ORIGINAL ARTICLE

Study of the Trace Elements Profile in Patients with Type 2 Diabetes Mellitus and its Relationship to Glycemic Control and Microvascular Complications

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Abstract

Background: ***Type 2 diabetes mellitus(T2DM) involves metabolic disruptions affecting carbohydrates, fats, and proteins, coupled with impaired insulin secretion and action. Trace elements (TE) play a vital role in insulin synthesis, improving insulin sensitivity, and facilitating glucose metabolism.***

Objective: ***This study evaluated serum levels of magnesium(Mg), manganese(Mn), copper(Cu), chromium(Cr), selenium(Se), cobalt(Co), and zinc(Zn) in T2DM patients and their association with glycemic control and diabetic microvascular complications.***

Methods: ***150 participants were split up into three groups for the study: 50 had good glycemic control, 50 had poor control, and 50 were healthy controls. Clinical, biochemical, and electrophysiological assessments were conducted to evaluate TE levels, glycemic parameters, kidney function, and diabetes complications.***

Results: ***TE levels were significantly lower in diabetics, especially those with poor blood sugar control, than in healthy controls($p<0.001$). Glycemic indicators like HbA1c, fasting glucose, and postprandial glucose were found to be negatively correlated with TE levels, whereas kidney function(eGFR) was positively correlated with TE. Higher albuminuria, more advanced diabetic retinopathy (DR), and worsening neuropathy were all associated with lower TE levels ($p<0.05$). Logistic regression revealed strong associations between Zn, Se, and Cu deficiencies and poor glycemic control. Zn deficiency was linked to severe albuminuria, Mg deficiency to CKD stages≥3b, Cu and Se deficiencies to proliferative diabetic retinopathy(PDR), and Mn deficiency to overt neuropathy.***

Conclusion: ***TE deficiencies were significantly linked to inadequate glycemic control. And microvascular complications. Regular monitoring of TE levels may aid in early intervention and improve diabetic care.***

Keywords: Glycemic control; Type 2 diabetes mellitus; Trace elements

1. Introduction

Diabetes prevalence is rising globally, from 537 million in 2021, the number of people impacted is expected to rise to 783 million by 2045, with Egypt expected to rank 9th in cases.[1] Selenium, an essential TE, protects tissues from oxidative stress (OS) and exhibits insulin-like actions.[2] Zinc is crucial for insulin crystallization, signaling, carbohydrate metabolism, and GLUT4 translocation, supporting antioxidative functions.[3,4] Chromium enhances insulin sensitivity via cell receptors, while deficiency may promote diabetes.[5,6] Magnesium regulates insulin production and receptor phosphorylation, with deficiency linked to insulin resistance.[7] Cobalt, part of vitamin B12, acts as a coenzyme in glucose metabolism.[8] Copper levels in T2DM vary with glycemic control.[9]

Accepted 10 February 2025.
Available online 30 April 2025

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https://doi.org/10.21608/aimj.2025.446530

Excessive manganese disrupts antioxidative activity and induces OS.[10,11] Diabetic retinopathy, a complex disorder, remains poorly understood. Hyperglycemia-induced OS accelerates pathogenic lesions despite optimal glycemic control.[12,13] Diabetic kidney disease (DKD) affects 20-30% of T2DM cases, with OS implicated in metabolic disease progression.[14,15] Diabetic neuropathy causes nerve damage, manifesting as pain, numbness, and weakness.[16]

The link between T2DM, microvascular complications, and serum TE levels remains unclear, warranting further investigation.

This study aimed to assess the status of TE among patients with T2DM and their relationship to glycemic control and diabetic microvascular complications.

2. Patients and methods

Based on their HbA1c levels, 100 diabetic patients were split into two groups for the study. There were 50 patients in Group I who had poor glycemic control (HbA1c of 7 or higher), and 50 patients in Group II who had good glycemic control (HbA1c ≤ 7). A control group of fifty healthy people was also included.

