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Full Length Research Paper

Clinical diagnostic indices of hypertensive patients

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The aim of the study was to investigate the efficacy of the use of some clinical indices as diagnostic parameters of hypertension. Twenty (20) healthy, normotensive human subjects and twenty (20) hypertensive patients were subjected to diastolic (D) and systolic (S) blood pressure, body mass index (BMI), serum creatinine (C), serum magnesium (Mg^{2+}), serum sodium (Na^{+}), serum total cholesterol (Ch), serum triglyceride (T), aspartate aminotransferase (AST) and fasting blood sugar (FBS) analyses. The experimental design is a single factor completely randomized design (CRD). Results recorded of the healthy human subjects and hypertensive patients, expressed as mean ± standard error (S.E) (unit) were as follows : (D) 75.6±1.7 and 98.11±0.9 (mmHg), (S) 120.52 ± 9.14 and 156.87 ± 11.23 (mmHg), (BMI) 15.3±0.7 and 27.01±1.7(kg/m^2), (C)1.2 ± 0.1and 1.8 ± 0.2 (mg/dl), (Mg^{2+}) 2.4 ± 0.1 and 1.11±0.63 (mm/L), (Na^{+})138 ± 1.16 and 149± 0.23, (Ch) 175±1.2 and 225±8.1(mg/dl), (T) 76±1.3 and 125±2.1(mg/dl), (AST) 12.18 ± 1.16 and 22.5 ± 2.16 (U/l), (FBS) 82.18 ± 9.16 and 98.87± 14.23 (mg/dl). The mean values of D, S, BMI, Na^{+}, Ch, T, AST and FBS were significantly higher ($p<0.05$) : but those of Mg^{2+} were significantly lower ($p<0.01$) in hypertensive patients compared with healthy human subjects. Incidence of hypertension correlated positively and significantly ($p<0.05$) with significant increase ($p<0.05$) in S, D, BMI, FBS, AST, Ch, T, and Na^{+}; and significant decrease ($p<0.01$) in Mg^{2+}. The statistical regression and correlation between serum total cholesterol and systolic blood pressure of hypertensive patients were significant ($p<0.05$), r = 0.975. Observed values of systolic blood pressure could be used with high precision to predict serum total cholesterol levels of hypertensive patients.

Key words: Normotensive, hypertension, diastolic, body mass index, high blood pressure.

INTRODUCTION

Hypertension is a chronic disease characterized by elevation of blood pressure. Hypertension is classified by cause as either essential or secondary. Essential hypertension indicates that no specific medical cause could be implicated as causative agent of the hypertension (Carretero and Oparil, 2000). About 90-95% medical cases of hypertension is essential hypertension (Oparil *et al*., 2003). Secondary hypertension is a result of a specific underlying condition with a well-known mechanism, such as chronic kidney disease, narrowing of the aorta or kidney arteries, or endocrine disorders such as excess aldosterone, cortisol, or catecholamines (Pierdomenico *et al*., 2009).

Monogenic forms of hypertension caused by Mendelian forms of high blood pressure are as a result of single gene mutations about which ten have been identified (Lifton *et al*., 2001). These mutations affect blood pressure by altering renal salt handling, and are inheritable (Guyton, 1991). Excess activity of the sympathetic

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sympathetic nervous system increases blood pressure and contributes to hypertension (Somers *et al*., 1993). Hypertensive patients manifest greater vasoconstrictor responses to infused norepinephrine than normotensive controls (Ziegler *et al*., 1991).

The renin–angiotensin–aldosterone system is another system that maintains the extracellular fluid volume, peripheral resistance, and if disturbed may lead to hypertension (Fujino *et al*., 2004). Obesity is a risk factor for hypertension because of activation of the renin-angiotensin system (RAS) in adipose tissue. Renin-angiotensin system has been linked with insulin resistance (Segura and Ruilope, 2007).

