

Comparative Biochemical and Metabolic Alteration in Newly Diagnosed Hypertensive and Normotensive Subjects

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Abstract

This study was carried out to observe biochemical and metabolic alteration in newly diagnosed subjects with essential hypertension when compared with normotensive subjects in both men and women within the age ranges of 31-40, 41-50 and 51-60 years. The plasma electrolytes were measured photometrically while bicarbonate was measured using back titration method. Other plasma analytes were measured colorimetrically. The mean value of plasma sodium, creatinine, urea, total cholesterol, low density lipoprotein-cholesterol, triglyceride, and 2 hour post-prandial were found to be significantly increased ($p < 0.0001$) while plasma potassium, high density lipoprotein-cholesterol mean values were found to be significantly lowered ($p < 0.0001$) except calcium with clinical significance when compared with standard. Plasma mean values of fasting blood sugar, total protein and albumin showed no significant changes which may be due to non involvement of renal damage effect of hypertension especially for plasma proteins. This work shows that incidence of essential hypertension is accompanied by biochemical and metabolic alteration and this can serve as a line of investigation for proper diagnosis and treatment.

Keywords: Hypertension, Metabolic effect, Blood pressure, Sodium

1. Introduction

The global prevalence of chronic non-communicable diseases such as hypertension is on the rise with the majority of the growth occurring among populations in developing countries such as Nigeria (Ntentie *et al*, 2014). Hypertension is said to be a very common condition which remain undiagnosed until relatively late in its course, leading to variety of severe and life threatening conditions such as heart failure (Belue *et al*, 2009). The association of elevated blood pressure and metabolic abnormalities with poor cerebrovascular outcome had been recognized long before the concept of the metabolic syndrome became popular (Kannel, 1996). However, the establishment of hypertension as a component of the metabolic syndrome has enabled better insight into the condition and allowed for earlier detection and treatment. According to the Joint National Committee on prevention, detection, evaluation and treatment of high blood pressure (JNC VII, 2003), hypertension is said to be a systolic blood pressure (SP) of 140 mm Hg or greater, diastolic blood pressure (DP) of 90 mm Hg or greater and it has been estimated that over one billion people have hypertension worldwide (Kearney *et al*, 2005). Researches have shown that essential hypertension is the commonest form of hypertension whose etiology remains unknown (Bolivar, 2013). Researches have also shown that 45-81 % of patients with hypertension have their hypertension uncontrolled (Persell *et al*, 2008) which has made it a leading cause of death; hence, there is need for correct and early diagnosis. Findings have shown that hypertension is more than just elevated blood pressure, but it is intimately associated with metabolic and physiological derangement (Schillaci *et al*, 2004). Blood calcium, potassium and magnesium have been implicated in high blood pressure (Cunha *et al*, 2012). It has been discovered that there is a significant inverse correlation between serum magnesium levels and incidence of cardiovascular diseases (Bo and Pisu, 2008). Hypertension has been linked to dietary pattern and other sedentary life style with grave biochemical and metabolically consequences. As a result of this and due to the fact that diagnosis and treatment of hypertension is associated with laboratory findings, this work tends to investigate biochemical and metabolic alteration in newly diagnosed subjects with essential hypertension and normotensive subject in order to assess its metabolic alteration for proper management.

2. Subjects and Methods

This investigation was cross-sectional in nature and it was carried out in the Department of Chemical Pathology, Wesley Guild Hospital Unit of Obafemi Awolowo University Teaching Hospital, Ile-Ife, Osun- State, Nigeria. The subjects involved in this study were 250 subjects with age range of 31-60 years who were attending medical outpatient clinic for the first time and were confirmed to be hypertensive but have not been taking drugs. Subjects with secondary hypertension, nursing mothers and pregnant women were excluded. Informed consent was obtained from the subjects after the study guidelines had been explained to them before brief clinical history was obtained from the subjects. 250 subjects that have no history of hypertension and have not been taking hypertensive drugs were used as control. Blood pressure was measured on left arm by auscultatory method using