Exclusion criteria:

These include Type 1 diabetes, gestational diabetes, recent TE supplementation, malabsorption disorders, and active inflammatory or malignant conditions. Clinical assessment included a detailed physical examination, emphasizing anthropometric measurements (height, weight, and BMI). Diabetic retinopathy (DR) was evaluated via optic fundus examination using direct ophthalmoscopy and graded as mild, moderate, severe non-proliferative DR (NPDR), or PDR based on retinal findings.[17]

Table 1. Grading of DR

GRADING	RETINAL FINDING
MILD NPDR	Microaneurysms only
MODERATE NPDR	A microaneurysm or at least one hemorrhage, as well as any of the following: Hemorrhage in the retina. Tough exudate. Spots on cotton wool. Venous beading.
SEVER NPDR	Any of these, but with no indication of PDR: Each of the four quadrants contains more than 20 intraretinal hemorrhages. Two or more quadrants show clear venous beading. Significant microvascular anomalies within the retina in one or more quadrants.
PDR	Any of: Growth of new blood vessels (neovascularization). Bleeding in the vitreous or beneath the retina (preretinal hemorrhage).

Neuropathy assessment utilized the modified Michigan Neuropathy Screening Instrument (MNSI),[18] confirmed by nerve conduction studies (NCS).

UACR from a urine sample and eGFR using the CKD-EPI equation were used to evaluate DKD.[19] After three months, persistent abnormalities were verified, and CKD stages were categorized in accordance with KDIGO guidelines [20]

Table 2. The KDIGO classification of CKD.

GFR STAGES	GFR (ML/MIN/1.73 M2)	TERMS
G1	≥90	Normal or high.
G2	69- 89	Mildly decreased.
G3A	45-59	Mildly to moderately decreased.
G3B	30-44	Moderately to severely decreased.
G4	15-29	Severely decreased.
G5	<15	Kidney failure.
ALBUMINURIA STAGES	UACR (mg/g)	Terms
A1	<30	Normal to mildly increased.
A2	30-300	Moderately increased
A3	>300	Severely increased.

Laboratory investigations included glycemic parameters (FPG, 2HPPG, HbA1c). For TE analysis, 5 mL fasting blood samples were digested with nitric acid at 120°C, diluted to 10 milliliters, and then subjected to inductively coupled plasma mass spectrometry analysis.

The software program STATA (version 12; STATA Inc., College Station, TX, USA) was used for the statistical analysis; a significance level of $p < 0.05$ and a 95% confidence interval were established.

3. Results

TE concentrations varied significantly, according to the study. TE levels were lower in Group I than in Group II, and lower in both diabetic groups than in Group III. Group II had higher manganese (Mn) levels than Group I, and both diabetic groups had higher Mn levels than the healthy controls. Group I exhibited significantly lower Mn levels, while Group II had higher levels (Table 3).

Table 3. The baseline serum levels of TE of the studied group.

