Advances in Life Science and Technology
ISSN 2224-7181 (Paper) ISSN 2225-062X (Online)
Vol.99, 2023

Comorbid Status Evaluation of Some Serum ionic content in Glucose excess, Hypertension and obesity.

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ABSTRACT:
Changes in the Levels of serum ion have earlier been reported in some pathophysiological conditions. We studied these changes in the triad of Glucose Excess, Hypertension, obesity and in a comorbid state. We evaluated calcium (Ca^{2+}), magnesium (mg^{2+}), lithium (Li), zinc (Zn^{2+}), phosphorus (P_{04}), glycated haemoglobin (HbA1c) and hydrogen ion concentration (P^{H}) along with some other parameters. Adopting spectrophotometric, flame photometric and conductimetric methods parameters were analyzed. Using two way analysis of variance values obtained were statistically evaluated. The conditions presented varying degrees of elevation of Ca^{2+}, Zn^{2+} and HbA_1C. Mg^{2+}, P_{04-}, K^{+} and P^{H} however correlated inversely with HbA1c. Comorbid condition show a trend in which most of the parameters were slightly reduced. Values obtained is suggestive of these ions affecting acid base balance and homestasis.
Keywords: ionic content, Glucose excess, hypertension, obesity, comorbid.
DOI: 10.7176/ALST/99-04
Publication date: December 31st 2023

INTRODUCTION:

Diabetes mellitus prevalence has increased tremendously over the years and efforts at treatment have not yielded desired results. Complications arising from the disease has exacerbated the difficulties in management. Global current and future estimate looks worrisome with attendant morbidity and mortality. The rising prevalence gives support to the development of several metabolic disorders, acting as triggers to an array of metabolic syndrome notably hypertension, obesity, cardiovascular disease, retinopathy and dyslipidaemia [1]. Varieties in our daily food consumption with different ionic composition has the capacity to alter body metabolities and also change the metabolic processes and affect the body homeostasis [2]. The interplay of ionic components in our diet and glucose metabolism has not been fully understood. Insulin signaling pathway and how this relates to macro/micro nutrient and their interaction with other chemicals in the body requires further elucidation. In a disordered state of signaling, insulin resistance occurs. This can give rise to changes in normal energy use, lipid metabolism and inflammation [3]. Ionic substances are required in low concentration for normal body growth and survival. They are known to play important functions as activating co-factors, co-enzymes, in genetic transcription and oxidation stress monitors. The completion of work on the human genome has brought to the fore the presence of several genetic variants which have complex relationship with nutrition with associated individual variability that regulate gene expression and epigenetic features which have brought the need for personalized nutritional requirements [4,5]. Evidence from previous works have shown that hypertension is not a single disease entity with one identifiable etiology. It has been known that there exist vagaries of factors responsible for its triggering, sustenance and expression of the condition. Metabolic abnormalities in conditions such as obesity, glucose intolerance, dyslipidaemia have some degree of intricate association.

To a large extent, the relationship of hypertension and diabetes have been established [6] however the intricate underlying biochemical mechanism have not been clearly demonstrated. The need to evaluate some levels of

ionic component such as Ca^{2+}, Mg^{2+}, L^{i+}, Z^{2+} and K^{+} which are very important micronutrients have been the driving force in this research conception. Previous literature have shown that cardiovascular diseases in diabetes is associated with nephropathy while the accompanying artherosclerosis maybe related to microangiopathy as an important contributor. The effects of reactive oxygen free radicals have also been implicated in this vascular injury. In the presence of ascorbic acid and ∞-tocopherol an environment for the natural defence against increasing reactive oxygen specie is inhibited. A valuable clue to the relationship between hypertension and diabetes has been provided earlier [7] where it has been observed that the uptake mechanism for ascorbic acid and dehydroascorbic acid were impaired in lymphoblast with hypertension or diabetic nephropathy [8]. This asserts one of the claim of micronutrients in diabetic disease.

MATERIALS AND METHOD

The research was carried out at three tertiary hospitals, Federal Medical Centre, Diete Cooke Memorial Hospital and Niger Delta University Teaching Hospital all in Yenagoa, Bayelsa State of Nigeria. Thirty patients were selected in the respective disease category who met research inclusion criteria from each health facility. Another 30 apparently healthy individuals were selected and served as control. Serum samples were used for the tests except Glycated hemogboin.

Analytical Method

Glucose was determined by the glucose oxidase method using a product of Randox, UK. Glycated Haemoglobin (HbA1C) was quantitated by immuno assay. Sodium (Na^{+}) Potassium (K^{+}) and Lithium (Li+) was quantitated by flame photometry. Bicarbonate ($HC0_3^{-}$) was determined by Titration method.

Calcium (Ca^{2+}) was determined using the O-cresolpthalein complexion (CPC) method at 518nm. The Xyldyl method was used for the determination Magnesium (Mg^{2+}) and was measured at 546nm. While phosphorus was analyzed using molybdate method and was measured at 340nm.

