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Contrasting Variation of Cationic (Na^+ and K^+) Electrolytes in Blood Serum of Hypertensive and Diabetic Patients around Kano Central, Nigeria

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Abstract: ***This research aimed at comparing the serum level of sodium and potassium ions in hypertensive and diabetic patients in order to ascertain the influence of cationic electrolytes in hypertensive and diabetic patients. 400 patients were group into hypertensive and diabetic groups, 200 per group and 100 healthy controls. The serum level of both sodium and potassium ions were determined. Na^+ was found to have higher (hypernaremia) mean value statistically significant ($p>0.05$) in hypertensive group while below (hyponatremia) normal range in diabetic patients group ($p<0.05$) when compared with healthy control. On comparison, the serum mean level of K^+ was significantly ($p>0.05$) higher (hyperkalemia) in diabetic group while low (hypokalemia) ($p<0.05$) in hypertensive group compared with healthy controls. Body mass index of hypertensive patients was significantly higher ($p>0.05$) than that of diabetic patients and control group and that of diabetic patients was in turn higher than control group.***

Keywords: hypertension, diabetes, Body mass index, sodium ion and potassium ion.

1. Introduction

Hypertension is a persistent high blood pressure in the artery which measures as the ratio of systolic and diastolic pressures. Hypertension occur when blood pressure is above 140/90 mmHg.[8] Most hypertensive patients rarely show symptom and the recognition is by screening or test when discrete clinical case is reported for medication such as headache at the back of head mostly in the morning, altered vision, buzzing or hissing in the ear (tinnitus) and fainting episode,[6]which are even more associated with anxiety than hypertension.[14] Most hypertensive patients attending specialist hospital in Kano city are over 45 years and higher mortality rate was observed at age of 60 and above.[15] Hypertensive outcomes are highly complicated, ranging from mild to highly complex such as ischemic heart disease[11], stroke[17] and peripheral cardiovascular diseases.[19] The risk factor for dementia and cognitive impairment, chronic kidney failure, aortic aneurism, heart failure, atherosclerosis as well as pulmonary embolism was hypertension.[17]

Diabetes mellitus (DM) is a common human disease which is most likely inherited as an autosomal recessive trait.[3]DM is a collection of metabolic disorders in which the blood sugar is elevated over prolonged period.[24] Cause of which is paucity or diminished effectiveness of insulin, resulting in prolonged hyperglycemia and glycosuria.[3] DM causes dysfunction and injury of many tissues and thereby resulting in severe complications.[18]

Body mass index (BMI) is the weight of individual in kilogram per unit meter square. Abnormal BMI is associated with metabolic impairment and some diseases such as hypertension, diabetes gall bladder stroke among others [5, 13].

Compartmentalization of electrolytes into extracellular and intracellular fluids is crucial as any significant departure from normal range can result in severe illness or death [16].

Sodium (Na^+) is a major electrolyte found in large concentration in extracellular fluid compartment [3]. Physiologically, the relative requirement for adult is 1-3.5g, children 0.3-2.5g and 0.1-0.5g for infant [3]. Na^+ maintains crystalloid osmotic pressure and retains water in extracellular fluid.

Potassium (K^+) is the major intracellular cation that involved in neural function and interstitial fluid.[2] The mechanism for potassium excretion is so efficient that normal oral intake of potassium is difficult to cause hyperkalemia.[3] hyperkalemia is often associated with renal failure, dehydration shock and adrenal insufficiency.[7] Lower serum potassium (hypokalemia) is related to negative nitrogen balance, malnutrition, gastrointestinal fluid loss and hyperactivity of adrenal cortex.[7]

The aim of this study is to determine the contrasting variation of cationic (Na^+ and K^+) electrolytes in blood serum of hypertensive and diabetic patients attending Hospitals around Kano central.