		GROUP I (N=50)	GROUP II (N=50)	GROUP III (N=50)
SELENIUM (µG/DL)	Mean ± SD	6.35 ± 1.21	8.79 ± 1.72	16.3 ± 2.02
PAIRWISE: P1<0.001*, P2<0.001*, AND P3<0.001*				
SUFFICIENT	N. (%)	32 (64%)	48 (96%)	35 (70%)
DEFICIENT	N. (%)	18 (36%)	2 (4%)	0 (0%)
COPPER (µG/DL)	Mean ± SD	72.54 ± 8.30	95.45 ± 10.79	129.44 ± 6.08
PAIRWISE: P1<0.001*, P2<0.001*, AND P3<0.001*				
SUFFICIENT	N. (%)	23(46%)	47(94%)	48(96%)
DEFICIENT	N. (%)	27(54%)	3(6%)	2(4%)
ZINC (µG/DL)	Mean ± SD	61.2 ± 2.43	70.54 ± 2.65	75.12 ± 2.92
PAIRWISE: P1<0.001*, P2<0.001*, AND P3<0.001*				
SUFFICIENT	N. (%)	0 (0%)	0 (0%)	27 (54%)
DEFICIENT	N. (%)	50 (100%)	50 (100%)	23 (46%)
MAGNESIUM(MG/DL)	Mean ± SD	1.85 ± 0.09	2.01 ± 0.16	2.87 ± 0.46
PAIRWISE: P1=0.019*, P2<0.001*, AND P3<0.001*				
SUFFICIENT	N. (%)	35 (70%)	48 (96%)	48 (96%)
DEFICIENT	N. (%)	15 (30%)	2 (4%)	0 (0%)
CHROMIUM(µG/DL)	Mean ± SD	4.74 ± 0.9	8.73 ± 1.08	10.86 ± 2.79
PAIRWISE: P1<0.001*, P2<0.001*, AND P3<0.001**				
SUFFICIENT	N. (%)	0 (0%)	0 (0%)	14 (28%)
DEFICIENT	N. (%)	50 (100%)	50 (100%)	36 (72%)
MN (MG/L)	Mean ± SD	0.48 ± 0.17	1.11 ± 0.27	0.71 ± 0.31
PAIRWISE: P1<0.001*, P2<0.001*, AND P3<0.001*				
SUFFICIENT	N. (%)	35 (70%)	27 (54%)	34 (68%)
DEFICIENT	N. (%)	15 (30%)	0 (0%)	9 (18%)
COBALT(MG/L)	Mean ± SD	0.03 ± 0.02	0.13 ± 0.08	0.52 ± 0.2
PAIRWISE: P1<0.001*, P2<0.001*, P3<0.001*				
SUFFICIENT	N. (%)	0 (0%)	23 (46%)	16 (32%)

DEFICIENT	N. (%)	50 (100%)	27 (54%)	0 (0%)

*: SIGNIFICANT AS P VALUE<0.05 P1: COMPARISON BETWEEN GROUPS I AND II P2: COMPARISON BETWEEN GROUPS I AND III P3: COMPARISON BETWEEN GROUPS II AND III.

Our study found significant negative correlations between serum levels of all studied TE and glycemic control parameters (P<0.001). Additionally, eGFR showed a positive correlation with TE levels. Negative correlations also were observed between TE and both UACR and MNS. A significant association was noted between TE levels and DR severity (Table 4).

Table 4. Correlations between serum levels of the studied TE with various clinical and laboratory variables among the studied diabetic patients

	SELENIUM (µG/DL)		COPPER (µG/DL)		ZINC (µG/DL)		MAGNESIUM (MG/DL)		CHROMIUM (µG/DL)		MANGANESE (MG/L)		COBALT (µG/L)	
	r/*rho*	P	r/*rho*	p	r/*rho*	P	r/*rho*	P	r/*rho*	p	r/*rho*	p	r/*rho*	p
FPG (MG/DL)	-0.614	0.000	-0.744	0.000	-0.839	0.000	-0.472	0.000	-0.807	0.000	-0.854	0.000	-0.827	0.000
2HPPPG (MG/DL)	-0.632	0.000	-0.726	0.000	-0.815	0.000	-0.426	0.000	-0.784	0.000	-0.815	0.000	-0.841	0.000
HBA1C%	-0.741	0.000	-0.859	0.000	-0.932	0.000	-0.573	0.000	-0.850	0.000	-0.917	0.000	-0.886	0.000
EGFR (ML/MIN 1.73/ M^2)	0.385	0.000	0.452	0.000	0.464	0.000	0.220	0.028	0.434	0.000	0.472	0.000	0.487	0.002
UACR (MG/G)	-0.460	0.000	-0.529	0.000	-0.612	0.000	-0.399	0.000	-0.588	0.000	-0.568	0.000	-0.613	0.003
ALBUMINURIA SEVERITY	-0.224	0.02	-0.458	0.000	-0.422	0.000	-0.331	0.008	-0.415	0.000	-0.421	0.000	-0.477	0.000
CKD STAGE DETERIORATION	-0.392	0.000	-0.432	0.000	-0.456	0.000	-0.229	0.02	-0.414	0.000	-0.447	0.000	-0.428	0.000
MNS SCORE	-0.308	0.002	-0.490	0.000	-0.432	0.000	-0.346	0.005	-0.298	0.003	-0.417	0.000	-0.358	0.001
DSPN SEVERITY	-0.428	0.000	-0.523	0.000	-0.506	0.000	-0.342	0.000	-0.446	0.000	-0.522	0.000	-0.421	0.000
DR SEVERITY	-0.578	0.000	-0.686	0.000	-0.660	0.000	-0.436	0.000	-0.594	0.000	-0.648	0.000	-0.609	0.000