Statistical Analysis

Concentration of analytes were statiscally evaluated and expressed as ± SEM with the aid of SPSS version 23. The means were compared at P Value of 0.05.

RESULT

Table 1: Observed levels of some metabolites in Diabetics, Hypertension and Obese and comorbid.

	Control	**Diabetic**	**Hypertensive**	**Obese**	**Comorbid**
Glucose (mmol/L)	4.3 ± 1.5	12.5 ± 1.5	10.0 ± 2.0	8.3 ± 5.0	11.5 ± 3.00
HBA IC (%)	4.7 ± 2.2	10.3 ± 3.4	8.5 ± 1.5	7.2 ± 2.0	10.6 ± 2.00
Na (mmol/L)	138 ± 5.5	146 ± 4.8	150 ± 3.6	14.9 ± 3.0	140 ± 2.07
K^{+} (mmol/L)	3.6 ± 1.3	3.3 ± 1.3	3.8 ± 2.5	3.9 ± 2.0	3.9 ± 0.25
Li (mmol/L)	0.8 ± 1.5	0.6 ± 0.1	0.5 ± 0.2	0.5 ± 1.0	0.6 ± 0.01
H_2CO_3(mmol/L)	28 ± 5.2	24 ± 3.0	30.4 ± 1.3	32.0 ± 3.2	33.3 ± 2.10

All results are expressed as ± SEM of triplicate determinations. P = 0.05

Table 2: Observed values of micronutrient and P^H in Diabetics, Hypertensive Obese and comorbid.

	Control	**Diabetic**	**Hypertensive**	**Obese**	**Comorbid**
Ca^{2+}(mmol/L)	2.0±0.02	2.8 ± 0.6	2.6 ± 0.6	2.2 ± 0.5	1.9 ± 0.3
Mg^{2+}(mmol/L)	0.5±0.1	0.3 ± 0.03	0.4 ± 0.03	0.4 ± 0.3	0.34 ± 0.5
Po^{-}_4 (mmol/L)	1.2 ± 0.18	0.8 ± 1.00	0.9 ± 0.8	0.7 ± 1.0	0.6 ± 0.04
Zn^{2+} (mmol/L)	14.0 ± 4	7.3 ± 2.4	6.8 ± 2.1	6.5 ± 2.3	7.5 ± 1.22
P^H	7.4 ± 0.1	7.3 ± 0.01	7.3 ± 0.01	7.2 ± 0.1	7.21 ± 0.01

All results are expressed as ± SEM of triplicate determination. P= 0.05

Table 1 show values obtained for some metabolites in the three disease conditions and comorbid state examined. While glucose is above threshold in diabetics it also correlated positively with glycated haemoglobin with significant variation in sodium and bicarbonate levels as compared with control.

Table 2 show values for micronutrients evaluated. We observed reduced value of Zn^{2+}, and Mg^{2+}. Potassium also correlated inversely with HbAIC. Cormobid elicited reduced values for most of the analytes when compared with control.

DISCUSSION

Awareness of an existing relationship between diabetes mellitus, hypertension and obesity has been recognized. The realization that there is a nexus between hypertension and insulin resistance has steered attention for a possible role in the pathophysiology of hypertension, obesity and artheroslerotic disease [9,10]. Previous works have opined that diabetes exercerbate hypertension especially as a result of hyperinsulinemia. Findings in this work elucidated the fact that diabetes mellitus, hypertension and obesity are associated in some ways as shown by the values obtained from the parameters. Some of these parameters which are used as markers analytes were elevated. We observed glucose levels above threshold for all the diabetic patients suggestive of the fact that glucose is not an inert participant in diabetes. It is now known that glucose is a strong contributing factor when it comes to maintenance of cellular ion homeostasis and contributes to the increased risk and pathohysiology of hypertension in conditions of glucose excess [11,12,13]. We have observed in this work levels of calcium that was at variance with the control (see figure 2). This finding was in agreement with [14]. Additionally HbAIC show positive correlation with glucose. We observed a reduced level of Ca^{2+} and Mg^{2+}. Similar observations were made for phosphate, Zinc and P^H.

Attempt is made to explain the mechanism that may lead to hypertension in glucose excess. It is important to note that metabolic disorders with the terad of glucose excess, obesity, hypertension and dyslipidaemia have a common nexus sharing insulin resistance that may be organ or cell specific reflecting presentation of a common cellular ionic effect [15,16]. For the maintenance of homeostasis, ionic concentration in the body must be sustained at a balance. Calcium plays a pivotal role in ionic regulation of cellular function in their presence as co-factors in vitamin and a vital role in control of blood pressure through protection of Nitrous Oxide. Previous evidence have pointed out a direct effect of glucose on red cell membrane calcium ATPase which has an association with end products of glycation of protein [17,18,19]. Moreover, elevated glucose uptake and subsequent glycolysis has the capacity to raise organic acid formation and reduce ultracellular P^H a similar

mechanism by which Mg^{2+} is also reduced. The fact that most of the level of ion was closely related in hypertension, diabetes and obesity strengthen the argument that they ionic disorder prevails in these conditions.

