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Original Research Article

Pattern of Serum Electrolytes Imbalance among Type 2 Diabetes Mellitus Patients in a Tertiary Hospital in North-Eastern Nigeria

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Abstract: ***Background***: Electrolyte abnormalities are common metabolic disturbances among patients with type 2 diabetes mellitus (T2DM). Hyperglycemia, osmotic diuresis, altered insulin action, gastrointestinal losses, renal dysfunction and medications may contribute to disturbances in sodium, potassium, chloride and bicarbonate concentrations. Despite their potential clinical significance, routine assessment of serum electrolytes may receive inadequate attention, particularly in resource-limited settings. ***Objective***: To determine the prevalence and pattern of serum electrolyte imbalance among patients with T2DM in North-Eastern Nigeria. ***Methods:*** This descriptive cross-sectional study was conducted at the Chemical Pathology Department of a tertiary hospital in North-Eastern Nigeria between January and June 2025. One hundred (100) confirmed T2DM patients and fifty (50) apparently healthy non-diabetic controls were recruited. Serum sodium, potassium and chloride were measured using ion-selective electrode methods, while bicarbonate was measured by an enzymatic method. Data were analysed using SPSS version 25.0 and presented as mean ± standard deviation and percentages. ***Results***: The mean age of participants was 54.2 ± 10.1 years. Among T2DM patients, 58% were male and 42% were female. The mean duration of diabetes was 6.8 ± 4.2 years. At least one electrolyte abnormality was present in 68% of T2DM patients. Hyponatremia was the commonest abnormality (22%), followed by hypokalaemia (18%), hypochloraemia (15%), hyperkalaemia (8%) and hypernatremia (5%). Mean serum Na+, K+, Cl− and HCO3− were lower in T2DM patients than controls: 132.5 ± 4.2 vs 138.4 ± 2.8 mmol/L; 3.6 ± 0.5 vs 4.2 ± 0.4 mmol/L; 99.2 ± 3.8 vs 104.5 ± 2.9 mmol/L; and 22.1 ± 2.1 vs 24.8 ± 1.8 mmol/L, respectively. ***Conclusion***: Electrolyte imbalance was common among T2DM patients in this study, with hyponatraemia and hypokalaemia being the predominant abnormalities. Routine electrolyte monitoring should be considered as part of comprehensive diabetes care, especially in patients at risk of fluid, renal or medication-related disturbances.

Keywords: Chemical Pathology, Type 2 Diabetes Mellitus, Electrolyte Imbalance, Hyponatraemia, Hypokalaemia, North-Eastern Nigeria.

INTRODUCTION

Diabetes mellitus is a chronic metabolic disorder characterized by persistent hyperglycaemia resulting from defects in insulin secretion, insulin action, or both [1, 2]. Type 2 diabetes mellitus (T2DM) is the most common form and represents an increasing public health burden globally and in sub-Saharan Africa including Nigeria [3-5].

Electrolytes such as sodium, potassium and chloride play essential roles in maintaining extracellular fluid volume, cellular function, neuromuscular excitability and acid–base balance [6-8]. Bicarbonate is a major component of the extracellular buffering system and is an important laboratory marker in the assessment of metabolic acid–base status [9, 10].

Patients with diabetes are susceptible to disturbances of fluid and electrolyte homeostasis [11]. Hyperglycaemia increases extracellular osmolality and promotes movement of water from the intracellular to extracellular compartment. Severe hyperglycaemia may also cause osmotic diuresis, resulting in urinary losses of water and electrolytes [12-14].

Hyponatraemia may occur in hyperglycaemia because water shifts into the extracellular space,

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producing dilution of measured sodium [15]. In addition, osmotic diuresis may increase urinary sodium and water losses. Potassium abnormalities may arise from altered insulin action, osmotic diuresis, renal losses and treatment effects. Bicarbonate may be reduced in association with metabolic acidosis [16].

Electrolyte abnormalities can have important clinical consequences, including neuromuscular dysfunction, cardiac arrhythmias, impaired consciousness and increased morbidity. Early detection is therefore relevant to comprehensive diabetes management [17, 18].