FASTING PLASMA GLUCOSE IS ABBREVIATED AS FPG, TWO HOURS POSTPRANDIAL PLASMA GLUCOSE IS ABBREVIATED AS 2HPPPG, GLYCATED HEMOGLOBIN IS REPRESENTED BY HBA1C%, ESTIMATED GLOMERULAR FILTRATION RATE BY EGFR, URINE ALBUMIN/CREATININE RATIO BY UACR, CHRONIC KIDNEY DISEASE BY CKD, MICHIGAN NEUROPATHY, AND DIABETIC SENSORY PERIPHERAL NEUROPATHY BY DSPN. PEARSON'S CORRELATION COEFFICIENT IS DENOTED BY RHO, AND SPEARMAN'S CORRELATION COEFFICIENT BY R

Multiple logistic regression analyses assessed the relationship between TE deficiencies and diabetes-related parameters, adjusting for age, sex, BMI, and disease duration. Suboptimal glycemic control was linked to Zn, Se, and Cu deficiencies (in descending significance). Zn deficiency was associated with severe urinary albumin excretion (RR 8.9, 95% C.I. 6.9–10.5). Mg deficiency was linked to CKD stages ≥3b (RR 5.2, 95% C.I. 3.9–8.6). Cu and Se deficiencies were associated with proliferative diabetic retinopathy. Mn deficiency correlated with clinically overt diabetic neuropathy (Tables 5, 6).

Table 5. Regression analyses of the association of various TE deficiency with various parameters

	HbA1c%		UACR		eGFR		DR		DSPN	
	β	Sig.	β	Sig.	β	Sig.	β	Sig.	β	Sig.
Selenium(µg/dL)	-0.16	0.03	-4.43	0.92	0.33	0.74	-0.46	0.05	-0.20	0.28
Copper(µg/dL)	-0.21	0.04	-1.93	0.79	0.14	0.40	-0.09	0.02	-0.01	0.66
Zinc(µg/dL)	-0.49	0.001	-91.65	0.002	0.37	0.58	0.07	0.61	-0.07	0.58
Magnesium (mg/dL)	-0.08	0.24	125.5	0.79	-22.5	0.04	-1.29	0.59	-0.51	0. 8
Chromium (µg/dL)	-0.13	0.26	21.11	0.71	-0.79	0.54	0.09	0.73	0.32	0.2
Manganese (µg/L)	-.05	0.72	148.6	0.7	13.01	0.15	1.16	0.54	-4.21	0.02
Cobalt (µg/L)	0.16	0.07	30.29	0.98	30.72	0.31	-14.7	0.22	7.03	0.3

Table 6. The relative risk of various TE deficiency with various diabetic complications