mercury sphygmomanometer. The individuals were made comfortable and seated at least for thirty minutes on the chair before measurement. After an overnight fast, blood samples were collected for biochemical analysis into lithium heparinized bottle which includes total cholesterol (TC), high density lipoprotein cholesterol (HDL-C), low density lipoprotein cholesterol (LDL-C) and triglyceride(TG), electrolytes, urea, bicarbonate(HCO_3^-), total protein(TP), albumin(Alb), creatinine (Cr) and calcium(Ca^{2+}). Both fasting and 2hour post-prandial blood sugar samples were collected into fluoride oxalate bottle. The total cholesterol was determined using the enzymatic method (Allain *et al.*, 1974), HDL-C was determined using the precipitation method (Grove, 1979) and triglyceride was determined using the enzymatic method (Esders and Michira, 1997). LDL-C was determined using the Friedwald's formula (Friedwald *et al.*, 1972). Electrolytes were analysed using flame photometric technique(Burtis *et al.*,1999), urea was analysed colorimetrically using diacetylmonoxime method.(Cheesbrough, 1999), bicarbonate was determined using back titration technique(Burtis *et al.*, 1999), total protein and albumin were analysed colorimetrically using biuret and bromocresol green method respectively(Cheesbrough, 1999) and calcium was analysed colorimetrically (Cheesbrough, 1999).The plasma sugar was determined using the enzymatic method (Trinder, 1969).Both hypertensive and normotensive subjects' samples were subjected to the same procedure.

2.1 Statistical analysis: Results are presented as mean \pm SEM. Statistical significance and difference from control and test values evaluated by Student's t-test. Statistical difference at probability of $p < 0.05$ were considered to be significant.

3. Results

3.1. Blood pressure alteration in newly diagnosed hypertensive subjects compared with normotensive subjects.

Blood pressure alteration in newly diagnose hypertensive subjects compared with normotensive subjects are shown in tables 1 and 2. The study involved 250 newly diagnosed hypertensive subjects that have not being taking drugs of which 42% were men and 58% were women while another 250 normotensive subjects served as control of which 46% were men and 54% were women. The age were grouped into 3 categories(31-40, 41-50, 51-60 years) with respective frequencies as shown in tables 1 and 2. For normotensive/hypertensive men subjects, the mean value for SP for the 3 age group were found to be $120 \pm 1.55/147 \pm 1.24$, $123 \pm 1.03/151 \pm 1.00$, $132 \pm 1.36/153 \pm 1.33$ with DP of $84 \pm 2.26/99 \pm 0.85$, $84 \pm 1.58/100 \pm 1.43$, $82 \pm 2.04/95 \pm 0.65$ respectively. While SP mean value for normotensive/hypertensive women subjects were found to be $122 \pm 1.97/151 \pm 1.07$, $124 \pm 1.082/149 \pm 0.98$, $131 \pm 1.71/161 \pm 1.51$ with DP of $82 \pm 1.78/100 \pm 0.10$, $83 \pm 1.78(45)/96 \pm 1.50$, $85 \pm 1.63(50)/100 \pm 0.65$ according to age group. Comparing the normotensive with hypertensive in both men and women irrespective of the age, they were all significant ($p < 0.0001$) (tables 1 and 2).

3.2. Blood electrolyte, urea, creatinine, calcium, total protein and albumin alteration in newly diagnosed hypertensive compared with normotensive subjects.

