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Comparative electrolyte patterns in female type 2 diabetes mellitus patients

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Abstract
Diabetes mellitus is a prevalent metabolic disorder in which dysregulated insulin action and chronic hyperglycemia can disrupt electrolyte homeostasis, a core regulator of vital physiological processes. This study profiled serum electrolytes in 116 women 60 with type 2 diabetes mellitus (T2DM) and 56 apparently healthy controls. Ages ranged from 30-66 years in the T2DM group and 20-55 years among controls. Glycemic indices were markedly higher in T2DM, plasma glucose 187.83 ± 9.43 mg/dL vs 97.73 ± 6.30 mg/dL, and cumulative glycemia 12.83 ± 1.34 mg/dL vs 5.31 ± 0.25 mg/dL (controls). Among electrolytes, sodium was lower in T2DM while potassium and chloride were higher, though these differences were not statistically significant, sodium 120.02 ± 3.75 vs 141.27 ± 5.60 mmol/L, potassium 4.36 ± 0.61 vs 4.21 ± 0.31 mmol/L, and chloride 108.44 ± 9.25 vs 92.24 ± 10.77 mmol/L (T2DM vs control, respectively). In contrast, calcium was significantly reduced in the diabetic cohort (8.95 vs 10.65 mmol/L). In summary, women with T2DM in Maysan province exhibited poor glycemic control alongside electrolyte disturbance characterized chiefly by lower calcium levels compared with non-diabetic women.

Keywords: Sodium, potassium, chloride, calcium, diabetes, type 2

Introduction
Diabetes mellitus (DM) is a chronic endocrine-metabolic disease defined by persistent hyperglycaemia. In type 2 diabetes mellitus (T2DM), inadequate insulin secretion together with insulin resistance are central drivers of disease pathophysiology [1]. The International Diabetes Federation estimates that by 2025 more than 589 million adults aged 21-80 years will be living with DM worldwide, with T2DM comprising the majority of cases [2]. Between 1990 and 2017, incident DM rose by about 102.9% across countries from 11, 303, 084 new cases with projections indicating an additional 200 million people affected globally by 2040, underscoring the condition's growing public-health burden [3].

Electrolytes are fundamental to physiological regulation including body-fluid balance, acid-base homeostasis, nerve transmission, haemostasis, and muscle contraction and disturbances arising from kidney disease, dehydration, fever, diarrhoea, or vomiting can contribute to complications observed in DM and related endocrine disorders [4, 5]. Electrolyte abnormalities frequently accompany DM; impaired or diminished insulin action can shift ions between intra- and extracellular compartments, altering their measured concentrations [6, 7]. Population differences in genetics, nutrition, and environment may further shape electrolyte patterns.

Ionic mechanisms also interface with endocrine control. Ionised Na^+ and K^+ influence β-cell function and the modulation of insulin release [8]. Glucose enhances insulin secretion by closing ATP-sensitive K^+ channels, which triggers voltage-gated Ca^{2+} entry and exocytosis of insulin granules; additionally, ionised Na^+ can affect agonist/antagonist binding at the α_2-receptor and suppress insulin release [9]. The role of sodium as a β-cell stimulus remains debated, and there is evidence that the membrane enzyme Na^+/K^+-ATPase undergoes non-enzymatic glycosylation in DM, potentially reducing cellular K^+ uptake that depends on this pump; chronic hyperglycaemia or insulin deficiency may contribute to such enzymatic impairment [11].

Accordingly, the present study measures serum Na^+, K^+, and Mg^{2+} and examines their relationships with glycaemic status in a cohort of Iraqi patients with T2DM.

Materials and Methods

This study was conducted on a group of females with type 2 diabetes, where samples were collected from the Al-Qamat medina specialized laboratory and Al-Sadr teaching hospital in Amara, Maysan governorate confirmed their infection with this disease through the tests conducted on them by professional doctors, the period of this study was from 2 /1/2023 to 10/4/2023, the total number of samples was (116) included (60) women with type 2DM and (56) healthy women.

Venous blood (5) milliliter drowns from each fasting patient and control. The blood sample was divided in two parts; 2.5 mL of blood was transferred into EDTA tube for HbA1c determination, and other part of blood was left for 15 minutes at room temperature to be clotted, and then using the centrifuge, the serum was separated at 3000 rpm for five minutes, to measure glucose and ions.

Measurement of ions

The serum is taken and placed in the cuvette cell of the device where COBAS C111 is used (ROCHE COBAS C111 AUTOMATED CHEMISTRY ANALYZER).

Statistical analysis

The t-test was used to find significant differences between the mean values of the infected and healthy subjects at a significance level of 0.05 [12].

Results

The number of women with diabetes type two conducted in the current research was (30) women, and the number of healthy women was (28) women as presented in Table 1.

Table 1: Number and the percentage of women with diabetes and healthy

Gender	Number	Percentage%
Healthy women	56	48.28
Patient women	60	51.72
Total	58	100

The oldest age of the infected women was 66 age and the youngest age was 30 in age, while for healthy women, The cohort ranged in age from 20 to 55 years, as available in the Table 2.

Table 2: Distribution of the patients and healthy women according to the age

Women	Oldest age	Youngest age
Healthy women	55 years	20 years
Patient women	66 years	30 years

Table 3 indicates significantly higher blood glucose and HbA1c in women with T2DM than in healthy controls ($p< 0.05$).