2. Materials and Method

Study Design and population

This study was conducted "between July 2014 to March 2015" among patients, aged 40 to 60 years attending Hospitals in Kano State Nigeria. A total number of 500 subjects of both sexes participated in the study comprising 200 hypertensive, 200 diabetic patients and 100 healthy unrelated controls. Self-reported information regarding history of hypertension, diabetes and life styles were taking from individual subject. Physical examination, height and weight were recorded to calculate body mass index (BMI).

Blood pressure was measured using sphygmomanometer (KA-114) from the right arm in sitting position from all the participants. 4 ml of venous blood was collected from both patients and healthy control and spun at 3000revm^{-1} for 5 minutes. The serum was collected using pouch and transferred into a fresh labeled container. Test tubes were labeled as test, standard and blank. Measurements of (STD) both serum sodium and potassium were carried out according to the procedure stated below using Spectrophotometric method.

Determination of Sodium using Trinder, 1951 and Maruna, 1958 principles.[12, 22]

Sodium filtrate preparation

- 1ml of filtrate reagent (R1) was added to all test tubes
- 10µl of sample, sodium reagent (R2) and distilled water was pipetted to test, standard and blank respectively.

The test-tubes were centrifuged for 10 minutes at speed of 1,500G

Colour Development

- Fresh test tubes were also labeled corresponding to the above filtrate test tubes
- 10µl of acid reagent was added to all test tubes
- 50µl of filtrates' supernatant was pipetted to the corresponding test tube and mixed.
- Spectrophotometre was blanked at 550nm using distilled water.
- The absorbance of all test tubes were determined and calculated using the formula;

$$[Na^+] = \frac{\text{Absorbance blank} - \text{Absorbance of test}}{\text{Absorbance of blank} - \text{Absorbance of std.}} \times [\text{std.}]\ (\text{mEq/L})$$

The result of which was recorded in the table 1 below.

Potassium Ion Determination

Potassium determination using Terri and Sesin, 1958 principles.[21]

- Test tubes were labeled as test, standard and blank
- 1ml of potassium reagent was added across the labeled test tubes
- 10µl of sample, standard reagent and nil was pipetted to test standard and blank respectively mixed and allowed to settle for 3 minutes
- Spectrophotometre was blanked at 500nm using blank and the absorbance of tests and standard were recorded and calculated using formula;

$$[K^+] = \frac{\text{Absorbance of sample}}{\text{Absorbance of standard}} \times [\text{std.}]\ (\text{mEq/L})$$

Ethical consideration

Participants of this study were voluntary with written informed consents prior to recruitment. Ministry of Health Kano State of Nigeria ethical committee approved the study. Administrative authorization was obtained from the Hospitals in Kano, and the data treated in the strict respect of anonymity.

Statistical analysis

The data obtained from the participants were compared using Student t-test (two-tailed and paired) between the patients groups and One-Way ANOVA was applied between patients' group and healthy control.

3. Results

Table 1: Sodium level in hypertensive patient, diabetic patient and Healthy control group

Test group	*Mean Na^+ Value (mEg/L)*	*Standard deviation (SD)*
Hypertension	150	3.20
Diabetes	130	3.1
Healthy control	141	3.7

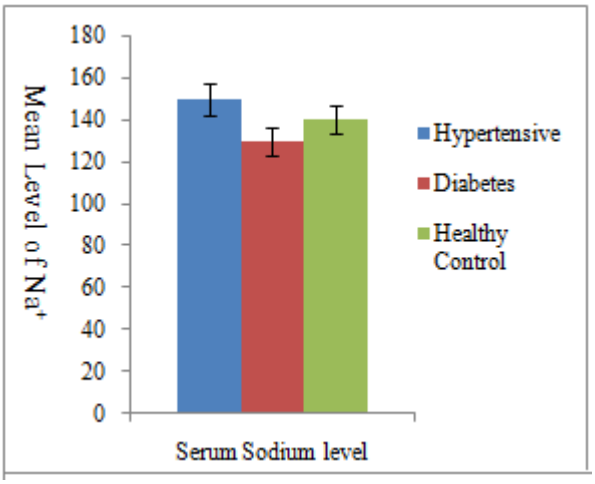

Figure 1: Comparative serum sodium level in Hypertensive Patient, Diabetic patient and healthy Control group