Blood electrolyte, urea, creatinine, calcium, total protein and albumin alteration in newly diagnosed hypertensive subjects compared with normotensive subjects for both men and women are shown in table 3 and 4. For men, there were significant differences between the mean values of Na^+ , K^+ and Cr when comparing the mean value of normotensive with hypertensive subjects ($p < 0.0001$) within the age groups except Cr at age 31-40 years. Also, there was significant difference between the mean value of Ca^{2+} of both normotensive and hypertensive clinically especially when it was compared with the standard (2.25-2.75mmol/l) (tables 3 and 4) irrespective of the age group and sex. Alteration similar to the above is observed when normotensive women were compared with hypertensive women. Plasma urea level of 40% of the hypertensive subjects both male and female were significantly different from normotensive subjects ($P < 0.0001$) especially between the ages 31-40 and 51-60 years. HCO_3^- , TP and Alb were not statistically significant when comparing normotensive mean values with hypertensive mean value except HCO_3^- at age 41-50 years in men ($27 \pm 0.29(50)/24 \pm 0.38*(40)$) and 51-60 years in women ($24 \pm 0.55(50)/25 \pm 0.22*(60)$)

3.3. Blood lipid profile and sugar alteration in newly diagnosed hypertensive subjects compared with normotensive subjects.

Tables 5 and 6 shows the blood lipid profile and blood sugar alteration in newly diagnosed hypertensive subjects compared with normotensive subjects in both men and women with respect to age groups. There were significant differences ($p < 0.0001$) when the mean values of TC, LDL-C, HDL-C, TG of normotensive men subjects were compared with hypertensive men. 32% of the entire male hypertensive subjects have their blood TC and FBS significantly different ($p < 0.0001$) out of total 42% while 10% which fell within the age range of 31-40 years showed no significant change when compared with normotensive subjects. LDL-C, HDL-C and 2HPP were found to be significantly different irrespective of the age range in male hypertensive subjects when compared

with normotensive subjects ($p < 0.0001$) For women, there were significant difference ($p < 0.0001$) especially in TC, LDL-C, HDL-C, FBS, 2HPP within all age ranges with the exception of age group 41-50 years in TC and FBS and 31-40 years in 2HPP (table 5). In both male and female hypertensive subjects, TG was found to be insignificant different in all ages under consideration except in age 31-40 years in men and 41-50 years in women when compared with normotensive.

