EVALUATION OF SERUM CORTISOL, LIPID PROFILE AND ELECTROLYTE AMONG HYPERTENSIVE SUBJECT IN KANO, NIGERIA

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ABSTRACT

Background: Hypertension has now become a global epidemic as a result of lifestyle changes and has become one of the chronic silent killer disease in Nigeria, Some studies have estimated serum cortisol, lipid profile levels in a metropolitan city of Kano-Nigeria.

Aim: The aim of this study was to evaluate between serum cortisol, lipid profile and electrolyte among hypertensive subject inKano.

Methodology: Serum cortisol were analyzed using competitive ELISA method, serum glucose was analyzed using glucose oxidase method, serum lipids were analyzed using enzymatic colorimetric methods and some electrolyte (Na+and K^+) analyzed using flame photometric method.

Results: The mean serum cortisol (22.16±11.01ug/mL) was higher in hypertensive patient with ($p \leq 0.05$) than in control (1.94± 2.38). There were no significant differences in serum glucose, total cholesterol, LDL-C and triglyceride,{ (4.48±2.35), (3.91±3.2) (2.31±0.62) and (1.39±0.71) for control respectively}, between patients and controls. Mean serum HDL-C (1.02±0.23mmol/L) mmol/L was significantly lower in hypertensive patients than in control (1.09±0.24mmol/L) ($p=0.016$). Mean values of serum sodium (144.96±9.62 mmol/L) and potassium concentrations (4.32±0.70mmol/L) were higher in patients than the respective mean values in controls (136.27±5.84) and (3.86±0.36) mmol/L respectively all with ($p \leq 0.001$).

Conclusions: It could be concluded from the finding of the present study that serum cortisol, sodium and potassium concentrations were elevated with consequent advancing in hypertension.

There is a need for Routine evaluation of serum cortisol in addition with aldesterone for the investigation of hypertension. This could improve the management of this group of individuals.

Keywords: Cortisol, Lipid, Potassium, Sodium.

INTRODUCTION

Hypertension simply means high blood pressure. More specifically, it is blood pressure of 140/90 mm Hg or higher. Hypertension has now become a global epidemic as a result of lifestyle changes and has become a chronic silent killer disease (Isezuo 2015). The World Health Organization rates hypertension as one of the most important causes of premature death worldwide and the problem is growing (WHO, 2015). However, many other factors can affect blood pressure including kidneycondition and levels of various hormones in the body (WHO, 2015). Hypertension can be either primary or secondary. Essential hypertension (also called primary hypertension or idiopathic hypertension) is the form of hypertension that by definition has no identifiable cause (Hatakeyama et al., 2000). It is the most common type of hypertension, affecting 95% of hypertensive patients. It tends to be familial and is likely to be the consequence of an interaction between environmental and genetic factors Walker *et al.* (2000). Prevalence of essential hypertension increases with age, and individuals with relatively high blood pressure at younger ages are at increased risk for the subsequent development of hypertension.

Hypertension can increase the risk of cerebral, cardiac and renal events (Whitworth J. A. *et al., 2000*).

Secondary hypertension results from an identifiable cause. Kidney disease is the most common secondary cause of hypertension. Hypertension can also be caused by endocrine conditions such as Cushing's syndrome (glucocorticoides), hyperthyroidism, hypothyroidism, acromegaly, Conn's syndrome or hyperaldosteronism, hyperparathyroidism and pheochromocytoma (O'Brian's, 2007). Other causes of secondary hypertension include obesity, sleep apnea, pregnancy, coarctation of the aorta, excessive alcohol consumption and certain prescribe illegal drugs as well as herbal remedies (Grossman *et al*.,2012). Other rare causes of secondary hypertension include agromegaly (a pituitary tumor that produces excess of growth hormone), adrenocorticotrophic hormone (ACTH) producing tumor of the pituitary gland. The pituitary normally makes a small amount of ACTH daily. Excess ACTH production and secretion causes the adrenal glands to overproduce cortisol, raising blood pressure. Also an ACTH producing cancer of the lung caused hypertension (Williams *et al*., 2013).

MATERIALS AND METHODS

Study Area: The study was conducted at hypertensive (cardiology) clinic of Murtala Muhammad Specialist Hospital, Kano. The area is located in the Kano city, North-western Nigeria with coordinates 11^0 30 N and 8^0 30 E.It share borders with Kaduna State to the South-West, Bauchi State to the South-East, Jigawa to the East and with Katsina State to the West. It has total area of 20,131km^2 (7,773sqm) with an estimated population of approximately10, 334,000.