Local nitric oxide and endothelin are secreted by the endothelium, and are the major regulators of vascular tone and blood pressure. The balance between the vasodilators and the vasoconstrictors is upset, in patients with essential hypertension which leads to changes in the endothelium and sets up a “vicious cycle” that contributes to the maintenance of high blood pressure (Cai and Harrison, 2000).

Renovascular hypertension (RVH) is a leading cause of potentially curable hypertension. Although RVH affects less than 1% of the unselected hypertensive population, between 10% and 35% of properly screened patients referred to specialised centres for problematic hypertension may prove to have renovascular disease (Jensen, 1995).

Hypertension can affect eyesight and lead to eye disease causing damage to the blood vessels in the retina, the area at the back of the eye where images focus. This eye disease is known as hypertensive retinopathy (Kozarsky, 2014).

Incident hypertension increases markedly with age, blood pressure level and body mass index. Diastolic blood pressure (DBP), serum potassium and creatinine concentrations have been used in predicting renovascular hypertension (Anderson *et al*., 1988). The kidney is one of the principal target organs of hypertension, and most renal diseases are associated with elevated blood pressure (Chasis and Baldwin,1966). Elevated serum creatinine concentration has frequently been used as a criterion for renal morbidity in clinical trials of hypertension (Amery *et al*., 1985). Shown in table 1 are the blood pressure indicators of stages of hypertension.

Hypercholesterolemia and hypertension were correlated significantly ($p<0.05$) with an increased hazard ratio (HR) of fatal cardiovascular disease (Yang *et al*., 2007). Several epidemiological studies have revealed a close relationship between abnormalities of the lipid metabolism and arterial hypertension (Ferrara *et al*., 2002). Total and non-HDL cholesterol levels and triglyceride levels increased significantly with increasing systolic or diastolic blood pressure in a study carried out with adult 8,081 and 7663 adult men and women. Respectively. The correlation between blood pressure and total cholesterol level, decreased with age, in men but increased with age, in women (Bonaa and Thelle, 1991). Human subjects with high blood pressure often have the metabolic syndrome characterized by an aggregation of abnormalities in glucose and lipid metabolism (Jeppesen *et al*., 2000). Hypercholesterolaemia, hypertriglyceridaemia and increase in low density lipoprotein are the main lipid abnormalities in the incidence of hypertension in the study area of Northern Bangladesh (Saha *et al*., 2006).

Fasting blood glucose was significantly raised ($p \leq 0.05$) in 55% of hypertensive individuals in a study conducted on patients of the Liaquat University Hospital Hyderabad (Shaikh *et al*., 2012).

Among patients with hypertension, increasing body mass index (BMI) was a significant predictor of isolated diastolic hypertension (IDH), and systodiastolic hypertension (SDH) (Chirinos *et al*., 2009). The correlation of BMI with high blood pressure and abnormal lipids were statistically significant ($p<0.05$) in an adult population in which the relationship between body mass index (BMI) and blood pressure, cholesterol, high-density lipoprotein-cholesterol (HDL-C), and hypertension and dyslipidemia were evaluated (Brown *et al*., 2000). Mean blood pressure levels increased with increasing BMI and the risk of hypertension was significantly higher ($p<0.05$) among population groups with overweight and obesity compared with normal human subjects, in a study conducted in three populations across Africa and Asia viz : Ethiopia, Vietnam and Indonesia (Tesfaye *et al*., 2007).

Electrolyte tests are commonly used as diagnostic indices to monitor treatment of certain problems, including high blood pressure (hypertension), heart failure, liver and kidney disease and diabetes. Significant negative correlations were found between serum potassium levels and systolic BP and diastolic BP of patients suffering from hypertension (Pikilidou *et al*., 2007).

