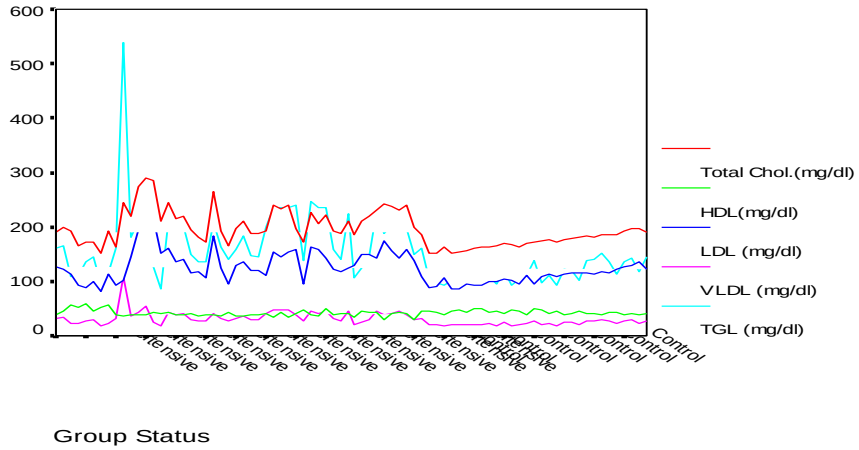


The mean serum magnesium of hypertensive subjects is 1.94 ± 0.05 mEq/L and that of the controls is 1.92±0.08 mEq/L. The mean of hypertensive cases is higher than controls but the increase is not statistically significant (p>0.05) (table-3; fig: 5).

Table 4

Group Status		TC (mg/dl)	HDL (mg/dl)	LDL (mg/dl)	VLDL (mg/dl)	TGL (mg/dl)
Case (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

Fig 6



The mean total cholesterol in hypertensive cases is 209±31.63 mg/dl. The mean total cholesterol of controls is 172.8±13.43 mg/dl. The mean of hypertensive cases is higher than controls ($p<0.001$). The mean HDL of hypertensive cases is 41.1±5.92 mg/dl and that of controls is 42.94±3.56 mg/dl. The increase in mean of HDL in controls than cases is not statistically significant ($p>0.05$). The mean LDL of hypertensive cases is 134.31±29.24 mg/dl. The mean LDL of controls is 107±13.1 mg/dl. The mean LDL of cases is higher than controls ($p<0.001$). The mean VLDL of hypertensive subjects is 35.77±13.43 mg/dl. The mean VLDL of controls is 22.87±3.46 mg/dl. The mean VLDL of cases is higher than controls ($p<0.001$). The mean TGL of hypertensive subjects is 180.88±68.5 mg/dl and that of controls is 114.7±17.62 mg/dl. The mean TGL of cases is higher than controls ($p<0.001$) (Table-4; fig: 6).

Discussion

More than 80% of people with hypertension have additional comorbidities, such as obesity, glucose intolerance, hyperinsulinemia, reduced HDL cholesterol, elevated LDL cholesterol, elevated triglycerides etc. More than 50% of people with hypertension have two or more comorbidities. The present study is done to study the pattern of lipid profile in hypertensive patients compared to the controls. In the present study, we also measured serum magnesium and fasting blood sugar to check whether there is any significance in cases compared to controls. 80 cases were studied including 50 hypertensive cases and 30 controls. The blood pressures of the two groups were recorded. Fasting blood samples were taken for the estimation of blood glucose, lipid profile and serum magnesium.