4. Discussion

Finding from this study indicates that increase in plasma sodium (Na^+) concentration, one of the major physiological extracellular element is associated irrespective of the age and sex with essential hypertension showing significant difference when compared with normotensive subject ($P < 0.0001$). The above finding support the fact that increase in dietary salt intake usually result into increase in plasma concentration of Na^+ accompanied by increase in blood pressure (Komiya *et al.*, 1997). Therefore, the rise in plasma level of sodium may be traced to dietary salt intake. (Matkovic *et al.* 1989) which has been implicated in stiffness of cardiac arteries (Sasfar *et al.*, 2000; Simon *et al.*, 2001). 100% of the entire test subjects in both sexes and across age groups had their plasma concentration of potassium significantly decreased when compared with normotensive subjects ($P < 0.0001$). The plasma K^+ is inversely related to arterial blood pressure (Treasure and Ploth, 1983). It has been recorded that K^+ has diuretic potential which could be its mechanism of reducing blood pressure, therefore, its decrease may be a contributing factor to the elevated blood pressure (Treasure and Ploth, 1983). Plasma urea level of 40% of the hypertensive subjects both male and female were significantly increased compared to normotensive subjects ($p < 0.0001$) especially between the ages 31-40 and 51-60 years. This suggests, as supported by other findings, that plasma urea is significantly related to age, systolic and diastolic pressure with high incidence in men than women up to 60 years of age (Bulpitt and Breckenridge, 1976). Plasma Ca^{2+} of both hypertensive and normotensive subjects showed no significance statistically but with clinical implication. First, the fact that both test and control subjects irrespective of sex fell below standard reference range of 2.25-2.75mmol/l may suggest a poor dietary intake of calcium among the populace (Martinez, 1998). Secondly, raised plasma Na^+ concentration has been linked with increased in urinary Ca^{2+} excretion and bone turnover which could be another reason why plasma Ca^{2+} is low in hypertensive subjects apart from low dietary intake when compared with normotensive subjects (Wardener and MacGreger, 2002). There were significant increase in plasma creatinine level across the age ranges and sexes in hypertensive subjects when comparing normotensive with hypertensive subjects with the exception of age 31-40 years that have the mean values of normotensive/hypertensive to be $110 \pm 4.62 / 129 \pm 1.71$. Though plasma creatinine concentration is age, sex and muscular size dependent but in heart disease like hypertension, the rate of creatinine production is enhanced in the heart muscle depending on the severity of the hypertensive condition thereby account for usual increase in plasma concentration of creatinine in hypertensive subjects (Coresh *et al.*, 2001). Plasma proteins (total protein and albumin) and bicarbonate (except at age 41-50 years in men ($27 \pm 0.29(50) / 24 \pm 0.38*(40)$) and 51-60 years in women ($24 \pm 0.55(50) / 25 \pm 0.22*(60)$) of hypertensive subjects showed no significant differences irrespective of age range and sex when compared with normotensive subjects which may be as a result of non involvement of renal damage effect of hypertension. The exceptions noticed in plasma HCO_3^- concentration may have no clinical implication because all the values of both test and control subjects fell within the standard range (20-30mmol/l) which further justify noninvolvement of renal damage effect of hypertension. Alterations in plasma lipid profile and sugar metabolism were noticed as a marker in essential hypertension (table 5 and 6). This study revealed that TC, LDL-C and 2HPP mean value of hypertensive subjects were statistically increased ($p < 0.0001$) while HDL-C was reduced which was in line with other findings (Demacker *et al.*, 2000) when comparing normotensive with hypertensive subjects. The above significance patterns were also similar in both sexes and age groups in 2HPP with the exception of 2HPP ($5.9 \pm 0.141 / 6.1 \pm 0.50$) at age 31-40 years in women hypertensive subjects. This explains the reason why dyslipidemia is a major risk factor and biomarker in hypertension. 2HPP results revealed hyperglycemic condition, which usually accompany dyslipidemia and has made both hypertension and diabetes to be co-morbidity. 86% of the entire test subjects (both men and women) have their 2HPP significantly different when compared with normotensive value while 44% already have their 2HPP mean values exceeding glucose renal threshold (10.0mmol/L or 180mg/dl) though FBS mean value showed no significant difference. This implies that 44% of the entire test subjects may have glucosuria, a condition that leads to end organ damage effect of hypertension if not controlled. Increase in blood glucose may have accounted for dyslipidemic condition which may have increase cardiac burden and eventually lead to an elevated blood pressure. About 90% of the entire test subjects had their lipid profile deranged which was in line other finding (Choudhury *et al.*, 2014). 30% of the entire hypertensive group has their TG mean value to be significant increased when compared with normotensive group especially at age 31-40 years in men and 41-50 in women. But clinically, the difference between the plasma TG mean values of normotensive and hypertensive subjects irrespective of the sex suggest that the plasma TG concentration in hypertensive subjects may be moderately higher than normotensive subjects which might have consequence on the blood pressure. Since dyslipidemic

and hyperglycemic conditions noticed in essential hypertension has been linked to dietary style (Kahn, 2012), it may be speculated that whatever dietary style that will alter blood TC will also affect blood TG which eventually affect the blood pressure (Kahn, 2012). The prevalence of the hypertensive condition varies with age though it is more prevalent in women at later age (Gordon, 2000)

5. Conclusion

From the above study, we found out that comparing normotensive subjects with newly diagnosed hypertensive subjects with respect to age and sex, plasma Na^+ , Cr, Urea, TC, LDL-C, TG and 2HPP were found to be significantly increased, K^+ , HDL-C and Ca^{2+} were found to be significantly decreased while FBS, TP, Alb, HCO_3^- shows no significant differences. The above observed metabolic alteration can serve as a line of investigation for proper diagnosis and treatment of essential hypertension.