Uncomplicated hypertension could be treated with thiazide diuretic either alone or combined with drugs from other classes. Other antihypertensive drug classes are angiotensin-converting enzyme inhibitors, angiotensin-receptor blockers, beta-blockers, and calcium channel blockers. Two or more antihypertensive medications will be required to achieve goal BP (<140/90 mm Hg, or <130/80 mm Hg) for patients with diabetes and chronic kidney disease. for patients whose BP is more than 20 mm Hg above the systolic BP goal or more than 10 mm Hg above the diastolic BP goal, initiation of therapy using two agents, one of which usually will be a thiazide diuretic, is considered (Chobanian *et al*., 2003).

A significantly greater lowering of diastolic blood pressure (12.4 mm Hg) was achieved in an atenolol administered group of hypertensive patients compared with a low sodium/high potassium administered diet group of hypertensive patients (7.9 mm Hg, $p = 0.001$) and a group of hypertensive patients who were made to lose

Table 1. Blood pressure indicators of stages of hypertension.

Hypertension stages	Systolic (BP mmHg)	Diastolic (BP mmHg)
Pre-hypertension	120-139	80-89
Stage 1 hypertension	140-159	90-99
Stage 2 hypertension	≥160	≥100

(Chobanian *et al*., 2003).

weight (Langford *et al*., 1991). Systolic pressure is a more important independent cardiovascular risk factor than diastolic pressure in hypertensive subjects over 50 years of age. Supine systolic pressure of hypertensive patients decreased to a greater extent with lacidipine and enalapril drugs compared with nitrendipine drug, the difference between lacidipine and nitrendipine reaching statistical significance. A decrease in systolic blood pressure may be produced by vasodilators through a reduction in peripheral resistance with or without an active change in arterial compliance. (Chaignon *et al*., 1993).

Cytomegalovirus (CMV), an infection affecting between 60 and 99 percent of adults worldwide, can cause high blood pressure (Moskowitz, 2009). A direct relationship between the levels of subgingival periodontal bacteria and both SBP and DBP as well as hypertension prevalence was observed by Desvarieux (2010). Toll-like activation of non-parenchymal liver cells by pathogens results in portal hypertension (Steib *et al*., 2015).

The aim of the study was to investigate the efficacy of the use of the clinical indices: diastolic and systolic blood pressure, body mass index (BMI), serum creatinine (C), serum magnesium (Mg^{2+}), serum sodium (Na^{+}), serum cholesterol (Ch), serum triglyceride(T), aspartate aminotransferase (AST) and fasting blood sugar (FBS) as diagnostic parameters of hypertension.

MATERIALS AND METHODS

Experimental Design

The experimental design used in the present study is a single factor completely randomized design (CRD) whose linear equation is $\hat{Y} = \mu + Ti + Єij$

$\hat{Y}$ = individual observation
μ = overall mean
Ti = ith type of disease, and is significant of hypertension.
Єij = error which is independently, randomly and normally distributed with zero mean and constant variance.

SPSS for windows (version 17.0, SPSS, Chicago, IL, USA) was used to perform the statistical analyses. The significance levels were $p<0.05$ and $p<0.01$.

The research was given Ethical approval from the Depart Department of Biochemistry, School of Biological Sciences, Federal University of Technology, Owerri, because it was carried out in compliance with the Declaration on the Right of the Patient (WMA, 2000).

Selection of human subjects

Twenty (n = 20) clinically confirmed hypertensive male patients, of age bracket 50-70 years, who had been irregular in their oral administration of anti-hypertensive drugs, and twenty normal (normotensive), healthy human subjects (n=20) of the same age bracket, voluntarily participated in this study, at Federal Medical Centre Owerri, Imo State, Nigeria. The subjects were randomly selected between September and October 2014. Exclusion criteria included: respiratory tract infection, protein energy malnutrition, smoking, alcoholism, persons living with HIV, and malaria patients.

Determination of blood pressure

An electronic sphygmomanometer (DM 3000, Kawamoto Corporation, Osaka Japan, digital blood pressure monitor), was used in measuring systolic and diastolic blood pressures of the hypertensive patients and normal human subjects.