In table-1, it has been observed that out of 50 cases aged 30-71 years maximum number of cases was between age group of 60-69 years. In table 2, it has been observed that out of 50 cases 27 were males and 23 were females. In table 3, it has been observed that SBP (163.7mmHg ± 16.72) is higher than that of Controls (114.66 mg/dl±6.62). This increase is significant ($p<0.05$). The SBP is increased as the age progresses in the cases While the DBP in hypertensive subjects (87.64mmHg±17.85) is also higher than the controls (73.33±0.53mmHg) ($p<0.05$). The DBP is not increased as the age increased, it is stabilized. These observations suggest that SBP is the best predictor in elderly than DBP (Franklin *et al*., 2001). It is recommended that SBP needs to be lowered below 140mmHg and the DBP below 90mmHg (Moncia *et al*., 2007). In table 3, it has been observed that Fasting Blood Glucose (FBS) of hypertensive cases (101.62mg/dl ±33.78) is higher than that of Controls (82.46 mg/dl±10.8). This increase is statistically significant ($p<0.001$).But this increase may be due to the presence 10% diabetic cases present in the cases. Even then, there is a tendency of developing impaired glucose tolerance in hypertensive subjects.

From the table 3, it has been observed that serum magnesium of hypertensive cases (1.94mEq/L ±0.05) is slightly higher than that of Controls (1.92mEq/L±0.08). However, this increase is not statistically significant ($p>0.05$) which correlates with the observed higher incidence of hypomagnesaemia in women compared to men. The patients with essential hypertension exhibited higher intra-erythrocyte Mg concentrations than the healthy controls. The serum albumin concentration positively correlated only with serum Mg. Recent studies had shown that subjects with hypertension have a marked increase in the prevalence of hypercholesterolemia, hypertriglyceridemia, hypomagnesaemia, diabetes, insulin resistance, and obesity. Genetic predisposition may be responsible for the inheritance of these metabolic disorders. Mitochondrial inheritance through the maternal lineage may be responsible for the incidence of hypomagnesemia in women than men. A mutation in mitochondrial tRNA is the cause for the hypomagnesemia linked with hypertension and dyslipidemia. Members of the maternal lineage showed a marked increase in the urinary fractional excretion of Mg^{+2} (mostly seen among subjects with hypomagnesaemia)establishing impaired renal Mg^{+2} reabsorption as the cause of hypomagnesaemia in kindred syndrome. Evaluation of other urinary electrolytes had shown reduced urinary calcium on maternal lineage despite normal serum Ca levels. In addition, hypokalemia was observed due to inappropriate renal loss and no difference in 24-hour urinary sodium excretion between maternal and nonmaternal lineages.

In the present study, there is no correlation of serum magnesium in hypertensive cases with controls. As we did not estimate serum proteins in our study, so we could not have found any relation with serum Mg. There was also no gender difference or age related to Mg. From the

table 4, it has been observed that serum total cholesterol of hypertensive cases (209mg/dl±5.92) is higher than that of controls (172.8mg/dl±13.43). The increase is statistically significant (p<0.001). The serum HDL of hypertensive cases (41.1mg/dl ± 5.92) is less than controls (42.94mg/dl ± 3.56) but it is not statistically significant (p>0.05). The serum LDL of hypertensive cases (134.31mg/dl±29.24)is higher than controls (107mg/dl±13.1).The increase is statistically significant (p<0.001). The serum VLDL of hypertensive cases (35.77mg/dl±13.43)is higher than controls (22.87mg/dl±3.46). The increase is statistically significant (p<0.001). The serum TGL of hypertensive cases (180.88mg/dl ± 68.5) is higher than controls (114.7mg/dl±17.62). The increase is statistically significant (p<0.001). Therefore, it is observed that dyslipidemia is seen in hypertensive subjects with no change in HDL concentration.