Previous studies in Nigeria and elsewhere have reported electrolyte abnormalities among people with diabetes, but data from North-Eastern Nigeria remain limited. This study was therefore undertaken to determine the prevalence and pattern of serum electrolyte abnormalities among T2DM patients attending a tertiary hospital in North-Eastern Nigeria [19-21].

MATERIALS AND METHODS

Study Design and Setting:

This was a descriptive cross-sectional study conducted at the Chemical Pathology Department of a tertiary hospital in North-Eastern Nigeria between January and June 2024.

Study Population:

One hundred (100) confirmed T2DM patients attending the Medical Outpatient Clinic and fifty (50) apparently healthy non-diabetic controls were consecutively recruited. The total study population was therefore 150 participants.

Inclusion Criteria: Known T2DM for more than one year and age 30 years or above.

Exclusion Criteria:

Patients with diabetic ketoacidosis, chronic kidney disease (eGFR <60 mL/min/1.73 m²), current diuretic therapy, or vomiting/diarrhoea within the preceding two weeks were excluded [22,23].

Ethical Consideration:

Ethical clearance was obtained from the Hospital Research and Ethics Committee. Written informed consent was obtained from all participants and confidentiality was maintained.

Sample Collection and Analysis:

Five millilitres of venous blood were collected, allowed to clot and centrifuged at approximately 3000 rpm for five minutes. Serum was separated. Sodium, potassium and chloride were measured using the direct ion-selective electrode (ISE) principle [24, 25]. Bicarbonate was measured using an enzymatic method [26]. Internal quality control was performed before and during analysis.

Reference ranges used in the original study were sodium 135–145 mmol/L, potassium 3.5–5.0 mmol/L, chloride 95–110 mmol/L and bicarbonate 20–30 mmol/L. Laboratory-specific validated intervals should be used for final clinical interpretation.

Data Analysis:

Data were entered into SPSS version 25.0. Continuous variables were presented as mean ± standard deviation and categorical variables as frequencies and percentages. The analysis was predominantly descriptive [27].

RESULTS

A total of 150 participants were studied, comprising 100 T2DM patients and 50 apparently healthy controls. The mean age of participants was 54.2 ± 10.1 years. Among T2DM patients, 58% were male and 42% were female. The mean duration of diabetes was 6.8 ± 4.2 years.

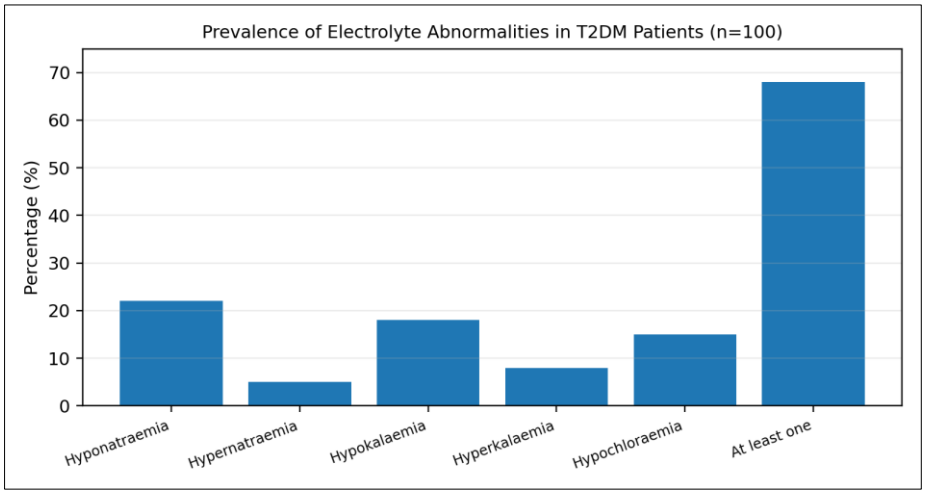


Figure 1: Prevalence of Electrolyte Abnormalities in T2DM Patients (n=100)

Table 1: Mean Serum Electrolyte Concentrations in T2DM Patients and Controls

Electrolyte	T2DM (n=100) Mean ± SD	Control (n=50) Mean ± SD
Na+ (mmol/L)	132.5 ± 4.2	138.4 ± 2.8
K+ (mmol/L)	3.6 ± 0.5	4.2 ± 0.4
Cl− (mmol/L)	99.2 ± 3.8	104.5 ± 2.9
HCO3− (mmol/L)	22.1 ± 2.1	24.8 ± 1.8

At least one electrolyte abnormality was present in 68% of T2DM patients. Hyponatraemia was the most frequent abnormality (22%), followed by hypokalaemia (18%), hypochloraemia (15%), hyperkalaemia (8%) and hypernatraemia (5%).