Table 2: Potassium level in Hypertensive patient, diabetic patients and Healthy control

Test Group	*Mean K+ Level (mEg/L)*	*Standard Deviation (SD)*
Hypertension	2.50	0.51
Diabetes	6.10	0.31
HealthyControl	4.5	0.75

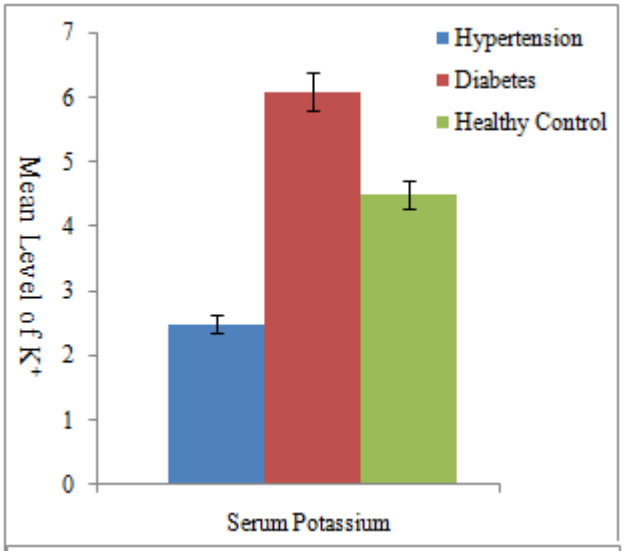

Figure 2: Comparative serum potassium level in Hypertensive patient, diabetic patient and Healthy Control.