Hypertensive patients (essential and secondary forms) attending MMSH hypertensive (cardiology) clinic. They were divided in to two groups (control and hypertensive subjects).

Subject: A total of 300 participants consisting of 200 hypertensive patients and 100 apparently healthy individuals as control were recruited as the study subjects. Subject were selected from a population of hypertensive patient attending MMSH hypertensive clinic. For the selection, an interviewer administered Questionnaire was used to elicit the patients socio economic and Demography data and relevant clinical information. Physician on-duty helped the selection process of the patients.

Ethical Consideration: Ethical approvals were obtained from the Research and Ethics Committee of Ministry of Health Kano, in accordance with Helsinki declaration and informed Consent was obtained from each participant, who responded to a standard questionnaire on medical history and biometrics.

Blood sample collection and processing: From each selected subject, 3 ml of venous blood sample was collected using a sterile disposable syringe and needle and allowed to clot at room temperature after which it was centrifuged at 3000 rpm for 5 minutes to obtain a clear unhaemolyzed serum

ANALYTICALMETHODS

Serum Cortisol concentration was estimated using method by Bondy. (1980). Serum Total cholesterol level was estimated using Enzymatic method by Wybenga, *et al*., (1970).Serum HDL-cholesterol level was estimated by Wybenga, *et al*., (1970).Serum triglyceride was estimated using enzymatic method by Wybenga, *et al*., (1970). Serum LDL-Cholesterol level were calculated using Friedewald's Equation by friedewald *et al* (1974).Serum glucose was estimated using enzymatic method(Glucose oxidase).

STATISTICAL ANALYSIS

The data generated was analyzed using statistical package for social sciences (SPSS) software version 20. The results were expressed as mean plus/minus standard deviation (Mean ±SD). Pearson correlation was used to correlate data.

Multiple comparison; variance (ANOVA) were used to compare the results of both the clinical and biochemical parameters obtained from hypertensive patients with those of controls. Pearson correlation was used to correlate data. The p value less than or equal to 0.05 ($p \leq 0.05$) was considered to be significant.

RESULTS

Three hundred (300) subjects were recruited for the study out of which 200 were hypertensive subjects (cases) and 100 were normotensive (controls).

The results of the present study are shown in tables 4.1-4.3 and Figures 4.1-4.5. Table 4.1 shows the values of clinical variables of patients and control group. There were no significant differences between the ages, whereas the mean BMI of the patients group was significantly higher than that of control ($p \leq 0.05$).

The results of biochemical parameters for hypertensive patients and controls were shown in Table 4.2. The mean serum cortisol (22.16±11.01µg/L) was higher ($p \leq 0.05$) while ACTH (9.07±5.5ng/L) was lower($p \leq 0.001$) in patients than in controls There were no significant differences in serum glucose, total cholesterol, LDL-Chol and triglyceride between patients and controls group. Mean serum HDL-C (1.02±0.23 mmol/L) was significantly lower in patients than in controls (1.09±0.24 mmol/L) (p=0.016). Mean values of Serum sodium (144.96 mmol/L) and potassium concentrations (4.32±0.70mmol/L) were higher in patients than the respective mean values in controls (p=0.001).

The results of correlation study between serum cortisol and various biochemical parameters in hypertensive patients are shown in Table 4.3 and figures 4.1-4.4. There were significant positive correlations between the cortisol and TG (r = 0.130 and p = 0.019), sodium (r = 0.175 and p=0.013) and potassium, (r = 0.307 and p = 0.001) (Figs 4.1-4.4), whereas serum HDL-C showed negative correlation with cortisol concentration (r = -

Table 1: Clinical Variables (Mean ± SD) of Hypertensive Patients and Controls

Subject	N	Age (Yrs)	Weight(kg)	Height(m)	BMI(kg/m^2	SBP(mmHg)	DBP(mmHg)
Controls	100	54.47±14.43	66.83±16.10	1.61±0.10	25.10±6.26	122.96±11.86	84.00±8.63
Patients	200	56.45±12.74	69.02±15.63	1.53±0.89	29.23±6.94	140.36±22.33	87.95±11.87
P-value		0.155	0.040	0.081	0.001	0.001	0.005