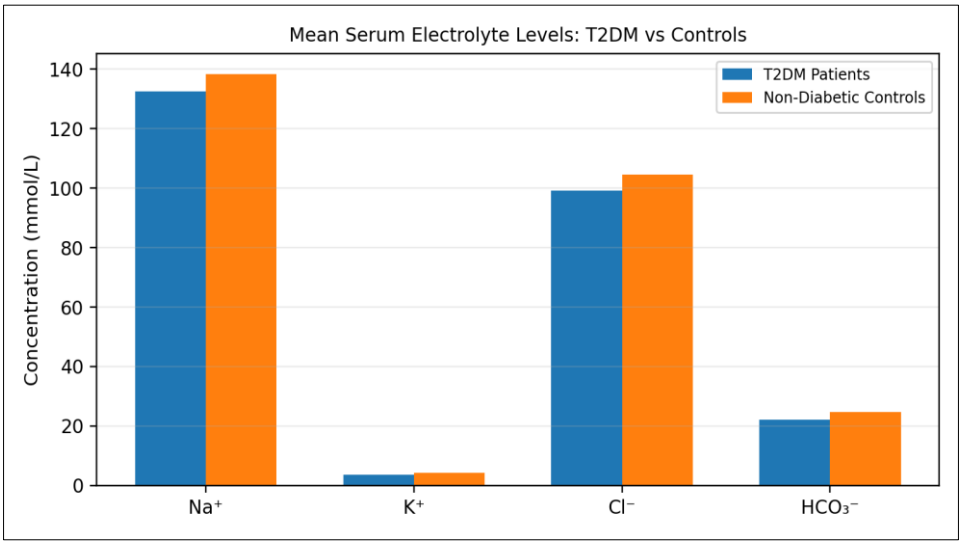


Figure 2: Mean Serum Electrolyte Levels: T2DM versus Non-Diabetic Controls

The mean concentrations of sodium, potassium, chloride and bicarbonate were lower in the T2DM group than in the non-diabetic control group.

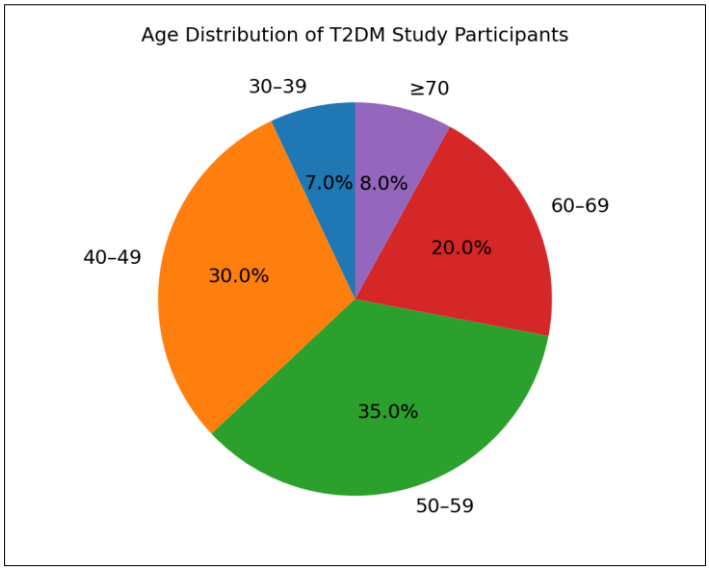


Figure 3: Age Distribution of T2DM Study Participants

DISCUSSION

The present study demonstrated a substantial burden of electrolyte abnormalities among patients with T2DM. At least one electrolyte abnormality was observed in 68% of diabetic participants, with hyponatraemia being the most frequent abnormality.