Table 3: Body Mass Index in Hypertensive Diabetic patients and Control

BMI	Mean value (Kg/m^2)
Hypertensive	36.1
Diabetic	26.2
Healthy Control	23.3

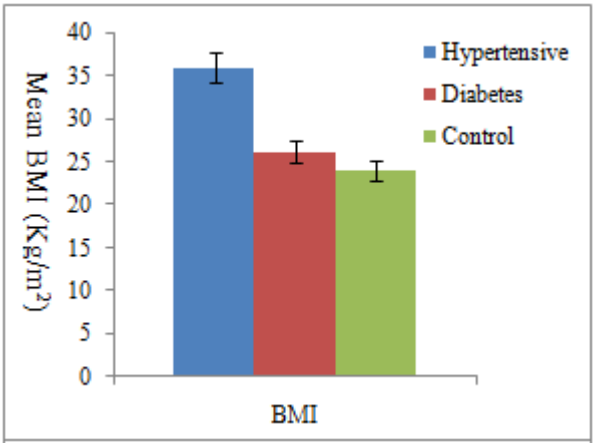

Figure 3: Comparative BMI Value

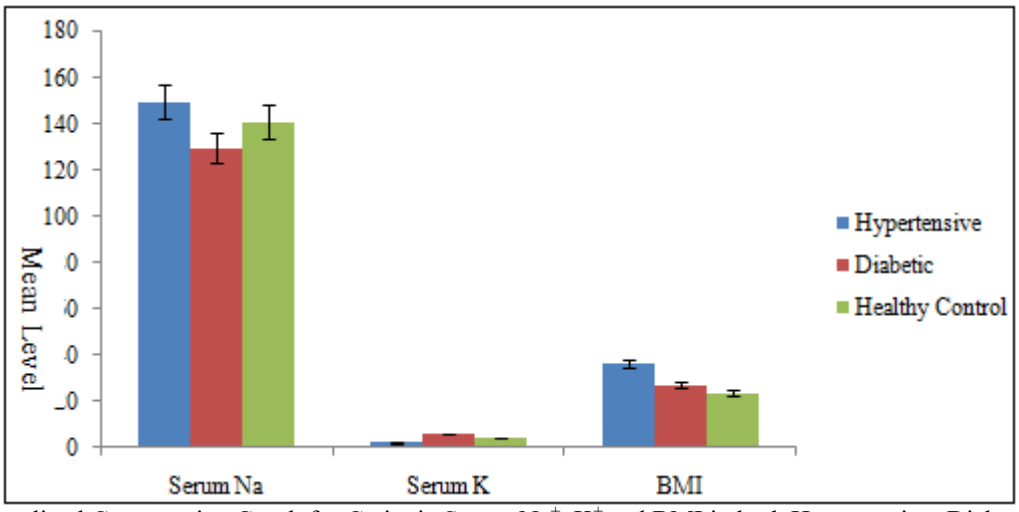

Figure 3: Generalized Comparative Graph for Cationic Serum Na^+, K^+ and BMI in both Hypertensive, Diabetic and Healthy Control Groups

4. Discussion

From table 1 above, the mean serum level of sodium in hypertensive patients was found to be statistically higher ($p>0.05$) than that of diabetic patients. According to the research made in1996 by Elliot and 1997 by Stamler, the sodium salt intensifies blood pressure.[4, 20] It has been reported that the serum level of sodium in hypertensive was found to be increased by ≥2mmol/L among Japanese hypertensive patients.[9] Increased serum sodium concentration (hypernatremia) in hypertensive patients stimulates rennin activity which catalyses the conversion of angiotensinogen to angiotensin I the latter is converted to angiotensin II by angiotensin converting enzyme to stimulate adrenal cortex to secrete aldosterone leading to more water and sodium retention and increase in extracellular pool causing more pressure exertion on arterial wall.[23] The hyponatremic condition in diabetic patients was due to excessively dilutional excretion of sodium and water through urine[10].

Table 2 above shows that, the statistical mean serum level of potassium in hypertensive patients was significantly below (hypokalemia) normal range ($p<0.05$). This result from increased level of aldosterone secretion due to high arterial pressure which in turn causes the excretion of potassium.[18] A statistical significant increase ($p>0.05$) in serum potassium level (hyperkalemia) among diabetic patients was observed. Possibly, fat was broken down to release energy in preference of glucose and excess of which causes acidosis [18]. In ketoacidosis, there was substantial loss of intracellular potassium to extracellular fluid (ECF) causing hyperkalemiain ECF.[18] This could also be related to increased activity of Na^+-K^+ *ATPase* which results from impaired glucose metabolism.[8]

The BMI in hypertensive patients group was higher than that of diabetic patients and control group. This was due to high body fat in hypertensive patient than diabetic and control group [1], and low BMI in diabetic patients than hypertensive group was due to impaired glucose metabolism causing fat metabolism in preference of glucose [18].

5. Conclusion

Hypertensive patients attending MMSH were found to be hypernatremic (higher serum sodium level) while low sodium (hyponatremia) in diabetic patients. Comparing serum potassium level, hypertensive group was found to be hypokalemic whereas the diabetic group was hyperkalemic.

6. Recommendation

We suggest that at age of 40 and above, low salt containing sodium should be taken to avoid rapid raise in BP and make

exercise to avoid over weight. Vegetable containing electrolytes should also be eaten, even though it is expensive in Kano central.