Measurement of body mass index (BMI)

Weight and height of human subjects were measured using the weight watchers ultimate precision electronic scale and meter rule. The BMI was calculated as

$$\frac{\text{weight}}{\text{height}^2}\left(\frac{\text{kg}}{\text{m}^2}\right)$$

Blood was obtained by veni-puncture carried out by a Phlebotomist nurse. The method described by Thavasu *et al*. (1992) was used in obtaining the serum. Whole blood was collected in a covered test tube, and allowed to clot by leaving it undisturbed for 15-30 minutes at room temperature. The clot was removed by centrifuging at 1,000-2,000 x g for 10 minutes in a refrigerated centrifuge, to obtain the blood serum.

In Vitro Quantitative Determination of Serum Fasting Blood Sugar (FBS)

Serum was obtained from patient/individual who had not

taken any victual (food or drink, except water), for an 8-hour period. Glucose oxidase catalyses the oxidative transformation of β D-glucose present in the serum to D glucono -1 ,5 - lactone with the formation of hydrogen peroxide. The lactone is slowly hydrolysed to D-gluconic acid. The hydrogen peroxide produced is broken down to oxygen and water by a peroxidase enzyme. Oxygen reacts with ortho-toluidine to produce a coloured complex, the intensity of which is proportional to the concentration of the D-glucose in the serum, and measurable at 540nm.

In Vitro Quantitative Determination of Serum creatinine (C)

Creatinine amidohydrolase was used to measure serum creatinine in a totally enzymatic procedure. Creatine, produced by hydrolysis, was acted upon by creatine kinase, and then by pyruvate kinase and lactate dehydrogenase, to result in a change in absorbance at 340 nm. The amount of creatinine present was related to the rate of change in A340 and was determined from a standard curve (Moss *et al*., 1975).

Lipid Profile Assays

Serum total cholesterol (Ch), and serum triacylglycerol/triglyceride (TG) were determined using commercial kits (Randox Laboratories Ltd., UK), in conformity with the methods employed by Ibegbulem and Chikezie (2012); Chikezie and Okpara (2013).

In Vitro Quantitative Analysis of Aspartate Amino Transferase (AST)

In vitro quantitative determination of serum aspartate amino transferase (AST) was carried out using the method employed by Reitman and Frankel (1957). The test based on the reaction in which l-aspartate and α-ketoglutarate are converted to l-glutamate and oxaloacetate by the catalytic activity of AST. The oxaloacetate forms a complex known as oxaloacetate hydrazone with 2,4-dinitrophenyl hydrazine. The intensity of the colour of the hydrazone, measurable with a colorimeter at 578nm, is directly proportional to the AST enzyme activity.

In Vitro Quantitative Determination of Serum Electrolytes

Serum electrolytes were determined consistent with methods described by NKF (2002) and Shenqi *et al*. (2013). The serum electrolytes were analyzed using an auto analyzer (Hitachi 7600 analyzer, Hitachi, Japan). The inter- and intra-assay coefficients of variation for Na^+ and Mg^{2+} were 0.77% and 1.13%; and 1.15% and 1.92% respectively.

RESULTS

Results on blood pressure and body mass index (BMI) shown in Table 2 indicate that mean values of systolic blood pressure, diastolic blood pressure and body mass index were significantly higher ($p<0.05$) in hypertensive patients compared with healthy human subjects.

Table 3 shows results on the biochemical indices: Fasting blood sugar, Serum creatinine, and serum aspartate aminotransferase (AST) of the hypertensive patients, and healthy human subjects. Mean values of fasting blood sugar and serum aspartate aminotransferase were significantly different ($p<0.05$) in order of consecutive decrease as listed : hypertensive patients, healthy human subjects. Mean values of serum creatinine were numerically higher, but with no recorded significance difference ($p<0.05$) in hypertensive patients compared with healthy human subjects.