The predominance of hyponatraemia is biologically plausible because hyperglycaemia increases extracellular osmolality and causes movement of water from the intracellular to extracellular compartment. This can lower measured serum sodium concentration.

Osmotic diuresis may additionally produce urinary water and electrolyte losses [1-11].

Hypokalaemia occurred in 18% of participants. Diabetes may predispose to potassium depletion through osmotic diuresis and urinary potassium loss. Changes in insulin availability and insulin treatment also influence transcellular potassium distribution [14, 15].

Hyperkalaemia occurred in 8% of participants. Although potassium depletion may accompany osmotic diuresis, hyperkalaemia can occur when insulin deficiency, metabolic acidosis or impaired renal potassium excretion is present. Medication effects may also contribute [28, 29].

Hypochloraemia occurred in 15% of patients and may accompany sodium and water disturbances. The lower mean chloride concentration in T2DM patients compared with controls is consistent with an overall disturbance in extracellular fluid and electrolyte homeostasis.

The mean bicarbonate concentration was lower in the diabetic group. Because patients with diabetic ketoacidosis were excluded, the finding should not by itself be interpreted as diabetic ketoacidosis. Bicarbonate should be interpreted alongside glucose, ketones, renal function and, where clinically indicated, blood gas measurements [30, 31].

The findings support the importance of electrolyte assessment in diabetes care, particularly in patients with symptoms, poor glycaemic control, dehydration, renal impairment, gastrointestinal losses or medication exposure that can alter electrolyte balance [32, 33].

STRENGTHS AND LIMITATIONS

Strengths: The study provides local data from North-Eastern Nigeria and evaluates several clinically important electrolytes. The inclusion of a non-diabetic comparison group provides context for the observed mean electrolyte concentrations.

Limitations: This was a single-centre cross-sectional study, limiting generalisability and preventing causal inference. The study did not provide detailed analysis according to HbA1c, plasma glucose, body mass index, diabetes treatment, renal function or comorbidities. Future studies should incorporate these variables and appropriate inferential and multivariable statistical analyses.

CONCLUSION

Electrolyte abnormalities were common among patients with T2DM in this tertiary hospital in North-Eastern Nigeria. Hyponatraemia was the most frequent abnormality, followed by hypokalaemia and hypochloraemia. Routine assessment of serum electrolytes should be considered as part of comprehensive diabetes care, particularly in patients at increased risk of fluid, renal or medication-related disturbances.

RECOMMENDATIONS

Serum electrolyte assessment should be incorporated into the biochemical evaluation of selected patients with T2DM, especially those with poor glycaemic control or symptoms suggestive of electrolyte disturbance.

Patients with significant sodium or potassium abnormalities should undergo appropriate clinical assessment to identify and treat the underlying cause.

Serum sodium should be interpreted alongside plasma glucose, particularly in patients with marked hyperglycaemia.

Future studies should use larger multicentre populations and examine relationships between electrolyte abnormalities, HbA1c, fasting plasma glucose, duration of diabetes, renal function, medication use and diabetic complications.

