Original Research Article

Comparative study of electrolyte levels with various level of HbA_1C in diabetes mellitus patients

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Abstract

Objective: The aim of this study is to estimate of electrolyte levels in patient with diabetes mellitus and compare the result among the various levels of HbA_1C and also to find out the correlation between electrolytes and HbA_1C. Methods**:** This longitudinal study was conducted in a saveetha medical college hospital in Chennai for a period of 3 months. Blood samples were collected from 200 diabetic subjects. Samples were analysed for HbA_1C, sodium, potassium, chloride, and bicarbonate levels. **Results:** A significant decrease in levels of electrolyte Na and Cl with various HbA1c levels and there is no statistical significant in potassium and bicarbonate was observed when compare among the various levels of HbA1c. **Conclusion:** It may be concluded from this study that hyperglycemia(uncontrolled DM) dysregulation of glucose homeostasis may lead to electrolyte imbalance.

Key Words: Bicarbonate, Chloride, Diabetes, Electrolytes, HbA_1C, Potassium, Sodium.

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INTRODUCTION

Diabetes mellitus is heterogenous group of metabolic disorder characterized by high blood glucose levels. It is alteration in carbohydrates, lipids, and proteins metabolism resulting from defects in due to one of two mechanisms,[1] inadequate production of insulin (which is made by the pancreas and lowers blood glucose) and inadequate sensitivity of cells to the action of insulin, the main type of diabetes corresponds to these two mechanisms are called insulin dependent (type-1) and non insulin dependent(type-2) diabetes.[2] Clinically it is common condition affecting about 10% of the total population and 1/5th of persons above the age of 50 years.[3]there are probably 100 million peoples in the world. India has now more than 50 million peoples with type 2 diabetes mellitus and is being called as the "Diabetic Capital" of the world. It is major cause for morbidity and mortality.[4] Electrolytes are the substance conducts electricity in the body. which means it carries tiny electrical charges(potential)[5] Fluid and electrolyte balance play important roles in intermediary metabolism, maintaining homeostasis in the body, and also protecting cellular function, tissue perfusion, acid basebalance, muscle contraction, and nerve conduction.[6,7] electrolyte imbalance resulting from kidney failure, dehydration, fever and vomiting has been suggested as one of the contributing factors towards complications observed in diabetes and other endocrine disorders.[8] Electrolyte imbalance in diabetes is primarily results of elevated blood glucose with hyperglycemia, the tries to rid of itself of the excess blood glucose by increasing urinary output. Increased urination produces water and electrolyte loss, which then upsets the body's balance of electrolytes. The

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balance is especially disturbed between sodium and potassium. Symptoms of electrolyte imbalance includes headache, fatigue, muscle pain, irritability, according to the mayo clinic. As cells becomes more starved of glucose of their energy needs, the body needs to compensate by providing another energy source, that source comes from fatty acids, which are less efficient energy producing chemicals. Fatty acid metabolism can leads build of a byproduct called ketones, which can upsets the acid base upset relation of the body. That acid-base upset may results in a condition known as ketoacidosis which can serve and even life threatening.[9] The prevalence diabetes mellitus in india is increasing rapidly.[10] however scant data are available on serum electrolytes levels in adults with diabetes mellitus.[11]

MATERIALS AND METHODS

The present study was conducted in the Department of the Biochemistry, Saveetha Medical college hospital.200 diagnosed cases of diabetes mellitus patients were chosen for the study which belongs to age group 35-70 years. The study group in 200 subjects were divided into 4 groups with various HbA1c levels in each 50 subjects, as group-1 (4.5-5.5), group-2 (5.6-6.8), group-3 (6.9-7.6) and group-4 (>7.6). Subjects with metabolic syndrome, pregnancy, thyroid disorder, liver disorder, cardiovascular complications, renal disorders, steroids and hypertensive drugs usage, acute and chronic illness, alcohol intake and other disorders were excluded from the study. Informed consent form was obtained from the all cases. Due permission was obtained from the ethical clearance committee for this study. (10 ml of blood sample were collected by vein puncture from the all the cases fasting, postprandial and randomly. HbA_1C levels were estimated by high performance liquid chromatography. Blood sugar (by GOD POD) and electrolyte levels were estimated by Reflectance spectrometry method (Na and K–ion selective electrode), (cl-ferric thiocynate), (HCO_3-PEPC). All the results were expressed as ± SD and statistical comparisons were done by using ANOVA. Due permission was obtained from institutional ethics committee prior to the starting of the work.