Multiple metabolic abnormalities often accompany essential hypertension. Decreased HDL together with increased plasma levels of LDL and VLDL, as well as hypertriglyceridemia, hypercholesterolemia, and insulin resistance, were found in many hypertensive patients. Unfavorable lipid and hemostatic profile is observed in hypertensive men aged 50-59 years. In addition, anti-hypertensive treatment with β-blockers is associated with lower levels of HDL- related parameters, whereas treatment with ACE inhibitors appears to exert a small beneficial effect on total cholesterol and LDL-related parameters. The studies regarding hypertension commonly associated with dyslipidemia and that dyslipidaemic hypertension increased mortality compared with hypertension only and dyslipidemia only, suggesting an important clinical entity. The prevalence and compared the potential insulin resistance of dyslipidaemic hypertension with two other groups (hypertension only, dyslipidemia only). The studies regarding the prevalence of dyslipidaemic hypertension in 7-17 years population with type-2 DM. The relationships among serum lipid levels, ApoE alleles and genotypes, and stroke risk factors (hypertension, diabetes etc) the results concluded that ApoE4 is an independent risk factor associated with an altered lipid profile.

Increased GGT activities are independently associated with a more atherogenic lipid profile in population having cardiovascular and its related disorders (Lippi *et al*., 2007). Thus, blood pressure and serum lipids are two important and modifiable vascular risk factors that should be taken into consideration for the prevention of secondary stroke. In addition, the apoB, apoB/apoA-I ratio and apoA-I should be regarded as highly predictive in evaluation of cardiac risk. The reason behind the abnormal lipid metabolism in hypertension may be the genetic locus associated with dyslipidemia accompanying hypertension or diabetes seems to be closely linked to the LDL receptor and insulin receptor locus. In future, the traditional hypertension and dyslipidemia units should probably evolve into global cardiovascular risk management units (Tunon *et al*., 2007).

Master chart of cases

S No	SBP (mmHg)	DB (mmHG)	FBS (mg/dl)	Mg (mg/dl)	TC (mg/d)	HDL (mg/dl)	LDL (mg/dl)	VLDL (mg/dl)	TGL (mg/dl)
1	150	90	80	1.92	190	38	126	32	60
2	160	100	78	1.96	200	45	122	33	165
3	180	100	75	1.8	192	56	114.2	21.8	109
4	160	90	75	1.9	165	51	92	22	110
5	160	90	80	1.9	173	58	88	27	135
6	200	130	89	1.9	173	45	98.5	29.2	146
7	180	80	75	1.9	151	51	80.8	19.2	96
8	150	80	85	1.9	192	56	113.6	23	115
9	150	90	148	2	162	38	92	32	160
10	160	100	94	2	245	36	101	108	540
11	240	140	145	2.1	220	38	146	36	180
12	170	100	280	1.92	275	38	195	42	210
13	150	96	75	1.96	290	39	196.2	54.8	274

14	180	100	158	2	285	44	215.6	25.4	127
15	160	100	98	1.92	210	41	151.6	17.4	87
16	150	100	80	1.92	245	42	161	42	210
17	170	100	105	1.94	215	39	136.4	39.6	198
18	160	96	108	1.92	219	38	141	40	201
19	150	70	95	1.92	195	40	115	30	150
20	170	100	75	1.9	180	36	117	27	135
21	150	95	85	1.92	172	38	107	27	135
22	170	100	109	2.2	264	39	183.4	41.6	208
23	150	90	103	1.96	192	36	123.5	32.5	163
24	160	100	85	1.92	166	43	95	28	140
25	180	90	109	1.96	197	36	129.4	31.6	158
26	200	110	145	1.92	210	37	136.4	36.6	183
27	150	100	97	1.93	188	39	119.4	29.6	148
28	150	90	85	1.92	187	38	120	29	145
29	160	60	110	1.96	193	41	112	40	200
30	150	100	100	1.92	239	34	154.2	47.8	236
31	150	70	172	1.92	234	44	145.2	46.8	236
32	170	80	104	1.96	239	34	154.2	47.8	236
33	170	100	88	1.9	198	41	158.4	39.6	239
34	160	60	96	1.9	171	47	96.2	27.8	139
35	180	100	106	1.94	226	38	162.8	45.2	246
36	150	100	99	1.92	206	37	157.8	41.2	236
37	160	70	106	1.94	221	49	142.8	44.2	236
38	160	60	107	1.94	192	38	122.2	31.8	158
39	150	70	94	1.92	187	41	118	28	140
40	150	60	97	1.9	210	40	125	45	225
41	145	60	80	1.98	185	35	128.5	21.5	106
42	150	80	78	1.96	210	45	150.2	24.8	124
43	160	60	103	1.94	220	42	149	29	145
44	170	70	106	1.94	232	44	142.4	45.6	228
45	160	60	109	1.92	242	30	174.6	37.4	187
46	155	65	68	2	238	40	157.2	40.8	204
47	165	80	70	1.92	232	44	142.4	45.6	228
48	180	80	88	1.94	239	41	158.4	39.6	198
49	170	70	85	1.98	199	30	139.2	29.8	149
50	160	100	99	1.94	185	45	108	32	160