References

- Ntentie, F.R., Ngondi, J.L., Azantsa, K.B.G., Santy, E.V. & Dimodi, H.T. (2014). Urbanization and Metabolic Syndrome in Cameroon: Alertness on Less Urbanised Areas. *Endocrinology Metabolic Syndrom* 3, 137-145.
- Belue, R., Okoror, T.A., Iwelunmor, J., Taylor, K.D. & Degboe, A.N. (2009). An overview of cardiovascular risk factor burden in sub-Saharan African countries: a socio-cultural perspective. *Global Health* 5: 10.
- Kannel, W.B. (1996). Blood pressure as a cardiovascular risk factor: prevention and treatment. *Journal of America Medical Association* 275(20), 1571-1577.
- Joint National Committee on prevention, detection, evaluation and treatment of high blood pressure (JNC VII report) 2003.
- Kearney, P.M, Whelton M, Reynolds K, Muntner P., Whelton P.K., He, J. (2005). Global Burden of Hypertension: Analysis of Worldwide Data. *Lancet* 365(9455), 217-223.
- Bolivar, J.J. (2013). Essential hypertension: An approach to its etiology and neurogenic pathophysiology. *International Journal of hypertension* 2013:2013-2024.
- Persell, S.D. (2011). Prevalence of Resistant Hypertension in the United States, 2003-2008. *Hypertension* 57(6), 1076-1080.
- Schillaci, G., Pirro, M. & Vaudo, G. (2004). Prognostic value of the metabolic syndrome in essential hypertension. *Journal of the American College of Cardiology* 43, 1817-18229
- Bo, S. & Pisu, E. (2008). Role of dietary magnesium in cardiovascular disease prevention, insulin sensitivity and diabetes. *Current opinion in Lipidology* 19(1), 50-6.
- Allain, C.C., Poon, L.S., Chan, C.S.G. & Richmond, W. (1974). Total cholesterol assay. *Clinical Chemistry* 20: 470-471.
- Grove, T.H. (1979). Grove's method of high density lipoprotein estimation. *Clinical Chemistry* 25, 560-562.
- Esters, T.N. & Michira, C.A. (1997). Triglyceride estimation. *J. Biol. Chem* 254, 710-712.
- Friedwald, W.T., Levy, R.I., Fredrickson, D.S. (1972). Estimation of the Concentration of Low-Density Lipoprotein Cholesterol in Plasma without use of Preparative Ultracentrifugation. *Clinical Chemistry* 18, 499-502.
- Burtis, C.A., Ashwood, E.R. (199). *Tietz Textbook of Clinical Chemistry*. 3rd ed. W.B Saureler company USA.
- Cheesbrough M. (1999). *District laboratory practice in tropical countries, part 1*, Cambridge low price editions, cambridge press, UK.
- Trinder, P. (1969). Determination of blood glucose using 4-aminophenazone as oxygen acceptor. *Journal of Clinical Pathology* 22, 246.
- Komiya, I., Yamada, T., Takasu, N., Asawa, T., Akamine, H., Yagi, N., Nagasawa, Y., Ohtsuka, H., Miyahara Y., Sakai, H., Sato, A., & Aizawa, T. (1997). An abnormal sodium metabolism in Japanese patients with essential hypertension, judged by serum sodium distribution, renal function and the renin-aldosterone system. *Journal of Hypertension* 15, 65-72
- Matkovic, V., Illich J.Z., Anodon, M.B., Hsieh, L.C., Tzagournis M.A., Lager, B.J., Goel, P.K. (1995). Urinary calcium, sodium and bone mass of young females *American Journal of clinical Nutrition* 62:417-442
- Safar, M.E., Thuilliez, C., Richard, V. & Benetos, A. (2000). Pressure-independent contribution of sodium to large artery structure and function in hypertension. *Cardiovasc Research* 46, 269-276
- Simon, G. & Illlyes, G. (2001). Structural vascular changes in hypertension: role of angiotensin II, dietary sodium supplementation, and sympathetic stimulation, alone and in combination in rats. *Hypertension* 37, 255-260.
- Treasure, J., Ploth, D. (1983). Role of dietary potassium in the treatment of hypertension. *Journal of Hypertension* 5(6), 864-72.
- Bulpitt, C.J. & Breckenridge, A. (1976). Plasma urea in hypertensive patients. *British Heart Journal* 38(7), 689-694.
- Martinez, C. (1998). Calcium and hypertension. *Nutrition bytes* 4(2)

deWardenr, H. E.&MacGregor, G. A.(2002).Harmful effects of dietary salt in addition to hypertension. Journal of Human Hypertension16(4),213-23.