Results on the serum electrolytes : serum sodium (Na^+), and serum magnesium (Mg^{2+}) of the hypertensive patients, and healthy human subjects are shown in Table 4, and indicate that mean values of serum sodium were significantly higher ($p<0.05$) in hypertensive patients in comparison with healthy human subjects. Conversely, the mean values of serum magnesium were significantly lower ($p<0.01$) in hypertensive patients compared with healthy human subjects.

Results on the serum lipids : total cholesterol and triglyceride, shown in Figure 1, observed of the human subjects, indicate a difference in mean values of both diagnostic indices, in the order of consecutive significant decrease ($p<0.05$) as follows : hypertensive patients, healthy human subjects.

DISCUSSION

Systolic and diastolic blood pressure(s) were significantly higher ($p<0.05$) in hypertensive patients compared with healthy human subjects, consistent with the findings in a study of differences in blood pressure profile between young and elderly hypertensive patients by Canonico *et al*. (1990).

Furthermore, in keeping with the findings in the present study, statistically significantly higher levels ($p<0.05$) of systolic and diastolic blood pressure(s) were observed of hypertensive patients compared with normotensive human subjects, by Pooja and Yashoda (2014).

Significant differences ($p<0.05$) recorded of the hypertensive patients in comparison with healthy human subjects, in the present study, is corroborated by the findings that significant decrease in mean value of BMI was observed of normotensives compared with those of hypertensives as postulated by Cristiano *et al*. (2007).

World Health Organization (WHO) has identified Body Mass Index as the most useful epidemiological measure of obesity which is a major risk factor for hypertension.

Table 2. Results on blood pressure and body mass index (BMI) of the hypertensive patients, and healthy human subjects.

	Systolic blood pressure (mmHg)	**Diastolic blood pressure** (mmHg)	**Body mass index (BMI) Kg/m^2**
Healthy human subjects	120.52 ± 9.14 [a]	75.6±1.7[a]	15.3±0.7[a]
Hypertensive patients	156.87 ± 11.23 [b]	98.11±0.9[b]	27.01±1.7[b]

Results are expressed as mean ± standard error (S.E) (unit) (n = 20).
Values that are labeled, in the same column, with the same superscripts, are not significantly different (p<0.05).

Table 3. Results on the biochemical indices: Fasting blood sugar, Serum creatinine, and serum aspartate aminotransferase (AST) of the hypertensive patients, and healthy human subjects.

	Fasting blood sugar (mg/dl)	**Serum creatinine (**mg/dl)	**Serum aspartate aminotransferase (U/l)**
Healthy human subjects	82.18 ± 9.16 [a]	1.2 ± 0.1[a]	12.18 ± 1.16 [a]
Hypertensive patients	98.87± 14.23 [b]	1.8 ± 0.2[a]	22.5 ± 2.16 [b]

Results are expressed as mean ± standard error (S.E) (unit) (n = 20).
Values that are labeled, in the same column, with the same superscripts, are not significantly different (p<0.05).

Table 4. Results on the serum electrolytes: serum sodium (Na^+), and Serum magnesium (Mg^{2+}) of the hypertensive patients, and healthy human subjects.

	^Serum sodium (Na^+) (mm/L)	***Serum magnesium (Mg^{2+})(mm/L)**
Healthy human subjects	138 ± 1.16 [a]	2.4 ± 0.1[a]
Hypertensive patients	149± 0.23 [b]	1.11±0.63 [b]

Results are expressed as mean ± standard error (S.E) (unit) (n = 20).
Values that are labeled, in the same column, with the same superscripts, are not significantly different (p<0.05)^; (p<0.01)*.

BMI is a significant predictor of blood pressure (Patil *et al*., 2014).