REFERENCES

1. Palmer BF, Clegg DJ. Electrolyte and acid-base disturbances in patients with diabetes mellitus. N Engl J Med. 2015;373(6):548-559.
2. Liamis G, Liberopoulos E, Barkas F, Elisaf M. Diabetes mellitus and electrolyte disorders. World J Clin Cases. 2014;2(10):488-496.
3. Kitabchi AE, Umpierrez GE, Miles JM, Fisher JN. Hyperglycemic crises in adult patients with diabetes. Diabetes Care. 2009;32(7):1335-1343.
4. Datchinamoorthy S, Balakrishnan V, Rajagopalan B. Electrolyte imbalance in diabetes mellitus. Int J Adv Med. 2017;4(1):11-14.
5. Uloko AE, Musa BM, Ramalan MA, et al. Prevalence and risk factors for diabetes mellitus in Nigeria. Diabet Med. 2018;35(9):1307-1314.
6. Chinenye S, Ofoegbu EN. National diabetes survey. Diabetes Care Nigeria. 2012;2:1-8.
7. Burtis CA, Ashwood ER, Bruns DE. Tietz Textbook of Clinical Chemistry. 5th ed. Elsevier; 2012.
8. Marshall WJ, Lapsley M, Day A, Ayling R. Clinical Biochemistry. 3rd ed. Churchill Livingstone; 2014.
9. Khan RN, Saba F, Kausar S, Siddiqui MH. Pattern of electrolyte imbalance in Type 2 diabetics. Pak J Med Health Sci. 2019;13(2):356-359.
10. Hasona NA, Al-Swailmi FK. Assessment of electrolytes imbalance in type 2 diabetic patients. Biomed Res. 2016;27(2):456-460.
11. Hillier TA, Abbott RD, Barrett EJ. Hyponatremia: evaluating the correction factor for hyperglycemia. Am J Med. 1999;106(4):399-403.
12. Adrogue HJ, Lederer ED, Suki WN, Eknoyan G. Determinants of plasma potassium levels in diabetic ketoacidosis. Medicine. 1986;65(3):163-172.

13. Ogunleye OO, Aisuzu MC. Evaluation of serum electrolytes in diabetic patients in Ibadan. Afr J Med Health Sci. 2014;13(2):88-91.
14. Chukwuemeka C, Ofoegbu E. Electrolyte disturbances in Nigerian diabetics. J Coll Med. 2008;13(1):22-26.
15. Agoreyo FO, Onuegbu AJ. Electrolyte profile in diabetes mellitus in Benin. Niger J Clin Pract. 2010;13(2):165-168.
16. Araoye MO. Research Methodology with Statistics. 2nd ed. Nathadex; 2004.
17. World Health Organization. Definition and diagnosis of diabetes mellitus. Geneva: WHO; 2006.
18. Kovesdy CP. Management of hyperkalaemia in chronic kidney disease. Nat Rev Nephrol. 2014;10(11):653-662.
19. Liamis G, Rodenburg EM, Hofman A, Zietse R, Stricker BH, Hoorn EJ. Electrolyte disorders in community subjects. Am J Med. 2013;126(3):256-263.
20. Koch DD, Parrish D. Ion-selective electrode measurement of electrolytes. Clin Lab Med. 1990;10(1):57-73.
21. Forrester RL, Wataji LJ, Silverman DA, Pierre KJ. Enzymatic method for bicarbonate. Clin Chem. 1976;22(2):243-246.
22. Westgard JO, Barry PL. Cost-effective quality control. Clin Chem. 1986;32(8):1573-1575.
23. Pesce MA, Kaplan LA. Reference ranges. In: Clinical Chemistry. Mosby; 1987.
24. Kirkwood BR, Sterne JA. Essential Medical Statistics. 2nd ed. Blackwell; 2003.
25. Shalini R, Sujatha R. Electrolyte imbalance in diabetic patients. Natl J Lab Med. 2015;4(4):1-4.
26. Al-Rubeaan K, et al. Prevalence of electrolyte disturbances in diabetic patients in Saudi Arabia. Saudi Med J. 2010;31(4):410-415.
27. Liamis G, et al. Electrolyte disorders in diabetes mellitus. World J Diabetes. 2015;6(3):454-462.
28. Khan AH, Waseem S. Low serum magnesium and outcomes in diabetics. Biol Trace Elem Res. 2018;183:45-50.
29. Erasmus RT, Olukoga AO, Bojuwoye B, Adewoye H. Plasma electrolytes in Nigerian diabetics. Trop Geogr Med. 1987;39(2):175-178.
30. Ilebauchuchi NM, Anionwu CS, Onyebukwe DN. Electrolyte pattern in diabetes mellitus in Lagos. Nig Q J Hosp Med. 1998;8(1):33-35.
31. Katz MA. Hyperglycemia-induced hyponatremia: calculation of expected Na decrease. Am J Med. 1973;55(4):503-506.
32. Hollander-Rodriguez JC, Calvert JF. Hyperkalemia. Am Fam Physician. 2006;73(2):283-290.
33. Gennari FJ. Hypokalemia. N Engl J Med. 1998;339(7):451-458.