Coresh, J., Wei, G.L., McQuillan, G., Branacati, F.L., Levey., A.S, Jones, C., Klag, M.J.(2001). Prevalence of high blood pressure and elevated creatinine level in the United States: findings from the third National Health and Nutrition Examination Survey(1988-1994). Archives of Internal Medicine161,1207-16

Demacker, P.N., Veerkamp, M.J., Bredio, S.J., Marcovina, S.M, de Graaf, J., Stalenhoef, A. F. (2000). Composition of measurement of lipids and lipoproteins versus assay for apolipoprotein B for estimation of coronary heart disease risk: a study in family combined hyperlipidemiaArtherosclerosis153,483-490.

Choudhury, K.N., Mainuddin, A.K.M.,Wahiduzzaman, M., Islam,S.M.S.(2014).Serum lipid profile and its association with hypertension in Bangladesh.Vascular Health and Risk Management 10: 327–332.

Kahan, A. (2012). High blood cholesterol and triglycerides (lipid disorder). Health line.

Gordon, H. (2000).Hypertension vascular disease. In Eugene Braunwald et al (ed).Harrison's Principles of Internal Medicine15th edition. McGraw-New York 1141-1430.

Table 1:
Blood pressure alteration in newly diagnosed hypertensive subjects (H) compared with normotensive (N) subjects (men).

Indices	Age range					
	31 – 40		41 – 50		51-60	
	Normo. (n)	Hyper. (n)	Normo. (n)	Hyper. (n)	Normo. (n)	Hyper. (n)
SP	120±1.55 (30)	147±1.24 (25) *	123±1.03 (50)	151±1.00 (40) *	132±1.36 (35)	153±1.33 (40) *
DP	84±2.26 (30)	99±0.85 (25) *	84±1.58 (50)	100 ±1.43 (40) *	82±2.04 (35)	95±0.65 (40) *

Values are mean ± SEM. Significant difference between normotensive and hypertensive group by t-test *p<0.0001.

Normo. = Normotensive, Hyper = Hypertensive, n = sample size, SP = Systolic blood pressure (mmHg), DP = Diastolic blood Pressure (mmHg).

Table 2:
Blood pressure alteration in newly diagnosed hypertensive subjects (H) compared with normotensive (N) subjects (women).

Indices	Age range					
	31 – 40		41 – 50		51-60	
	Normo. (n)	Hyper. (n)	Normo. (n)	Hyper. (n)	Normo. (n)	Hyper. (n)
SP	122±1.97 (40)	151±1.07 (35)*	124±1.082 (45)	149±0.98 (50) *	131±1.71 (50)	161±1.51 (60) *
DP	82±1.78 (40)	100±0.10 (35) *	83±1.78(45)	96 ±1.50 (50) *	85±1.63 (50)	100±0.65 (60) *

Values are Mean ± SEM. Significant difference between normotensive and hypertensive subjects by t-test *p<0.0001. Normo. = Normotensive, Hyper. = Hypertensive, n = sample size, SP =Systolic blood pressure (mmHg), DP =Diastolic blood pressure (mmHg).

Table 3:
Blood electrolyte, Urea, Creatinine, Calcium, Total protein, and Albumin alteration in newly diagnosed hypertensive (H) subjects compared with normotensive (N) subjects (men).

