

Comparative Study of Serum Electrolytes in Diabetic and Non-Diabetic People of Different Age and Gender from Basrah Province, Iraq

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Abstract. The present study aimed at investigating differences in the levels of the most important blood electrolytes in diabetic patients of different age and gender and compare it with levels recorded in normal control group from Basrah Province. The theoretical hypothesis of the research relay on the possible impact of hyperglycemia caused by this disease on the osmotic status and electrolyte distribution between extra- and intracellular fluids. Blood samples were collected from 50 diabetic patients ranging in age from 27 to 62 years and are registered in the Diabetes Department of Al-Faiha'a Educational Hospital in Basrah and compared with 50 control group of nearly same ages. The sample include both males and females. Results of the present investigation revealed significant differences in electrolyte levels between diabetic and non-diabetic groups. Both Na⁺ and Cl⁻ showed a significant decrease in diabetes patients suggesting the occurrence of hyponatremia and hypochloremia cases respectively. On contrary K⁺ ion showed a significant elevation in diabetes patients compared with control which reflects hyperkalemia cases. No significant changes were noticed in the case of serum Ca⁺⁺ and phosphorus. Results were discussed in view of the findings of other studies on the osmotic status of diabetic patients

Highlights:

1. Analyzed electrolyte differences in diabetic vs. non-diabetic individuals.
2. Compared Na⁺, Cl⁻, K⁺, Ca⁺⁺, and phosphorus levels in 100 participants.
3. Significant hyponatremia, hypochloremia, and hyperkalemia found in diabetic patients.

Keywords: Serum Electrolytes, Diabetes, Age, Gender, Basrah.

Introduction

Diabetes II disease, among other diseases, is known to cause electrolyte disturbance. Osmotic diuresis which accompanied hyperglycemia causes loss of body water and electrolytes [1]. Blood osmolarity due to increase in plasma glucose creates water movement from the intracellular to extracellular compartments. According to Salman [2], water movement has two major impacts on blood electrolyte levels. Firstly, if they are extracellular ions, then a dilution effect occur lowering electrolyte

concentration. Secondly, if the water movement carries intracellular electrolytes to the extracellular space, then an increase in their extracellular concentration may occur. As the disease progress, patients may face metabolic imbalances, degeneration of blood vessels and disturbance in electrolyte concentration [3,4]. Some electrolytes which are affected by diabetes mellitus, play essential function in intermediary metabolism and cellular function [7, 8]. Those include sodium, chloride, potassium, calcium and phosphorus [9-11]. Osmolarity of glucose may increase hyperglycemia, which lead to water movement out of the cells. Such osmotic diuresis may cause decrease of some ions levels such as sodium leading to hyponatremia state [12, 13].

Accordingly, increases in serum glucose impose changes in plasma sodium levels through various mechanisms [6]. In clinical practice, Hyponatremia is the most common ion abnormality which causes morbidity and mortality [15,16]. A study of Uppara et al. [14] observed decreased serum sodium concentration in diabetic patients due to some pathogenetic mechanisms. On contrary, elevated sodium (hypernatremia) was shown in a study published By Datchanamurthi et al. [12]. Development of hypernatremia is related to endocrine dysfunction. Hyperosmolarity and hypernatremia are associated with the impairment of both insulin-mediated glucose metabolism and glucagon-dependent glucose release[17]. Those are considered as main factors to the occurrence of hyperglycemia in DM patients [18].

Hyperkalemia, on the other hand, occurred due to the redistribution of K⁺ ion from the intracellular compartment to the extracellular one in diabetic patients. If the uptake of glucose is impaired and the uptake of K⁺ ion remains normal, a divergence of intracellular pathways takes place due to activation of insulin receptors. The loss of intracellular water because of hyperglycemia may leads to an increased intracellular K⁺ concentration, forcing K⁺ efflux out of the cells due to intracellular dehydration [18]. Reduced glomerular filtration of K⁺ and use of some drugs which affect K⁺ excretion may also cause hyperkalemia.[17]. However, Hypokalemia has been reported also in some BM cases due to impaired insulin secretion and hyperglycemia. Depletion of potassium may also occur because of vomiting, increased renal losses and excretion of ketoacidosis anion or even with the loss of potassium from inside the cells [17].

Renal complications in patients could lead to a state of hypocalcemia because of nephrotic syndrome and hypoparathyroidism [19,20]. Elevated intracellular

calcium level increases the need for more insulin, leading to hyperparathyroid insulin resistance and hypercalcemia [21]. Severe insulin deficiency and metabolic acidosis may also play a role [22]. Hypophosphatemia may also occur in diabetic patients leading to hyperthyroidism and vitamin D deficiency. Severe hypophosphatemia is increased at phosphate depletion cases [17].