SBP=Systolic Blood Pressure; DBP = Diastolic Blood Pressure BMI= Body Mass Index

Table 2: Serum Biochemical Parameters (Mean ± SD) in Hypertensive Patients and Control

Subjects/parameters	Control	Hypertensive Patient	
N	100	200	
Sex	50 Male, 50 Female	100 Male, 100 Female	
Cortisol(µg/L)	10.56±2.42	22.16±11.01	0.001
ACTH(ng/L)	16.47±12.85	9.07±5.50	0.001
TC(mmol/L)	3.91±3.20	3.75±0.88	
TG(mmol/L)	1.39±0.71	1.22±0.77	
HDL-C(mmol/L)	1.09±0.24	1.02±0.23	0.016
LDL-C(mmol/L)	2.31±0.62	2.14±0.82	
Na^+(mmol/L)	136.27±5.84	144.96±9.62	0.001
K^+ (mmol/L)	3.86±0.36	4.32±0.70	0.001
GLU(mmol/L)	4.48±2.35	4.31±0.85	

TC =Total cholesterol; TG= Triglycerides; HDL-C=High Density Lipoprotein Cholesterol; LDL-C= Low Density lipoprotein

Table 3: Correlation Between serum Cortisol & Biochemical parameters in Control and Hypertensive Patients

			TC	TG (mmol/L)	HDLC	LDLC	Na^+	K^+	GLU
Cortisol Vs									
Control group	n=100	r	0.002	0.017	0.083	0.102	0.000	0.110	0.092
		P	0.987	0.199	0.411	0.311	0.997	0.274	0.997
Hypertensive	n=200	r	0.040	0.130	0.298	0.028	0.175	0.307	0.038
group		p	0.574	0.019	0.001	0.699	0.013	0.001	0.538

TC =Total cholesterol; TG= Triglycerides; HDL-C=High Density Lipoprotein Cholesterol; LDL-C= Low Density lipoprotein Cholesterol