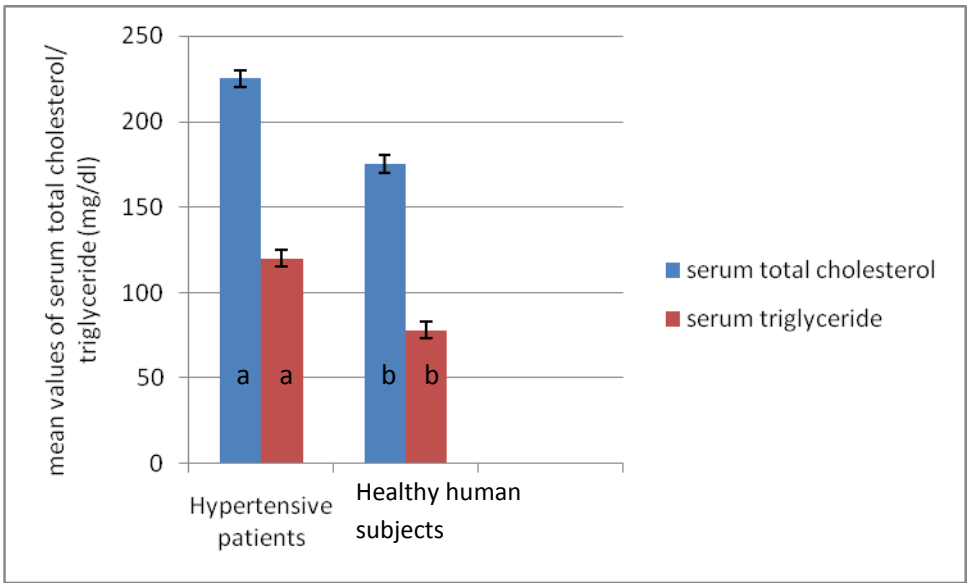
Credence is given to the findings on significant difference in mean values of fasting blood glucose level and AST enzyme activity of human subjects, in the present study by the research submissions which show that mean fasting blood glucose level and AST activity were significantly higher (p<0.05) in hypertensive/portal hypertensive patients, respectively, compared with normal healthy controls, made by Pooja and Yashoda (2014) and Gupta *et al*. (2012).

No significant differences were observed of AST values of normotensive, pre-hypertensive and hypertensive patients in Subharti Medical College, India (Gupta *et al*., 2012). This was because the hypertensive patients were on regular anti-hypertensive drugs: atenolol, amlodipine, and enalapril.

Elevated serum creatinine has been associated with increased mortality in hypertensive persons (Wannamethee *et al*., 1997). However as blood pressure increases, serum creatinine levels decrease and tend to baseline values in hypertensive patients (Lesho *et al*., 2003). This could explain the observed non-significant (p<0.05) but numerical difference between the serum creatinine levels of the hypertensive patients and the healthy human subjects in the present study.

Mean values of serum sodium were significantly higher (p<0.05) in hypertensive patients in comparison with healthy human subjects, a finding consistent with the postulate that a small increase in plasma sodium may be part of the mechanisms whereby dietary salt increases the blood pressure in hypertension as posited by De Wardener *et al*. (2004). Moreover, evidence is rife in patients with essential hypertension and the spontaneously hypertensive rat (SHR) that plasma sodium may be raised by 1 to 3 mmol/L.

Mean values of serum magnesium were significantly lower (p<0.01) in hypertensive patients compared with healthy human subjects, and is a finding similar to the scientific fact that occurrence of low serum magnesium



Statistical results are expressed as mean ± standard error (mg/dl) (n = 20). Error bars represent values of standard error (1.1 – 8.3 mg/dl). Corresponding bars labeled with the same letters represent mean values of serum total cholesterol or serum triglyceride which are not significantly different ($p<0.05$).

Figure 1. Graphical results on the biochemical indices : serum total cholesterol and serum triglyceride of the Hypertensive patients, and Healthy human subjects.

was observed in hypertensive patients as reported by Shaikh *et al*. (2012).

Magnesium deficiency or changes in its metabolism are related to the pathophysiology of hypertension, atherosclerosis, insulin resistance, and diabetes (Cunha *et al*., 2012). Magnesium intake of 500 mg⁄d to 1000 mg⁄d may reduce blood pressure (BP) as much as 5.6⁄2.8 mm Hg (Houston, 2011).

