

Serum VCAM-1, zinc status, and cardiorenal risk markers in Iraqi adults with type 2 diabetes: A case-control study

Tami HA¹[ID](#), Behayaa HR¹[ID](#)

¹Department of Chemistry and Biochemistry, College of Medicine, University of Babylon, Iraq

Submitted: 13th January 2026

Accepted: 18th April 2026

Published: 30th September 2026

[ID](#): Orcid ID

Abstract

Objective: To evaluate serum vascular cell adhesion molecule-1 (VCAM-1), zinc, renal indices, and lipid profile in adults with type 2 diabetes mellitus (T2DM) compared with healthy controls, and to explore their associations with glycaemic variables.

Methods: This hospital-based case-control study included 120 participants (60 with T2DM, 60 controls) from Karbala, Iraq. Serum VCAM-1, zinc, iron, fasting glucose, HbA1c, creatinine, lipid profile, urinary microalbumin, and urinary albumin-to-creatinine ratio (uACR) were measured using standard chemistry, HPLC and ELISA methods. Comparisons, correlations, ROC analysis and binary logistic regression were performed.

Results: Compared with controls, the T2DM group had poorer glycaemic status, higher uACR and serum creatinine, significantly higher VCAM-1 (29.6 ± 12.36 vs 25.6 ± 4.45 ng/mL, $P = 0.029$), lower zinc (99.4 ± 18.01 vs 107.9 ± 14.02 µg/dL, $P = 0.028$), and a more atherogenic lipid profile (higher triglycerides, LDL-C, VLDL-C; lower HDL-C). Triglycerides and VLDL-C correlated positively with fasting glucose and HbA1c, and LDL-C with HbA1c. In univariable binary logistic regression, VCAM-1 was not a statistically significant independent predictor of T2DM (OR = 1.070, 95% CI: 0.989–1.157, $P = 0.091$).

Conclusion: T2DM is associated with concurrent metabolic, renal, endothelial and trace-element disturbances. However, VCAM-1 showed limited discriminative ability and was not an independent predictor in this dataset, arguing against its use as a standalone clinical marker.

Keywords: Type 2 diabetes mellitus, VCAM-1, Zinc, Albumin-to-creatinine ratio, Diabetic kidney disease, Dyslipidaemia

Plain English Summary

Type 2 diabetes is a common condition that can affect the kidneys and blood vessels. In this study, we compared 60 Iraqi adults with diabetes to 60 healthy people. We measured a blood marker of vascular inflammation (VCAM-1), the mineral zinc, kidney function tests, and cholesterol levels. People with diabetes had higher VCAM-1, lower zinc, worse kidney tests, and an unhealthy cholesterol pattern. However, VCAM-1 alone was not strong enough to reliably identify diabetes or kidney damage. The results suggest that diabetes affects multiple body systems simultaneously, but simple single markers may not be sufficient for making clinical decisions.

Introduction

Type 2 diabetes mellitus remains one of the most important chronic non-communicable diseases worldwide and is a major driver of long-term vascular and renal morbidity (1, 2, 3). Up to 40% of people with diabetes develop chronic kidney

disease, and contemporary guidelines therefore treat kidney involvement as a core component of risk stratification rather than a late complication (2). Early identification of renal and vascular injury is clinically important because even modest urinary albumin excretion abnormalities may

Correspondence:

Hussein Allawi Tami

Department of Chemistry and Biochemistry, College of Medicine
University of Babylon

Iraq

+9647725632430; Husseinallawitami99@gmail.com

reflect broader microvascular dysfunction and future cardiorenal risk (4).

Diabetic kidney disease is increasingly understood as a complex microvascular complication shaped by metabolic, inflammatory, haemodynamic, and endothelial disturbances (5, 6). Endothelial dysfunction contributes to this process through oxidative stress, inflammation, reduced nitric oxide bioavailability, and maladaptive intercellular signalling (5). In the kidney, glomerular endothelial injury can disturb the filtration barrier and interact with podocyte and tubular injury pathways (6). Against this background, circulating markers linked to endothelial activation, such as vascular cell adhesion molecule-1, have attracted interest. VCAM-1 is involved in leukocyte adhesion and endothelial inflammatory activation; its elevation has been associated with diabetic microvascular complications, including kidney involvement (5, 6).

At the same time, trace-element disturbances in T2DM – particularly zinc homeostasis – have received increasing attention. Zinc participates in insulin synthesis, storage, and secretion, as well as antioxidant defence (7). Zinc deficiency may be linked to insulin resistance, impaired glycaemic control, and enhanced oxidative stress, while diabetes itself may lower zinc status through altered absorption and increased urinary loss (7, 8). In diabetic kidney disease, zinc may be biologically connected to renal outcomes through antioxidant and anti-inflammatory mechanisms, although direct clinical evidence remains limited (8).