Table 3: Blood sugar and HbA1c values in both Patients and healthy women

Parameters	Patient women	Healthy women
Blood sugar mg/dl	187.83± 9.43 [a]	97.73± 6.30 [b]
HbA 1c sugar mg/dl	12.83 ± 1.34 [a]	5.31± 0.25 [b]

*Data are presented as mean ± SD.

*Values bearing different superscript letters differ significantly ($p< 0.05$).

Table 4 shows lower sodium and higher potassium and chloride in diabetic women versus healthy controls, none reaching significance; in contrast, calcium was significantly reduced ($p< 0.05$).

Table 4: Values of sodium Na^+, potassium K^+, chloride Cl^-, and calcium Ca^{2+}, To two groups of patients and healthy women

Women	Patients	Healthy
Sodium (mmol/L)	120.02± 3.75 [a]	141.27± 5.60 [a]
Potassium (mmol/L)	4.36±0.61 [a]	4.21±0.31 [a]
Chloride (mmol/L)	108.44±9.25 [a]	92.24±10.77 [a]
Calcium (mmol/L)	8.95±0.93 [a]	10.65±1.14 [b]

*Values are expressed as mean ± SD. Means that do not share a superscript letter differ significantly ($p< 0.05$); matching letters indicate a non-significant difference ($p< 0.05$)

Discussion

Diabetes mellitus is defined by persistent dysregulation of blood glucose, arising from inadequate insulin secretion, resistance to insulin action, or both, and it represents a major worldwide health challenge for healthcare systems and for patients themselves [13]. Numerous studies have linked DM to alterations in key minerals Na^+, K^+, and Cl^- [14, 15, 16]. In our data (Table 3), women with type 2 diabetes showed significantly higher fasting glucose and HbA1c than healthy peers ($p< 0.05$). This aligns with prior work reporting elevated HbA1c in female patients [17], and with additional studies noting significant increases in fasting glucose alongside HbA1c in T2DM versus controls [18, 19].

Serum sodium was lower in the diabetic group (120.02 ± 3.75 mmol/L), a finding consistent with reports of reduced sodium levels among individuals with T2DM [20, 21, 22], although some studies observed no significant sodium difference [23]. Mechanistically, hyperglycaemia promotes osmotic diuresis and fluid shifts; increased extracellular osmolality draws water out of cells, diluting extracellular Na^+ and lowering measured serum sodium. Dysregulation of the renin-angiotensin system in diabetes may further influence sodium balance [24, 25]. Altered vasopressin control has also been proposed as a contributor to hyponatraemia in DM.

Potassium was higher in the diabetic cohort (4.36 ± 0.61) relative to controls, in agreement with studies that reported raised K^+ in diabetic patients [26, 27], though other investigations documented decreases [28, 29] or no material difference [26, 30]. Perturbations in insulin dynamics can modify cellular K^+ handling in liver and muscle via effects on the Na^+/K^+-ATPase, thereby influencing circulating potassium [31]; disruption of this transport machinery has been linked to various diabetic complications [32].

We also observed significantly higher serum chloride in T2DM (108.44 ± 9.25) compared with controls (92.24 ± 10.77), consistent with earlier findings [21, 33]. One plausible explanation is ketoacidosis, the associated acidaemia disturbs acid-base balance and can drive chloride elevations [31, 33].

Prior studies have noted slightly lower calcium in diabetic patients than in healthy groups, differences that may reflect context-dependent variation in mineral handling and metabolism; further work is needed to clarify determinants and clinical implications [34]. Our observations accord with

reports of significantly reduced calcium in T2DM with inverse correlations to disease duration and age [35] and with recent data showing lower calcium in T2DM than controls (8.46 ± 0.63 vs 8.86 ± 0.64 mg/dL) [36]. Beyond serum levels, T2DM has been associated with impaired bone remodelling [37]. Multiple influences may underlie mineral disturbances; for example, shifts in gut microbiota can modify intestinal calcium absorption and have been implicated in both diabetes and skeletal pathology, with potential consequences for bone health in diabetic states [38]. Another study reported hypocalcaemia (below 2.15 mmol/L) in 73.6% of T2DM patients, underscoring the need to investigate causes of electrolyte derangements comprehensively [39].

Finally, an inverse relationship between serum sodium and potassium has been described in diabetic coma, likely reflecting insulin-related shifts of ions between intra- and extracellular compartments [40]. Consistent with this, [41] reported significantly elevated potassium levels. Collectively, these electrolyte perturbations may contribute to biochemical processes that underlie long-term diabetic complications.

Conclusion

In this cohort of 116 women from Maysan, type 2 diabetes mellitus was marked by poor glycaemic control and a characteristic electrolyte profile, sodium tended to be lower and potassium and chloride higher without statistical significance, whereas calcium showed a clear and meaningful reduction. Together, these patterns underscore the tight coupling between disordered glucose regulation, membrane ion transport, and mineral homeostasis, highlighting calcium as a practical signal to monitor in routine care. Clinically, incorporating periodic electrolyte panels especially calcium into diabetes follow-up, alongside assessment of diet, vitamin D status, kidney function, and medications, may help identify patients at risk and guide timely correction. Future work should extend these findings with longitudinal designs, larger multi-site samples, and adjustment for confounders to determine whether correcting these imbalances improves symptoms and long-term outcomes.

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