RESULTS

Table 1: HbA1C Descriptives

HbA1C	Number	Mean	Std. deviation	Std.Error
4.5-5.5	50	5.178	0.3649	0.516
5.6-6.8	50	6.104	0.4030	0.570
6.9-7.6	50	10.806	0.2311	0.327
>7.6	50	7.329	2.5139	0.3563

Table 2: Electrolyte Descriptives

ELECTROLYTES	HbA1C	N	MEAN	SD	Std.error
Sodium	4.5-5.5	50	138.72	3.247	0.459
	5.6-6.8	50	138.32	3.930	0.556
	6.9-7.6	50	137.50	4.925	0.696
	>7.6	50	135.28	3.964	0.561
Potassium	4.5-5.5	50	4.036	0.5840	0.0826
	5.6-6.8	50	4.202	0.6592	0.0932
	6.9-7.6	50	4.337	0.6414	0.0907
	>7.6	50	4.168	0.7807	0.1115
Chloride	4.5-5.5	50	102.62	2.672	0.378
	5.6-6.8	50	102.17	4.351	0.615
	6.9-7.6	50	99.55	5.405	0.764
	>7.6	50	96.55	5.366	0.759
Bicarbonate	4.5-5.5	50	24.64	2.497	0.353
	5.6-6.8	50	24.82	2.513	0.355
	6.9-7.6	50	25.52	3.019	0.427
	>7.6	50	25.50	3.507	0.496

Table 3: ANOVA Test

Electrolyts	Comparison Of Groups	Sum Of Squares	Mean Square	F	Sig.
Sodium	Between groups	354.553	118.184		
	With groups	3231.881	16.489	7.167	0.000
Potassium	Between groups	2.296	0.765		
	Within groups	87.421	0.448	1.707	0.167
Chloride	Between groups	1032.135	344.045		
	Within groups	4119.790	21.019	16.368	0.000
Bicarbonate	Between groups	31.240	10.413		
	Within groups	1663.880	1663.880	1.227	0.301

Table 4: Multiple Comparison

(I)group	(J)group	Sig.			
		Na	K	Cl	Hco_3
4.5-5.5	5.6-6.8	0.960	0.605	0.962	0.990
	6.9-7.6	0.434	0.114	0.005	0.433
	>7.6	0.000	0.761	0.000	0.454
5.6-6.8	4.5-5.5	0.960	0.605	0.962	0.990
	6.9-7.6	0.743	0.714	0.024	0.627
	>7.6	0.001	0.761	0.000	0.648
6.9-7.6	4.5-5.5	0.434	0.114	0.005	0.433
	5.6-6.8	0.743	0.741	0.024	0.627
	>7.6	0.034	0.591	0.026	1.000
>7.6	4.5-5.5	0.000	0.761	0.000	0.454
	5.6-6.8	0.001	0.995	0.000	0.648
	6.9-7.6	0.034	0.591	0.026	1.000