Master chart of controls

S No	SBP (mmHg)	DBP (mmHg)	FBS (mg/dl)	Mg (mg/dl)	TC (mg/dl)	HDL (mg/dl)	LDL (mg/dl)	VLDL (mg/dl)	TGL (mg/dl)
1	110	70	92	1.9	152	45	88	21	105
2	120	80	100	1.9	152	42	90.4	19.6	98
3	110	70	107	1.9	162	38	105.8	18.2	93
4	120	80	84	1.9	152	46	85.4	20.6	103
5	110	70	72	1.9	153	47	85	21	105
6	115	80	92	1.9	156	42	94.2	19.8	99

7	120	70	68	1.9	161	49	91.8	20.2	101
8	110	75	74	2	162	49.2	92.2	20.6	103
9	120	80	74	1.8	164	42	100.4	21.6	108
10	110	70	85	1.9	165	46	99.8	19.2	96
11	110	75	90	2	169	41	104.2	23.8	119
12	115	80	87	1.9	168	47	102.6	18.4	92
13	120	80	85	2	163	46	96	21	105
14	110	70	81	1.9	170	38	110	22	110
15	120	75	84	1.9	172	49	95.4	27.5	139
16	120	70	62	1.8	175	47	108.4	19.6	98
17	110	70	85	1.9	176	41	112.6	22.4	112
18	100	60	97	1.9	173	46	108.4	18.6	93
19	115	65	68	1.9	177	39	113.6	24.4	122
20	120	80	68	2	179	40	115	24	120
21	120	80	78	2	182	45	116.4	20.6	103
22	115	70	84	2.1	184	41	115.4	27.6	138
23	125	85	82	1.9	181	40	113	28	140
24	120	60	86	2.2	186	38	116.8	30.2	151
25	120	80	65	1.8	186	44	115	27	135
26	115	75	83	1.9	186	42	121.4	22.6	113
27	110	70	84	2	192	39	126	27	135
28	120	80	69	2	198	41	128.6	28.4	142
29	110	70	92	1.8	197	38	135.6	23.4	117
30	100	60	96	1.9	191	40	123	28	146

Conclusion

The systolic blood pressure was more significant than the diastolic blood pressure with increasing age groups. Elevated levels of cholesterol, LDL, VLDL, triglycerides are observed and no significance in HDL and magnesium is seen. Fasting blood glucose is statically significant in hypertensive cases when compared to controls but the significance may be due to the presence of 12% diabetic cases among the hypertensive patients. From the above study that dyslipidemia is associated with hypertension is associated with hypertension this is may due to the genetic predisposition, secondary life styles, fatty food consumption, saturated fat, cholesterol in the food increase the blood cholesterol and saturated fat is the main culprit, Smoking and increased alcohol intake.

Acknowledgement

Authors express their sincere gratitude for giving support to publish this paper to Dr. N. Ammanna Pantulu, Professor and Head, Department of Biochemistry and to Professor and HOD of Department of General Medicine, GSL Medical College, Rajahmundry, East Godavari district, Andhra Pradesh, India.

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