Indices	Age range					
	31 – 40		41 – 50		51-60	
	Normo. (n)	Hyper. (n)	Normo. (n)	Hyper. (n)	Normo. (n)	Hyper. (n)
Na ⁺	129±0.93 (30)	139±0.89 (25)*	130±0.82 (50)	142±0.60 (40) *	130±0.81 (35)	145±0.82 (40) *
K ⁺	4.1±0.10 (30)	3.4±0.07 (25)*	4.0±0.01 (50)	3.6 ±0.06 (40) *	4.0±0.09 (35)	3.4±0.037 (40) *
Hco ₃ ⁻	24 ±0.34 (30)	23±0.38 (25)	27±0.29 (50)	24±0.38 (40) *	24±0.27 (35)	24±0.29 (40)
Urea	3.6±0.18 (30)	4.7±0.14 (25)*	4.1±0.13 (50)	4.7±0.12 (40)	3.4±0.19 (35)	4.8±0.11 (40)*
Cr	110±4.62 (30)	129±1.71 (25)	92±3.24 (50)	143±2.54 (40)*	108±3.76 (35)	144±2.90 (40)*
Ca ²⁺	2.05±0.06 (30)	2.06±0.07 (25)	1.9±0.05 (50)	1.87±2.54 (40)	2.0±0.06 (35)	1.93 ±0.05 (40)
TP(g/l)	78±2.04 (30)	77±2.13 (25)	82±1.61 (50)	83±1.93 (40)	87±1.56 (35)	83 ±2.08 (40)
Alb(g/l)	41±1.03 (30)	43±0.88 (25)	37±0.96 (50)	38±0.89 (40)	44±0.65 (35)	41 ±0.93 (40)

Values are Mean ± SEM. Significant difference between normotensive and hypertensive subjects by t-test *p<0.0001. Na⁺ = Sodium (mmol/l), K⁺ = Potassium (mmol/l), HCO₃⁻ = Bicarbonate (mmol/l), Cr = creatinine (µmol/l), Ca²⁺ = Calcium (mmmol/l), TP = Total protein (g/l), Alb = Albumin(g/l).

Table 4:
Blood electrolyte, Urea, Creatinine, Calcium, Total protein, and Albumin alteration in newly diagnosed hypertensive (H) subjects compared with normotensive (N) subjects (women)

Indices	Age range					
	31 – 40		41 – 50		51-60	
	Normo. (n)	Hyper. (n)	Normo. (n)	Hyper. (n)	Normo. (n)	Hyper. (n)
Na⁺	129±0.89 (40)	141±0.70 (35)*	129±0.84 (45)	145±0.71 (50) *	130±0.73 (50)	142±0.71 (60) *
K⁺	4.0±0.08 (40)	3.5±0.06 (35)*	4.4±0.06 (45)	3.3 ±0.04 (50) *	4.1±0.10 (50)	3.3±0.03 (60) *
Hco₃⁻	24 ±0.28 (40)	24±0.26 (35)	25±0.24 (45)	25±0.22 (50) *	24±0.55 (50)	25±0.22 (60)
Urea	3.3±0.13 (40)	4.7±0.14 (35)*	4.5±0.18 (45)	4.7±0.12 (50)	5.1±0.10 (50)	4.8±0.11 (60)*
Cr	81±3.77 (40)	133±10.83 (35)	104±3.43 (45)	147±1.25 (50)*	93±3.16 (50)	156±2.73 (60)*
Ca²⁺	1.87±0.05 (40)	1.88±0.06 (35)	2.0±0.05 (45)	1.99±0.05 (50)	2.0±0.05 (50)	1.98 ±0.05 (60)
TP(g/l)	79±1.93 (40)	83±2.19 (35)	90±2.25 (45)	80±1.58 (50)	81±1.68 (50)	82 ±1.49 (60)
Alb(g/l)	44±0.59 (40)	44±0.62 (35)	44±0.54 (45)	36±0.89 (50)*	38±0.85 (50)	38 ±0.85 (60)

Values are Mean ± SEM. Significant difference between normotensive and hypertensive subjects by t-test *p<0.0001. Na⁺ = Sodium (mmol/l), K⁺ = Potassium (mmol/l), HCO₃⁻ = Bicarbonate (mmol/l), Cr = Creatinine (µmol/l), Ca²⁺ = Calcium (mmol/l), TP = Total protein (g/l), Alb = Albumin (g/l).