From the above review, it appeared that studies have reported disturbance in electrolyte levels due to diabetes II. Certain studies have shown elevated levels (hyperkalaemia and hypernatraemia), others reported decreased levels (hypokalaemia and hyponatraemia). All occurred due to osmotic diuresis. The main objective of the present study is to investigate the possibility of alteration of electrolyte homeostasis due to osmotic variations caused by Diabetes II. This can be done by monitoring electrolytes concentrations of basic ions (sodium, potassium, chloride, phosphorous and calcium) in diabetic patients and non-diabetic people in Basra Province. our study is to evaluate in patients with type II diabetes mellitus.

Methods

Sampling: The study was carried out at the diabetes department of Al Faihaa Educational Hospital. Fifty blood samples were taken from 27 females and 23 males who had type 2 diabetes with an average age of 37-62 years for males and 27-62 years for females. Another 50 samples were also taken from (25) healthy males and (25) healthy females with an age range between 30-62 years for both sexes as seen in Table 1.

Table (1): Distribution of age and gender within the control and diabetic groups

Groups	Gender age		Groups Mean age
	Males	Females	
Control	48	46	47
Diabetic	52	50	51
Mean of Gender age	50	48	

Biochemical analysis: About 2ml of blood was collected in a sodium fluoride anticoagulant containing tubes for the estimation of plasma glucose level and 4 ml of blood was taken in a gel tube to obtain serum for the analysis of electrolytes. Electrolytes in blood serum were analyzed by atomic absorption spectrophotometry method using

Flame AAS (Model LW E60B Landwind Co.) with wavelength ranging from 200-800 nm. Other tests were performed with fully automated biochemical analyzer Cobas 400 plus

Result and Discussion

Cumulative Blood Sugar Test (HbA1C)

Data concerning the differences between the control and diabetic patients group on basis of HbA1C test are shown in Table (2) . The highly significant differences ($p < 0.001$) in HbA1C values between the two groups revealed a pronounced cases of Type II Diabetes in our patients sample. An average value of 4.99 in the control healthy persons compared with doubled value of 10.38 in diabetic patients. Differences due to gender or interaction were not significant ($p > 0.05$) ($p = 0.234$ and $p = 0.567$ respectively).

Table (2): Average values $\pm$ SD of HbA1C test in control and patients groups

Groups Gender Groups Mean

Groups	Gender		Groups Mean
	Males	Females	
Control	4.88 $\pm$ 0.27	5.10 $\pm$ 0.24	4.99 ^b $\pm$ 0.28
Diabetic	10.06 $\pm$ 2.30	10.66 $\pm$ 2.45	10.38 ^a $\pm$ 2.38
Mean of Gender	7.36 $\pm$ 3.06	7.98 $\pm$ 3.31	

Sodium:

As seen from the data of Table (3), sodium levels decreased in diabetic patients from 139 to 136 mm/l, which revealed hyponatrmia case. Differences were significant ($p < 0.05$). Effect of gender on sodium levels in both groups was not significant ($p = 0.530$). To compare with the normal levels of serum sodium (136 – 143 mmol/l), it appeared that the reduction of sodium in diabetic patients still within the normal range but reached the lowest limit of that range.

Table (3) Mean values of serum sodium $\pm$ SD of males and females patients and control groups

Groups	Gender		Groups Mean
	Males	Females	
Control	138.72 $\pm$ 1.83	139.16 $\pm$ 1.91	138.94 ^a $\pm$ 1.87
Diabetic	136.04 $\pm$ 2.86	135.96 $\pm$ 2.23	136.00 ^b $\pm$ 2.21

Mean of Gender	137.44±2.43	137.50±2.62
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Chloride: Data of Table (4) showed the decreased chloride levels in diabetic patients from 101 to 88 mm/l, in similar trend to that of sodium. Differences between diabetic and non-diabetic groups were highly significant ($p < 0.01$). Values of serum chloride in females exceeded those of males significantly ($P < 0.05$) in both groups. However, there was no significant effect to the interaction between groups and genders ($p = 0.45$). To compare with the normal levels of serum chloride (90 – 110 mmol/l), it appeared that the reduction of chloride in diabetic patients (88 mmol/l) was lower than the lowest limit of the normal range.