	Uncontrolled DM		A3 proteinuria		CKD stage ≥ 3b		PDR		Clinically overt DSPN	
	RR	95% C.I.	RR	95% C.I.	RR	95% C.I.	RR	95% C.I.	RR	95% C.I.
Se deficiency	2.5	(1.7-3.1)	1.1	(0.95-1.4)	1.4	(1.2-1.8)	2.24	(1.5-3.5)	1.6	(1.15-2.3)
Cu deficiency	2.3	(1.8-3.3)	1.3	(1.2-1.5)	1.55	(1.3-1.9)	3.41	(2.29-5.1)	2.23	(1.7-3)
Zn deficiency	9.7	(8.7–12.5)	8.9	(6.9-10.5)	3.2	(2.7-5.5)	2.1	(1.7–2.6)	1.3	(1.1-1.5)
Mg deficiency	2.1	(1.5-2.8)	1.2	(1.02-1.4)	5.2	(3.9-8.6)	1.92	(1.2-3)	1.63	(1.15-2.3)
Cr deficiency	1.3	(0.7-2.1)	1.1	(0.9-1.3)	2.1	(1.8 -2.5)	1.2	(1-1.5)	1.03	(0.95-1.3)
Mn deficiency	0.96	(0.8-1.03)	1.04	(0.92-1.2)	1.2	(1.01-1.4)	1.1	(0.92-1.3)	5. 3	(4.7-6.3)
Co deficiency	0.4	(0.3-0.48)	1.72	(1.2-2.56)	1.96	(1.2-3.3)	5.5	(1.4-21.2)	3.6	(1.4-8.9)

4. Discussion

TE concentrations were found to differ significantly between Group I and Group II, with Group I having lower levels of Se, Cu, Zn, Mg, Mn, Cr, and Co. Additionally, TE levels were lower in both diabetic groups than in the healthy control group (Group III).

Additionally, Se levels positively correlated with eGFR and negatively with UACR. Lower Se levels (less than 6 µg/dL) were linked to a 1.6-fold higher risk of DSPN and a 1.4-fold higher risk of CKD progression to stage ≥ 3b, although logistic regression analysis did not show any significant associations. These results demonstrate Se's possible involvement in microvascular problems and glycemic control. The association between lower Se levels and the severity of DR has also been confirmed by additional research. A more complicated relationship may exist, though, as one study found that patients with proliferative diabetic retinopathy (PDR) had higher Se levels.[21,22,23]

The study also evaluated the role of Cr, Zn, Cu, and Mn in T2DM and its complications. Serum Cr levels exhibited a negative correlation with glycemic control parameters. Although not statistically significant in logistic regression, each reduction in Cr levels below 13 µg/dL raised the likelihood of inadequate glycemic control, DR, CKD, and DSPN. These findings align with studies that highlighted the inverse relationship between Cr levels and glycemic regulation.[24,25]

Zinc deficiency showed a pronounced association with poor glycemic control and microvascular complications. Logistic regression revealed a 9.7-fold increased risk of poor glycemic control for each reduction in Zn levels below 75 µg/dL (RR = 9.7, 95% CI = 8.7–12.5). Reduced Zn levels were also linked to DR, CKD, and DSPN severity. These findings are consistent with studies that reported decreased serum Zn levels in patients with diabetes and their relationship with complications such as nephropathy.[26,27]

For every drop below 70 µg/dL, the probability of poor glycemic control increases by 2.3 times, suggesting a strong negative relationship between copper levels and glycemic parameters (95% CI = 1.8-3.3). Furthermore, there was a negative correlation between Cu levels and both DR severity and DSPN, consistent with findings by Zargar et al. and Al-Timimi et al.[28, 29] Conversely, some studies reported elevated Cu levels in T2DM patients, highlighting conflicting evidence regarding Cu's role in diabetes.[30]

Lastly, serum Mn levels showed variable correlations with glycemic control and microvascular complications. Although Group I's Mn levels were lower than Group III's, logistic regression analysis revealed no link between Mn deficiency and inadequate glycemic control. Walter et al. found lower Mn levels in diabetes individuals with diabetes and also emphasized its complex role as an antioxidant cofactor; thus, our results are in line with theirs.[31]

4. Conclusion

This study highlights the importance of TE (Se, Cr, Zn, Co, Mg, Cu, Mn) in T2DM with respect to glycemic control and microvascular complications, suggesting potential therapeutic insights for further research.

Disclosure

The authors have no financial interest to declare in relation to the content of this article.

Authorship

All authors have a substantial contribution to the article

Funding

No Funds : Yes

Conflicts of interest

There are no conflicts of interest.

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