Table 5:
Blood lipid profile and sugar alteration in newly diagnosed hypertensive (H) subjects compared with normotensive (N) subjects (men)

Indices	Age range					
	31 – 40		41 – 50		51-60	
	Normo. (n)	Hyper. (n)	Normo. (n)	Hyper. (n)	Normo. (n)	Hyper. (n)
TC	4.8±0.19 (30)	5.2±0.18 (25)	4.3±0.14 (50)	5.1±0.71 (40) *	5.0±0.73 (35)	5.7±0.12 (40) *
LDL	2.8±0.07 (30)	4.0±0.20 (25)*	1.5±0.05 (50)	3.7 ±0.15 (40) *	3.3±0.19 (35)	4.5±0.09 (40) *
HDL	1.71±0.05 (30)	0.64±0.04 (25)*	2.04±0.04 (50)	1.15±0.05 (40) *	1.37±0.07 (35)	0.75±0.03 (40)*
TG	0.6±0.03 (30)	1.2±0.09 (25)*	1.7±0.16 (50)	0.7±0.04 (40)	0.8±0.05 (35)	1.0±0.05 (40)*
FBS	3.1±0.04 (30)	3.4±0.06 (25)	5.8±0.16 (50)	5.4±0.17 (40)*	4.3±3.16 (35)	6.8±1.94 (60)*
2HPP	5.2±0.11 (30)	7.4±0.27 (25)*	6.1±0.13 (50)	8.3±0.29 (40)*	5.6±0.14 (35)	9.9±0.22 (40)*

Values are Mean ± SEM. Significant difference between normotensive and hypertensive subjects by t-test *p<0.0001. TC = Total cholesterol (mmol/l), LDL = Low density lipoprotein (mmol/l), HDL = High density lipoprotein (mmol/l), TG = Triglyceride (mmol/l), FBS = Fasting blood sugar (mmol/l), 2HPP = 2hour postprandial (mmol/l).

Table 6:
Blood lipid profile and sugar alteration in newly diagnosed hypertensive (H) subjects compared with normotensive (N) subjects (women)

Indices	Age range					
	31 – 40		41 – 50		51-60	
	Normo. (n)	Hyper. (n)	Normo. (n)	Hyper. (n)	Normo. (n)	Hyper. (n)
TC	3.9±0.18 (40)	4.8±0.12 (35)*	5.0±0.16 (45)	5.5±0.13 (50)	4.8±0.17 (50)	5.7±0.14 (60)*
LDL	2.3±0.09 (40)	3.6±0.19 (35)*	2.9±0.08 (45)	4.2±0.17 (50)*	2.7±0.07 (50)	4.0±0.12 (60)*
HDL	1.2±0.09 (40)	0.71±0.03 (35)*	1.9±0.04 (45)	0.8±0.03 (50)*	1.6±0.04 (50)	1.2±0.04 (60)*
TG	0.8±0.06 (40)	1.1±0.05 (35)	0.7±0.05 (45)	1.2±0.05 (50)*	1.1±0.05 (50)	1.2±0.05 (60)
FBS	4.8±0.15 (40)	5.0±0.16 (35)*	5.2±0.15 (45)	5.6±0.16 (50)	5.2±0.15 (50)	8.5±0.24 (60)*
2HPP	5.9±0.14 (40)	6.1±0.50 (35)	7.0±0.13 (45)	10.0±0.24 (50)*	7.2±0.18 (50)	10.5±0.16 (60)*

Values are Mean± SEM. Significant difference between normotensive and hypertensive subjects by t-test *p<0.0001. TC = Total cholesterol (mmol/l), LDL = Low density lipoprotein (mmol/l), HDL = High density lipoprotein (mmol/l), TG = Triglyceride (mmol/l), FBS = Fasting blood sugar (mmol/l), 2HPP = 2hour post prandial (mmol/l).