DISCUSSION

In our present study there is statistical significant difference observed in the level of electrolytes, in Na and Cl with various levels of HbA1cgroups and no statistical significant difference was observed in the level of potassium and bicarbonatewhen compared with control and other groups of HbA1c. In the present study there is statistical significant decrease in sodium with levels of group 1, group2, group3,group -4 this suggest that glucose is osmotically active substance. Hyperglycemia increase serum osmolality, regulating in movement of water out of the cells and subsequently in reduction of serum sodium levels.[12,13] The lower level of serum sodium (Na^+) in diabetic patients is possibly due to a hyperglycemia induced intracellular sequestration of sodium combined with increase sodium (Na^+) excretion as a result of osmoregulatory response [14]. Some studies have also shown that hyponatremia is common in diabetes patients and is response for increased morbidity and mortality.[15,16,17]. Acute and symptomatic hyponatremia can lead to significant rates or morbidity and mortality.[18,19,20] In the present study alsothere is a statistical significance in lowering of chloride levels with various group of HbA1c. Chloride (cl^-) is mainly regulated by the intestinal duct and kidney.[21] In the stomach, chloride is secreted as a gastric fluid and enteric fluid and then reabsorbed throughout the intestinal duct, in kidney chloride is filtered at glomeruli and more than 99% is reabsorbed at the distal tubules. Thus the major cause of hypo-chloremiain diabetes mellitus (hyperglycemia) was considered to be due to loss of chloride through increased output of urine, which could be caused by the use of diuretics, loss of gastric fluid, and over volume of water.[22,23] Our results showed that there is no statistical significant difference was observed in potassium and bicarbonate levels. Some other studies have shown that there is alteration in potassium and bicarbonate levels in diabetic patients.[24,25,26, 27] in contrast to those studies, over all analysis of our study shown that there is no significant variation in potassium and bicarbonates levels with various HbA1c levels in diabetes mellitus. Some studies shown there is decreased in Bicarbonate levels this may due to, in hyper glycemia and ketoacidosis over production of β-hydroxy butyric acid and acetoacetic acid are dissociate completely and the excess hydrogen ionsbind with bicarbonates, resulting in decreased bicarbonate levels. Some studies shown that there is decreased in potassium levels in diabetes.[28, 29,30]known as hypokalemia. This may due to inadequate intake of K^+ but is unlikely to be only cause, increased net loss of fluids. However, dietary K^+ restriction may exacerbate the hypokalemia. And some other studies shown there is Increased in potassium levels in diabetes patients known as hyperkalemia[26,31] this may be due to insulin deficiency and hypertonicity due to hyperglycemia promotr K^+ shift from intracellular fluid to extracellular fluid.

CONCLUSION

Electrolyte abnormalities are common in diabetic patients and may be associated with increased Morbidity and mortality. These disturbance are particularly common in decompensated diabetes mellitus. As diabetic considered as systemic disease, more awareness should be planned to conduct, include monitoring clinical parameters and treatment to maintain healthy state beneath normal.

REFERENCES

1. Kittabachi A.E, Umpierre, G.E; Murphy, R.A: Hyperglycemic crisis in adults patients with Diabetes A consensus, statement from the American diabetic association: diabetes care.2006;29,27,39-2748 [cross Ref] [Pub Med]