Mean values of serum total cholesterol and serum triglyceride of hypertensive patients, observed in this study were significantly higher ($p<0.05$) than those of the healthy human subjects, in conformity with the observed significant difference ($p<0.01$) between hypertensive and normotensive human subjects in a study area of Northern Bangladesh made by Saha *et al*. (2006).

Elevated serum total cholesterol levels are common in patients with high blood pressure (BP) and could aggravate the hypertensive disease (Borghi *et al*., 2004). The differences in mean of serum total cholesterol, and triglyceride between hypertensive patients and healthy human subjects were statistically significant and in case of serum triglyceride it was statistically highly significant (Sarkar *et al*., 2007).

Multiple regression studies revealed that body mass index (BMI) regressed significantly ($p<0.05$) with serum total cholesterol concentration, serum triglyceride concentration, systolic and diastolic blood pressure(s) of hypertensive patients. The correlation statistical analysis between serum total cholesterol and systolic blood pressure of hypertensive patients was significant ($p<0.05$) with a Pearson's product moment correlation coefficient of 0.975. The concentration of serum total cholesterol could be predicted from the regression curve [$\hat{Y}$ (predicted value of serum total cholesterol) = $\hat{Y}$ (mg/dl) = $33.6 + 1.22x_i$ (mmHg)], xi is observed value of systolic blood pressure (mmHg) (figure 2).

CONCLUSION

Incidence of hypertension correlated positively and significantly ($p<0.05$) with significant increase ($p<0.05$) in systolic blood pressure, diastolic blood pressure, body mass index, fasting blood sugar, serum aspartate aminotransferase, serum total cholesterol, serum triglyceride, and serum sodium; and significant decrease ($p<0.01$) in serum magnesium. There was a significant, positive association between serum total cholesterol and systolic blood pressure of hypertensive patients. Observed values of systolic blood pressure could be used with high precision to predict serum total cholesterol levels of hypertensive patients.

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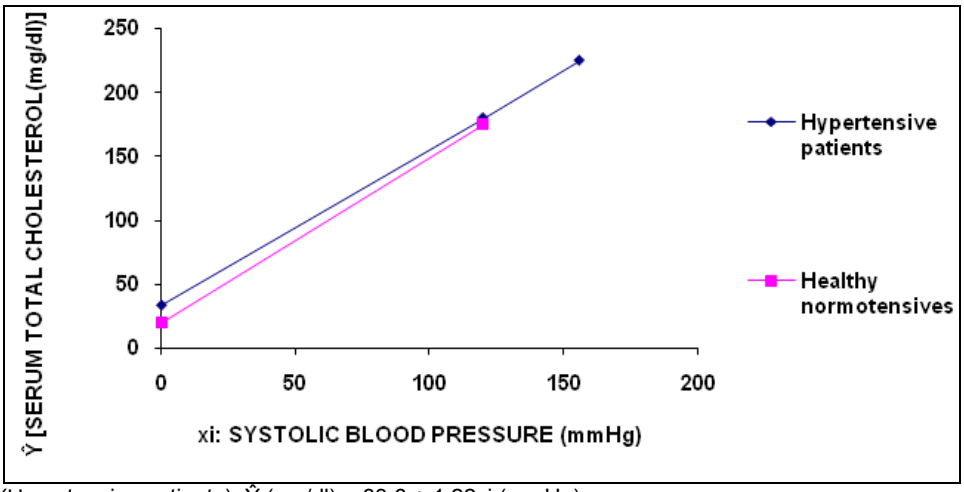

(Hypertensive patients): Ŷ (mg/dl) = 33.6 + 1.22xi (mmHg).
(Healthy normotensives): Ŷ (mg/dl) = 20 + 1.29xi (mmHg).

Figure 2. Regression curve of serum total cholesterol (mg/dl) and Systolic blood pressure (mmHg).

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