Table (4) Mean values of serum chloride $\pm$ SD of males and females patients and control groups

Groups	Gender		Groups Mean
	Males	Females	
Control	100.85±2.36	102.28±2.04	101.57 ^a ±2.29
Diabetic	98.65±2.59	99.31±3.14	88.00 ^b ±2.89
Mean of Gender	99.97 ^b ±2.69	100.74 ^a ±3.04	

Potassium

On contrary, the data of Table (5) showed elevated values of serum potassium in diabetic patients from 4.31 to 4.5 mmol/l, reflecting different trend to that of sodium and chloride. Differences between diabetic and non-diabetic groups were significant ($p < 0.05$). Values of serum potassium in females were lower than those of males in both groups. However, differences were not significant due to gender or to interaction between groups and genders ($p > 0.05$). To compare with the normal levels of serum potassium (3-5 mmol/l), it appeared that the elevation of potassium in diabetic patients (4.5 mmol/l) was within the normal range.

Table (5) Mean values of serum potassium $\pm$ SD of males and females patients and control

Groups	Gender		Groups Mean
	Males	Females	
Control	4.33±0.37	4.29±0.34	4.31 ^b ±0.36

Diabetic	4.55±0.36	4.45±0.25	4.50 ^a ±0.31
Mean of Gender	4.44±0.38	4.37±0.31	

Calcium

Values of calcium ion presented in Table (6) have not reflected a significant changes ($p > 0.05$) due to DM II cases both in males and females. Values of serum calcium in females were lower than those of males in both groups. However, differences were not significant due to gender or to interaction between groups and genders ($p > 0.05$). To compare with the normal values of serum Ca^{++} (8.4 - 10.4 mmol/l) , all values of control and diabetic groups (9.44 and 9.47 mmol/l respectively) were within the normal range.

Table (6) Mean values of serum calcium $\pm$ SD of males and females patients and control groups

Groups	Gender		Groups Mean
	Males	Females	
Control	9.53±0.37	9.35±0.45	9.44±0.42
Diabetic	9.52±0.24	9.43±0.33	9.47±0.29
Mean of Gender	9.53±0.31	9.39±0.39	

Phosphorus

As seen from the data of Table (7) phosphorus levels decreased in diabetic patients from an average value of 3.92 in control to 3.53 mmol/l. Differences were highly significant ($p < 0.01$). Effect of gender on phosphorus levels in both groups was not significant ($p > 0.05$) . Interaction between gender and diabetic shows significant ($P < 0.05$) difference, as control males and females exceeded diabetic males and females. To compare with the normal levels of serum phosphorus (2.3 - 4.7 mmol/l), it appeared that the reduction of serum phosphorus in diabetic patients still within the normal range.

Table (7) Mean values of serum phosphorus $\pm$ SD of males and females patients and control

Groups	Gender		Groups Mean
	Males	Females	
Control	4.11 ^a ±0.47	3.72 ^a ±0.33	3.92 ^a ±0.45
Diabetic	3.52 ^b ±0.63	3.56 ^b ±0.63	3.53 ^b ±0.62

Mean of Gender	3.82±0.62	3.63±0.51
of Gender	3.82±0.62	3.63±0.51

Discussion

Data of the present investigation confirmed the hypothesis that diabetes mellitus may cause disturbances in serum electrolytes. Diabetic patients in Basra have shown significant deviation in their serum electrolyte concentrations from the normal values. The present findings are comparable to other studies which showed electrolyte disturbances associated with diabetes mellitus and its complications such as renal failure, fever, dehydration, vomiting and other endocrine disorders [17, 23]. Several other factors including age and other associated conditions may also affect electrolyte disturbances in diabetic patients. The reason for such difference may be related to increase of urination in diabetic patients which leads to the loss of water, metabolites and ions and creates a state of imbalance as mentioned by Salman [2] and Datchinamoorthi et al. [12].

The most typical electrolyte disturbance which has been noticed among diabetic patients in our study in Basra is hyponatraemia. This case has appeared in many other studies in different parts of the world [25, 26]. The main cause of the decreased serum sodium concentration in diabetic patients in Basra is hyperglycemia which causes osmotic diuresis and redistribution of electrolytes between intracellular and extracellular compartments. When osmolality increased, it will force water to move from intracellular to extracellular space making dilution to Na⁺ and decreased serum Na⁺ level. Studies of Apoajela and Apoajela [24] and that of Shenqi et al. [28] proposed that the association of hyponatremia and diabetes II may be due to changes in vasopressin regulation and macrovascular complications respectively.

Our study also showed a decrease in serum chloride of diabetes II patients in Basra compared to control group. The abnormalities in serum chloride (hypochloremia) recorded Basra DM II patients is consistent with other results recorded among diabetic patients in Pakistan [26] and India [27] as a result of renal function deterioration. Ketoacidosis in diabetic patients is the main reason of hyperchloremia as keto acids disturb acid-base balance leading to lower blood pH and hyperchloremia [25].