References

[1] Bray, G.A, 2001. Evaluation of fat in Fatter and learner 10-year-old African American and white children: the baton rouge children's study. Am j. clin nutr., 73(4), pp687-702
[2] Campbell and Neil (1987). Biology. Menlo Park Califonia: Beyamin/Cummings pub.co. p. 795 ISBN 0-8053-1840-2.
[3] Chatterjea MN.; Shinde R.; (2007)" Textbook of Medical Biochemistry. 7th Edition p. 358, 571-574.
[4] Elliot P.; (1996). Intersaltrevisited'.; further analysis of 24 hours sodium excretion and BP within and across population; Intersalt Cooperative Research Group. BMJ 312:1249-1253.
[5] Engstrom G. Hedblad B, Stavenow L. Lind P. Janzon L. and Lingarde F. Inflammation-sensitive plasma proteins are associated with future weight gain. Diabetes. Aug 2003; 52(08): 297-101.
[6] Fisher ND, Williams GH (2005). "Hypertensive vascular disease". In Kasper DL, Braunwald E, Fauci AS et al. *Harrison's Principles of Internal Medicine* (16th ed.). New York, NY: McGraw-Hill. pp. 1463–81. ISBN 0-07-139140-1.
[7] Henry, R.F, harper R. Hagerstown, (1974). Clinical Chemistry Principles and Techniques, 2nd Edition.
[8] James, PA.; Oparil, S.; Carter, BL.; Cushman, WC.; Dennison-Himmelfarb, C.; Handler, J.; Lackland, DT.; Lefevre, ML. (Dec 2013). "2014 Evidence-Based Guideline for the Management of High Blood Pressure in Adults: Report From the Panel Members Appointed to the Eighth Joint National Committee (JNC 8)". *JAMA* **311** (5): 507–20. doi:10.1001/jama.2013.284427. PMID 24352797.
[9] komiya, I. Yamada T., Takasu N., Asawa T., Akamine H., (1997). An abnormal Sodium Metabolism in Japanese Patients. J Hypertens P. 65-72.
[10] Komoi K, Tamura T, Tanaka K, Ishibashi M, Yamaji T, (1993). Hyponatremia and Osmoregulation of thirst and Vasopressin Secretion in Patients with Adrenal Insufficiency. J Clin Endocrinal Metab; 77: p 1584-1588
[11] Lewington S, Clarke R, Qizilbash N, Peto R, Collins R; Clarke; Qizilbash; Peto; Collins; Prospective Studies (December 2002). "Age-specific relevance of usual blood pressure to vascular mortality: a meta-analysis of individual data for one million adults in 61 prospective studies". *Lancet* **360** (9349): 1903–13. doi:10.1016/S0140-6736(02)11911-8.PMID 12493255.
[12] Maruna, R.F.L, (1958). Clinical chemistry Acta; 2:581.
[13] Marseglia L, Manti S, D'Anagelo G, Nicotera A, Parisi P, Drosa G, Gitto E, Arrigo T. Oxiadative stress in obesity: a critical component in human diseases. International of molecular sciences. Dec 2014; 16(1): 378-400.
[14] Marshall, IJ; Wolfe, CD; McKevitt, C (Jul 9, 2012). "Lay perspectives on hypertension and drug adherence: systematic review of qualitative research. *BMJ (Clinical research ed.)* **345**: e3953. doi:10.1136/bmj.e3953. PMC 3392078. PMID 22777025.
[15] Musini VM, Tejani AM, Bassett K, Wright JM; Tejani; Bassett; Wright (2009).Musini, Vijaya M, ed. "Pharmacotherapy for hypertension in the elderly". *Cochrane Database of Systematic Reviews* (4):CD000028. doi:10.1002/14651858.CD000028.pub2.PMID 19821263
[16] Nelson D.L.; Cox M.M.; (2007). Lehninger principles of Biochemistry.P. 901.
[17] O'Brien, Eoin; Beevers, D. G.; Lip, Gregory Y. H. (2007).*ABC of hypertension*. London: BMJ Books. ISBN 1-4051-3061-X.
[18] Sembulingam K,;Prema, (2010). Essential Medical Physiology 5th Edition. P. 404-407.
[19] Singer DR, Kite A; Kite (June 2008). "Management of hypertension in peripheral arterial disease: does the choice of drugs matter?" *European Journal of Vascular and Endovascular Surgery* **35** (6): 701–8. doi:10.1016/j.ejvs.2008.01.007. PMID 18375152
[20] Stamler J. (1997). The intersalt study: Background, Methods, Findings and Implications. AmJ cli Nutr 65(suppl): 626-642.
[21] Terry, A., E., Sesin P., G., (1958). Am. J. Clin. Path. 29:86.
[22] Trinder, P. (1951) Analyst, 76:596.
[23] Tripathi KD.; (2009). Essential of Medical Pharmacology, 6th Edition. P. 480
[24] World Health Organization About diabetes". Retrieved 4 April 2014