2. Diabetic fact sheet.N^0 3/2 WHO. October 2013. Archived from (The Original) on 26 august 2013 retrived 25th March 2014
3. Goldberg, A. Hammerman, H. Petcheerski, S.Nassar, M. Zdorovyak A. Yalonestsky: Et.al. Hyponatremia and long term mortality in survivors of acute ST- Evaluation mayo cardialinfaraction: Arch intern Med 2006;166(7);781-6
4. Krauss RM, Eckel RH, Howard B: et al. AHA dietary guidelines. Revision 2000: A Statement for health care professionals from the nutrition committee of the American heart association. Circulation.2000;102;2284-99
5. Joshi S.R: Management of obes Indian patients: Indian journals of obesity. 2005; I (1) ;11-20.
6. Lee C.T Guo HR, Chen J B: cy Hyponatremia in the emergency department A.M..J emerg med. 2000;18;268[Pub Med]
7. Lobo DN: fluid electrolytes and nutrition: physiological and clinical aspects: ProcNutrsoc; 2004;63(3):453-466
8. Husain. f. Arifmann, M; Sheikh, M.A Nawaz. H.H Jamil, A trace element status in type-2 biabetes. Bangladesh;j MED .SC. 2009;8,52-56[Cross Ref]
9. Larry Arm strong –Electrolytes imbalance in diabetes-Live strong.com
10. Yanprevalg W. Lu J,Weng J, Jia W, Ji L, Xiao J,Shan Z, Liu J ,Tian H, Ji Q: et al.Ence of diabetes among men women in china: New England MED.2010;362
11. Villegas R, Gao YT, Dai Q, Yang G, Cai H, Li H, Zheng W, Shu XO: Dietary calcium and magnesium intake and the risk of type-2 diabetes: The shanghai women's health study: AM J Clinnutr. 2009;89(4): 1059-1067.10.3945/ajcn.2008.27182.PubMed
12. Grun Feld C, Chappell DA: Hypokalemia and D:AJM.1983;75(4):553-4
13. Sotirakopouls N, Kalogiannidou I, Teris M, Armentzioiou K, Sivridis D, M avromatidis KI: Acid-base and electrolyte disorder in patients with DM, Saudi journal of kidney diseases and transplantation: Saudi J kidney Dis Transpl.2012;23(1):58-62
14. M C Nair P, Madsbad S, christainsen MS, Transol I:hyponatremia and hyperkalemia in relation to hyperglycemia in insulin related diabetic out-patients:ClinchemActa. 1982;120:243-250
15. Anderson RJ, Chung HM, Kluge R, Schrier RW. Hyponatremia: a prospective analysis of its epidemiology and pathogenetic role of vasopessin: Ann intern Med.1995;102:164-68
16. Chung HM, Kluge R, Schrier RW, Anderson RJ: Postoperative hyponatremia. A prospective study: Arch inter Med.1986;146:333-36
17. Areiff AI. Acid-base, electrolyte and metabolic abnormalities. In: Parrillo JE, Dellinger RP, editor. Critical care Medicine: Priniciples of diagnosis and management in the Adult.2.St.Louis:Mosby;2002.pp.1169-1203
18. Arieff AI:Hyponatremia, convulsions, respiratory arrest and permanent brain damage after elective surgery in health women: N Engl J Med. 1986;314:1592-35
19. Arieff AI,Ayus JC, Fraser CL:Hyponatrmia and death or permanent brain demage in healthy children: BMJ.1992;304:1281-22
20. Ayus JC, Wheeler JM, Arieff AI: Postoperative hyponatremic encephalopathy in menstruent women: Ann intern Med. 1992;117:891-7
21. Yunos NM, Bellomo R, Story D, Kellum J:Bench -to beside review of chloride in critical illness crit care 2010; 14:226
22. Terzano C, Distefano F, Conti V, Di Nicola M, Paole G, Petoroianni A, Ricci A: Mixed acid-base disorders, hydro electrolyte imbalance and lactate production in hyper kapnic respiratory failure:the role of non invasive respiration:PLosone. 2012;7:e35245
23. Tani M, Morimatsu H, Takatsu F, Monita K:Theincidence and prognostic value of hypochloremia in crcitacally ill patient:Sciworld J. 2012;474185
24. Menon V U, Kumar K V, Gilchrist A, Sugathan N T, Sundaram R K, Vasantha Nair: et al. prevalance of known and undetected diabetes and associated risk factors in Central Kerala-ADEPS: Diabetes Res Clin Pract.Dec2006;74(3):287-292
25. Palmer BF. Managing hyperkalemia caused by inhibitors of the rennin-angiotensin-aldosterone system.NEngl J Med.2004;351:585-592
26. Uribarri J,Oh MS, Carroll HJ: Hyperkalemia in diabetes mellitus: J Diabet complications.1990;4(1):3-7
27. Raphael KL, Murhy RA, Shlipak MG, Satterfield S, Huston HK,Sebastien A, Sellmeyer DE, Patel KV, Newman AB, Samak MJ: et al. Bicarbonate Concentration,acid-base status, and mortality in the health, aging, and body composition study:Clin J Am Soc Nephrol.2016;11:308-16
28. Wang, S.; Hou,X.H.; Lu,h.J.; Wei L.;Bao Y.Q.;Jia,W.P:Serum electrolyte levels in relation to macrovascular complications in Chinese patients with diabetes mellitus.Cardiovasc: Diabetol.2013;12.[CrossRef] [PubMed]
29. Haglin,L,Backman,L, Tornkvist,B: A Structural equation model for assessment of link between changes in serum triglycerides,urate and glucose and and changes is serum calcium, magnesium and phosphate in type 2 diabetes and non diabetes metabolism:Cardivasc: Diabetol.2011;10.[CrossRef] [PubMed]
30. Van Style DD, Serdroy j: Studies on diabetes and electrolyte equilibrium in blood: J Biol Chem1928;18:20-26
31. Linder G, Funk GC: Hypernatremia in critically ill patients: J Crit Care. 28,2013, 216.e11-216.e20.[PMID:22762930 DOI:10.1016/j.jcrc.2012.05.001]

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