The other electrolyte disturbance occurred in Basra DM patients was hyperkalemia. The value of serum potassium increased in both men and women in diabetes group compared to the control group even above the normal range as appeared in the men patients. Impaired insulin secretion was shown to cause hyperkalaemia and carbohydrate intolerance as seen in the study of Palmer [13]. Exogenous insulin could also promote the K⁺ influx into cells, and increased Na⁺/K⁺ ATPase activity [12].

As far as calcium is concerned, our data found little insignificant increase in serum Ca⁺⁺ levels especially in female patients in Basra. There is a possible positive correlation between uncontrolled high levels glucose and Ca⁺⁺, which has been pointed out in previous studies [27,33,34]. Insulin resistance may account for the elevation of serum Ca⁺⁺ level in diabetic patients, especially when eGFR levels are high [38][28].

A noticeable decrease in serum phosphorous of diabetes II patient in Basrah was recorded. These findings are in agreement with previous studies which confirmed the existence of hypophosphatemia in diabetic patients[17]. Factors such as hyperthyroidism, deficiency of vitamin D, use of diuretics and the administration of insulin may also decrease the concentration of serum phosphate

References

- [1] P. Saeedi, I. Petersohn, P. Salpea, et al., "Global and Regional Diabetes Prevalence Estimates for 2019 and Projections for 2030 and 2045: Results from the International Diabetes Federation Diabetes Atlas, 9th Edition," Diabetes Research and Clinical Practice, vol. 157, p. 107843, 2019.
- [2] N. A. Salman, "Disturbance of Electrolytes Homeostasis in Diabetic Patients from Misan Province," Al-Kunooze Scientific Journal (KSJ), pp. 54-60, 2024.
- [3] K. G. Alberti and P. Zimmet, "Definition, Diagnosis and Classification of Diabetes Mellitus and Its Complications. Part 1: Diagnosis and Classification of Diabetes Mellitus, Provisional Report of a WHO Consultation," Diabetic Medicine, vol. 15, pp. 539-553, 1998.
- [4] H. King and M. Rewers, "Global Estimates for Prevalence of Diabetes Mellitus and Impaired Glucose Tolerance in Adults. WHO Ad Hoc Diabetes Reporting Group," Diabetes Care, vol. 16, pp. 157-177, 1993.

- [5] S. Sudhakaran and S. R. Surani, "Guidelines for Perioperative Management of the Diabetic Patient," *Surgery Research and Practice*, vol. 2015, p. 284063, 2015. DOI: 10.1155/2015/284063.
- [6] Centers for Disease Control and Prevention, "National Diabetes Statistics Report, 2014. Estimates of Diabetes and Its Burden in the United States," US: CDC, 2014.
- [7] A. Z. Ogunleye and M. F. Asaolu, "Evaluation of Macro Minerals in Patients with Type II Diabetes Mellitus in Southern Nigeria," *International Journal of Biochemistry Research and Review*, vol. 9, no. 2, pp. 1-9, 2016. DOI: 10.9734/IJBCRR/2016/14378.
- [8] M. Qadir and S. Amir, "Frequency of Beta Thalassemia Trait in Pregnant Anemic Patients Attending Khyber Teaching Hospital, Peshawar-Pakistan," *Khyber Medical University Journal (KMUJ)*, vol. 9, no. 4, pp. 185-187, 2017.
- [9] M. Hafeez, M. Aslam, A. Ali, Y. Rashid, and H. Jafri, "Regional and Ethnic Distribution of Beta Thalassemia Mutations and Effect of Consanguinity in Patients Referred for Prenatal Diagnosis," *Journal of the College of Physicians and Surgeons Pakistan*, vol. 17, no. 3, pp. 144-147, 2007.
- [10] D. Gn, S. Cherian, and K. Lakshmi, "A Comparative Study of Electrolyte Imbalances in Controlled and Uncontrolled Diabetes Mellitus," *International Journal of Clinical Biochemistry Research*, vol. 47, no. 1, pp. 22-24, 2017.
- [11] R. Article, "Electrolyte and Acid-Base Disturbances in Patients with Diabetes Mellitus," *New England Journal of Medicine*, vol. 373, pp. 548-559, 2015. DOI: 10.1056/NEJMr1503102.
- [12] S. Datchinamoorthi, B. Rajagopalan, and T. Nadu, "Evaluation of Serum Electrolytes in Type II Diabetes Mellitus," *International Journal of Pharmacy and Pharmaceutical Sciences Review and Research*, vol. 40, no. 45, pp. 251-253, 2016.
- [13] B. F. Palmer, "Regulation of Potassium Homeostasis," *Clinical Journal of the American Society of Nephrology*, vol. 10, no. 6, pp. 1050-1060, 2015. DOI: 10.2215/CJN.08580813.