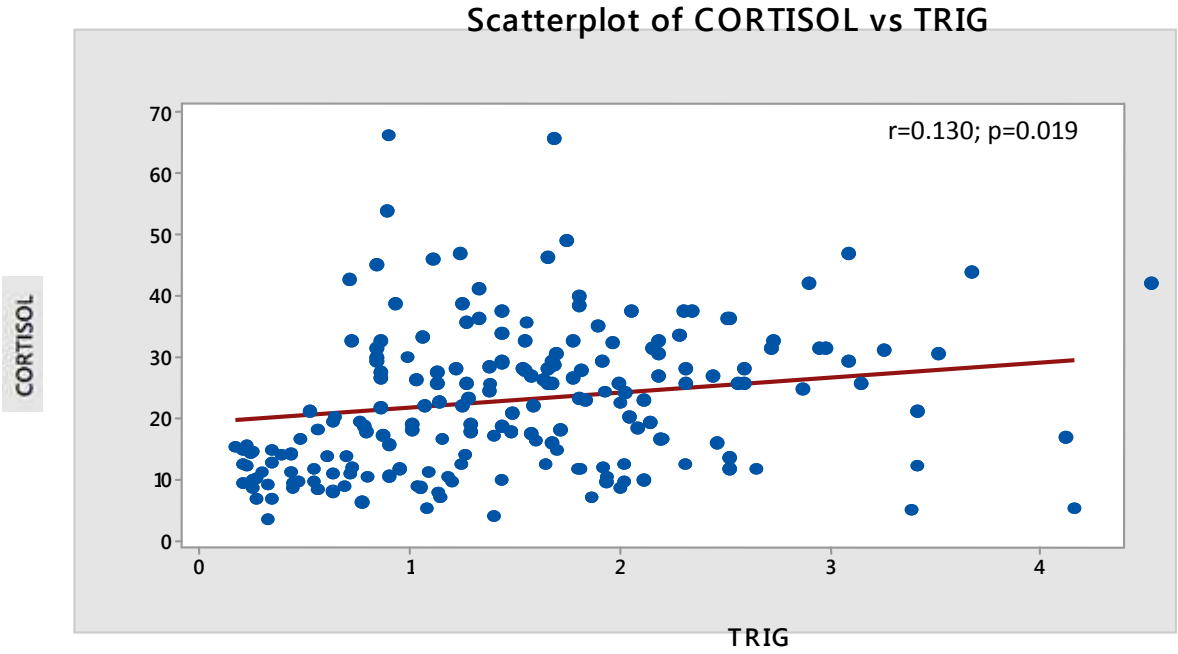

Figure 1: Correlation of serum Cortisol and Triglyceride in Hypertensives

'r=0.307,p=0.0

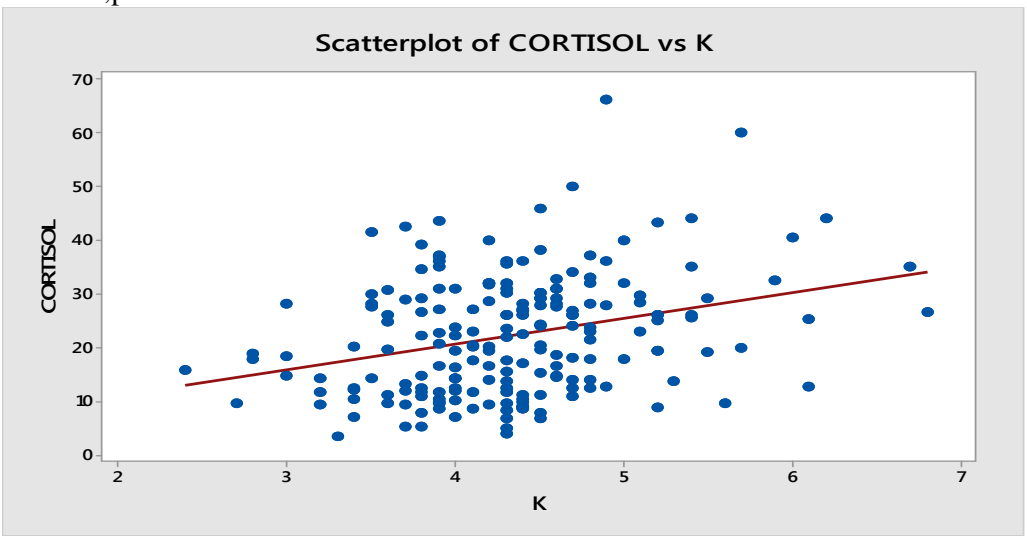

Figure 2: Correlation Between Serum Cortisol and K in Hypertensives

r=0.175;p=0.013

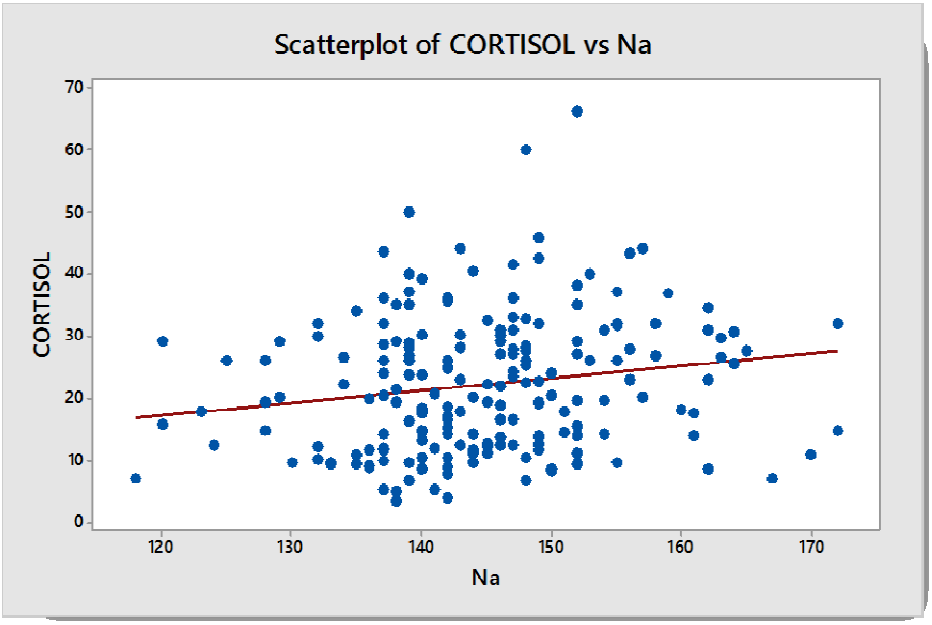

Figure 3 Correlation between serum Cortisol and Na in Hypertensives

r= - 0.298; p= 0.001

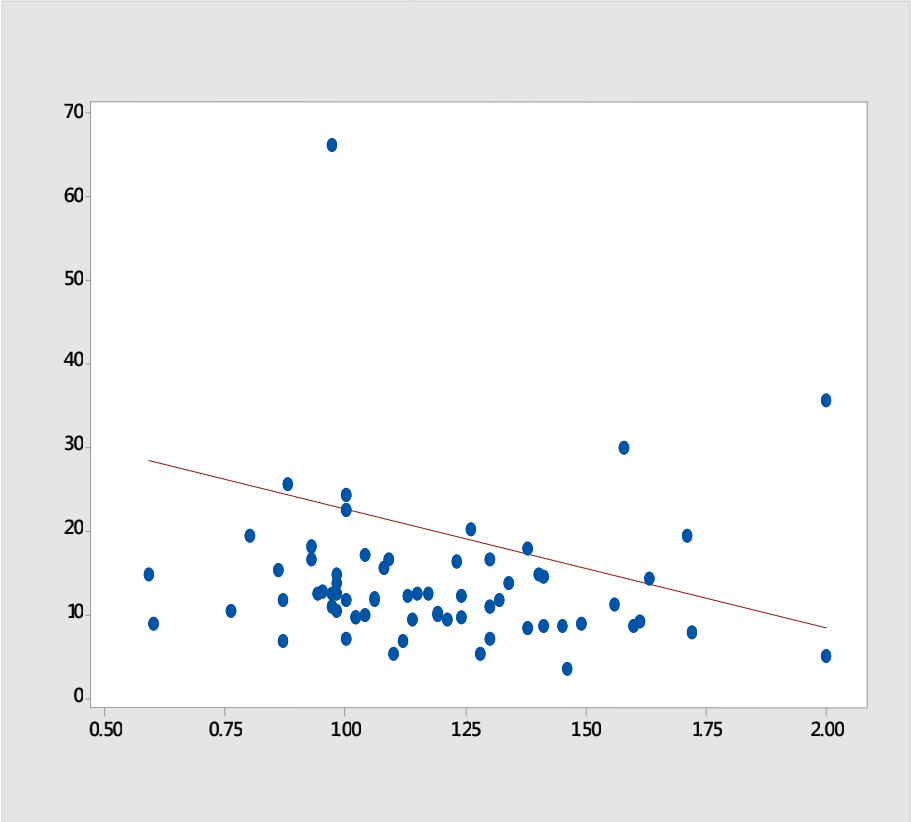

Figure 4 Correlation between serum Cortisol and HDL-C in Hypertensives

Fig. 4.5 CORRELATION BETWEEN SERUM CORTISOL AND ADVANCING HYPERTENSION

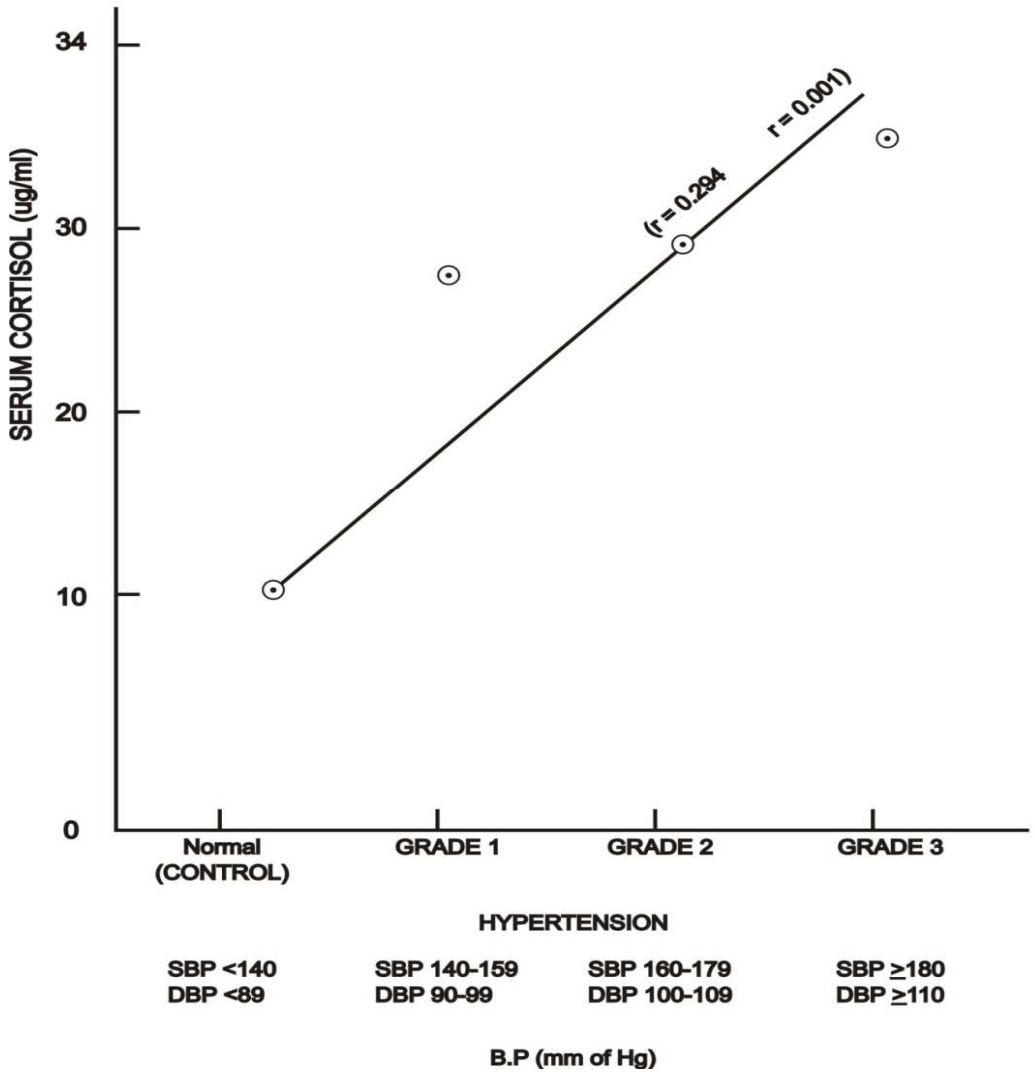

Figure 5: Correlation between Serum and Cortisol and advancing Hypertension

DISCUSSION

The findings of the present study demonstrate that serum cortisol concentration (22.16±11.0ug/ml)were significantly higher in hypertensive patient than in control (10.56±2.42ug/ml). This is in line with pathophysiology of the human cortisol metabolism. Cortisol is a stress hormone known to be secreted in excess in chronic phase response which may lead to hypertension the result is in line with the previous work (Whitworth *et al* 2005 and James *et al*., 2012). There was no significant difference in serum glucose for both hypertensive