References

1. Zimmet, P, Albert, K. G. and shaw, J. (2001) Global and Societal Implications of the diabetes epidemic, *Nature* 414, 782-787 doi: 10.1038/414782a.

2. J. Chebade, M. Sheikh-Kh-Ali and A.D. Mooradian (2009). The role of micronutrients in managing Diabetes. *Diabetes spectrum* 22(4),214-218, 2009.

3. Perry, R. J., Samuel, V. T., Petersen, K, F., and Shalman, G, I. (2014). The role of hepatic lipids in hepatic insulin resistance and type 2 diabetics. *Nature* 510,84-91 doi:10.1038/nature 13478.

4. M. Saif-Elnasr, I. M. Ibrahim and M. Alkady. (2007) Role of vitamin D on glycemic control and oxidative stress in type 2 Diabetes Mellitus. *Journal of Research in Medical Science* 22(1) 2017. hHps://doi.org/10.4103/1735-1995.200278

5. San-Cristobal, R., Navas-Carretero, S, Celis-Morales, C., Brannan, I., Walsh, N., Lovegrove, J. A., et al, (2015) Analysis of dietary pattern. Impact on weight status for personalized nutrition through on-line advice. The food 4me Spanish cohort. *Nutrients* 7, 9523-9537 doi;10.3390/nu7115482

6. K. Protich, D, Yegon, NC. & Walekhwa, M.N (2021) Level of Micro nutrients affect Pathophysiology of diabetes mellitus. A scoping review *European Journal of medical and health science.* 3(5), 6 -13. https://doi.Org/10/.24018. Ejmed. 2021. 3.5. 1019.

7. Marmicadi V, Camellini L, Bellodi G. (1986). Evidence for an association of high blood pressure and hyperinsulinemia in Obeseman. *Journal of Clinical Endocrinology and Metabolism* 62, 1302-1304.

8. Lobo V. Patil A, Phatak A, Chandra N. (2010). Free radical, antioxidants and functional foods: impact on health. *Pharmacosnosy reviews*. 2010, 4(8). 118

9. D. Farooq, A. Alamri, B. Alwhabi, A. Metwally and K. Korean (2020). The status of zinc in type 2 diabetic patients and its association with glycemic control. *Journal of family community medicine* 27 (1) 29-36 2020. https://dor.Org/10.4103/ifcm. JFCM_113_19

10. V. Higuera (2019). Magnesiun and diabetes. How are they related? *Diabetes care* 1-10, 2019

11. F.O.O. Olabiyi (2013). Role of nutrition in the management of diabetes. *Forum Nutr* 56, 170 – 174, 2013. https//dor.org/10.1/276/ap,hs. 2016.3.1.1.

12. A.D Mooradian, B. Hoogwert, M. Marynuik and J. Wyle – Rossett. (1994) Selected vitamin and minerals in diabetes (1994). *Diabetes care*, 7 (5) 464 – 497, 1994.

13. L, N Kohler, A Florea, CP Kelly, Schow, PH K Batai, K Saboda, P Lance and E.T. Jacobs (2015). Higher plasma selenuim concentrations are associated with increased odds of prevailing type 2 diabetes. *Journal of nutrition*, 148 (8) 1333 – 1340.

14. Ma Youssef, S.A Elkhodery and H.M.M Ibrahim (2013). Effect of selenium and vitamin C on clinical outcomes, trace elements status, and antioxidant enzyme activity in horse with acute chronic lower airway diseases. A randomized clinical trial. *Biological trace element research* 152 (3), 333-342, 2013. https://dor.Org/10.1007/512011-013-9636-5

15. Vaziri N.D. Liang K, Ding Y (1999). Increased nitric oxide inactivation by reactive oxygen species in lead-induced hypertension. *Kidney International* 56,1492-1498.

16. Sutter P.M. (1998). Potassium and hypertension. Nutrition Review 56,151-153.

17. Poitier M, Dancel N, Tome (2009). Protein, amino acids and control of food intake. Curr Opiniclin. Nutr. *Metab Care* 12, 54-58 doi:10.1097/MCO. Obo13e32831bge01

18. Vary T.C. and Lynch C. J. (2007). Nutrient signaling components controlling protein synthesis in striated muscles. J. *Nutr.* 137, 1835-1843 doi.1093/jn/137.8.1835

19. Chou, C.J, Affolter M, Kussmann M. (2012). A nutrigenomics view of protein intake, micronutrient, bioactive peptides and protein turnover. *Prog. Mol Biol Transl.* Sci.108,51-74 doi:1016/B978-0-12-398397-8.00003-4