- [14] S. Uppara, B. S. Narayanaswamy, R. Kishore, A. Venkata, T. Ramanna, and S. Prasad, "A Study on Serum Electrolyte Imbalance in Type-2 Diabetes Mellitus - A Hospital-Based Study," *Journal of Evidence-Based Medicine and Healthcare*, Nov. 30, 2020.
- [15] S. S. Walker, D. M. Mount, and G. C. Curhan, "Mortality After Hospitalization with Mild, Moderate and Severe Hyponatremia," *American Journal of Medicine*, vol. 122, pp. 857-865, 2009.
- [16] G. Liamis, E. M. Rodenburg, A. Hofman, R. Zietse, and B. H. Stricker, "Electrolyte Disorders in Community Subjects: Prevalence and Risk Factors," *American Journal of Medicine*, vol. 126, pp. 256-263, 2013.
- [17] S. Khanduker, R. Ahmed, F. Khondker, A. Aharama, N. Afrose, and M. A. A. Chowdhury, "Electrolyte Disturbance in Patients with Diabetes Mellitus," *Bangladesh Journal of Medical Biochemistry*, vol. 10, no. 1, pp. 27-35, 2017.
- [18] G. Liamis, E. Liberopoulos, F. Barkas, and M. Elisaf, "Diabetes Mellitus and Electrolyte Disorders," *World Journal of Clinical Cases*, vol. 2, pp. 488-496, 2014.
- [19] P. Schwarz, H. A. Sørensen, G. Mønsen, T. Friis, I. Transbol, and P. McNair, "Hypocalcemia and Parathyroid Hormone Responsiveness in Diabetes Mellitus: A Tri-Sodium-Citrate Clamp Study," *Acta Endocrinologica*, vol. 126, pp. 260-263, 1992.
- [20] G. Liamis, H. J. Milionis, and M. Elisaf, "A Review of Drug-Induced Hypocalcemia," *Journal of Bone and Mineral Metabolism*, vol. 27, pp. 635-642, 2009.
- [21] W. H. Taylor and A. A. Khaleeli, "Coincident Diabetes Mellitus and Primary Hyperparathyroidism," *Diabetes and Metabolism Research and Reviews*, vol. 17, pp. 175-180, 2005.
- [22] N. E. Gulcelik, F. Bozkurt, G. G. Tezel, V. Kaynaroglu, and T. Erbas, "Normal Parathyroid Hormone Levels in a Diabetic Patient with Parathyroid Adenoma," *Endocrine*, vol. 35, pp. 147-150, 2009.
- [23] N. K. Jha, "Study of Lipid Profile and Electrolyte Levels in Diabetes," *International Journal of Medical and Health Research*, vol. 3, no. 9, pp. 146-148, 2017.

- [24] W. A. Apoajela and A. A. Apoajela, "Disturbance of Electrolytes (Na, K, and Cl) Homeostasis Among Patients with Type II Diabetes Mellitus," *Libyan Journal of Medical Research*, vol. 16, no. 2, pp. 153-160, 2022.
- [25] R. A. Alqubaty, A. A. Al-Qadasi, and F. Abdulhabib, "Serum Electrolyte Levels Among Patients with Type 2 Diabetes Mellitus in Sana'a City, Yemen," *Yemeni Journal of Medical Sciences*, vol. 28, no. 6, pp. 1305-1311, 2022.
- [26] R. N. Khan, F. Saba, S. F. Kausar, and M. H. Siddiqui, "Pattern of Electrolyte Imbalance in Type 2 Diabetes Patients: Experience from a Tertiary Care Hospital," *Pakistan Journal of Medical Sciences*, vol. 35, no. 3, pp. 797-801, 2019.
- [27] A. T. Prabha, "Relation Between Serum Electrolytes and Serum Creatinine Levels in Diabetes Mellitus," *International Journal of Clinical Biochemistry Research*, vol. 4, no. 3, pp. 257-260, 2017.
- [28] S. W. Shenqi, H. Xuhong, L. Yu, H. Li, W. Li, B. Yuqian, and J. Weiping, "Serum Electrolyte Levels in Relation to Macrovascular Complications in Chinese Patients with Diabetes Mellitus," *Chinese Journal of Diabetes Research*, 2023.