patients and controls. This is in line with physiology of the body since the subjects studied were not diabetics. Hypertension is a multifactorial condition which includes disturbances in lipids and electrolytes metabolism. Derangement of lipid profile (dyslipidaemia) can predispose to hypertension and metabolic syndrome. But, the results indicate that there were no significant differences in serum total cholesterol, LDL-C and triglyceride between hypertensive patients and controls ($p>0.05$, $p=0.430$ and $p=0.072$ respectively).

Ordinarily dyslipidaemia is expected in hypertension, particularly if it associates with diabetes, but our results do not fall in line with previous findings by Karthikeyan *et al*. (2009). This may have been due to the fact that the patients were educated on the appropriate diet, exercise and even possibly intake of lipid lowering drugs (e.g. Tab lescol) which will probably normalize their lipid profile status.

The mean serum HDL-C was however significantly lower in patients than in controls (p=0.016); this agrees with the previous findings (Bruckert *et al*., 2005). Low HDL-Chol is increasingly recognized as an independent risk factor for adverse cardiovascular disease with increased risk of atherogenic coronary complication. The result further showed high sodium level in hypertension which is known to be associated with both essential and secondary form of the disease.

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Most prominent findings of this investigation is the emergence of direct correlation between the serum cortisol and severity of hypertension (Table 4.8 and Fig 4.5).The rising cortisol level in blood eventually suppresses the pituitary to produce decreased ACTH via negative feedback action, which appears to be intact in this disease.

CONCLUSION

It could be concluded from the findings of the presence study that, Serum cortisol concentrations were elevated with consequent depression of ACTH in hypertension. Serum glucose and lipids concentration were generally normal in hypertensive group. Serum concentrations of sodium and potassium were significantly elevated in hypertensive patients.

RECOMMENDATION

It could be recommended from the findings of the present study, that Routine evaluation of serum cortisol in addition to aldosterone could be considered for the investigation of hypertension. This could improve the management of this group of individuals.

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