Urinary albumin excretion remains the most established renal marker. Current diabetes standards recommend systematic assessment of albuminuria because it is central to chronic kidney disease detection and staging (2). Nevertheless, albuminuria alone does not fully explain the heterogeneity of diabetic renal injury, nor does it directly characterise endothelial or micronutrient dimensions. Combining urinary albumin indices with endothelial and trace-element markers may provide a more nuanced picture (9, 10).

Despite growing literature, several gaps remain. First, most studies examine endothelial dysfunction, albuminuria, and zinc status separately. Second, the extent to which VCAM-1, serum zinc, and urinary albumin measures co-vary in T2DM is not fully defined. Third, region-specific data from Middle Eastern and Iraqi populations remains limited (11, 12). The aim of this study was therefore to assess serum VCAM-1, serum zinc, and urinary albumin-related renal indices in adults with T2DM compared with healthy controls, and to explore how these markers relate to glycaemic and

biochemical characteristics. We hypothesized that T2DM would be associated with higher VCAM-1, lower zinc, and an adverse renal and lipid profile, but that VCAM-1 would not outperform established markers.

Materials and Methods

Study design and setting

This hospital-based case-control study was conducted in Karbala, Iraq, between January and June 2024. It included 120 participants: 60 patients with T2DM and 60 apparently healthy controls. Recruitment and specimen collection were carried out at the Imam Al-Hasan Center for Diabetes and Endocrinology and Imam Al-Hasan Teaching Hospital, Karbala Health Directorate (13).

Participant recruitment and selection

Eligible participants were recruited consecutively. The T2DM group comprised adults with previously diagnosed type 2 diabetes under follow-up. The control group comprised adults without diabetes and without major systemic disease. A structured case-record form collected demographic and clinical information (9, 10).

Inclusion criteria

T2DM group: adults of either sex with established T2DM attending the recruitment centres, willing to provide blood and urine.

Control group: adults of either sex without known diabetes, not receiving antidiabetic treatment.

Exclusion criteria

Type 1 diabetes, current acute infection or inflammatory disease, chronic liver disease, non-diabetic renal disease, malignancy, pregnancy or lactation, recent vitamin/mineral supplements, recent glucocorticoid therapy, or any severe systemic disorder likely to influence endothelial, renal, inflammatory, or trace-element measurements (9, 10).

Clinical and anthropometric assessment

Body weight and height were recorded, and body mass index was calculated as weight (kg) / height (m²). Diabetes duration and treatment history were documented from clinical records (13).

Sample collection and processing

After an overnight fast of at least 8 hours, 5 mL of venous blood was collected. A 3 mL portion was placed in a plain gel tube for serum separation (glucose, creatinine, lipid profile, zinc, iron, VCAM-1). A 2 mL portion was placed in an EDTA-K₂ tube for HbA1c. A spot urine sample was collected for microalbumin and urinary creatinine. Serum was separated by

centrifugation at 3000 rpm for 10 minutes. All samples were processed on the same day (13).

Laboratory measurements

Fasting blood glucose: glucose oxidase method (Dirui CS-T180, China (14).

HbA1c: HPLC (GH-900 Plus Lifotronic, China) (14).

Serum and urinary creatinine: enzyme method (Dirui CS-T180).

Urinary microalbumin: immunoturbidimetric assay. Urinary albumin-to-creatinine ratio (uACR) was calculated as mg albumin / g creatinine. Normoalbuminuria: <30 mg/g; microalbuminuria: 30–300 mg/g; macroalbuminuria: >300 mg/g (2).

Serum VCAM-1: ELISA (Biotech, USA; Cat. No. E0203Hu). Intra-assay CV <6%, inter-assay CV <9% (15).

Serum zinc: colourimetric method (Dirui kit) (7).

Serum iron: ferene method (Dirui kit).

Lipid profile: total Cholesterol, triglycerides, HDL-C (direct), LDL-C (direct), and VLDL-C (calculated as triglycerides/5) (1).

Quality control

All assays included routine internal quality controls. Calibration and reagent handling followed the manufacturer's instructions.

Statistical analysis

Sample size was not based on a formal power calculation; this was an exploratory study. Normality was assessed using the Shapiro–Wilk

test. Normally distributed data are presented as mean $\pm$ SD; non-normally distributed data as median (IQR). Group comparisons used an independent t-test, Mann–Whitney U, or chi-square as appropriate. One-way ANOVA was used for albuminuria subgroups. Pearson or Spearman correlation was used. Receiver operating characteristic (ROC) analysis was performed for VCAM-1. Univariable binary logistic regression examined the association of VCAM-1 with diabetes status (no multivariable adjustment was possible because data on key confounders such as medication, blood pressure, and smoking were not collected). A two-sided $P < 0.05$ was considered statistically significant. No adjustment for multiple comparisons was applied, given the exploratory nature.

Results

Demographic and clinical characteristics

Age, sex, and BMI were similar between groups (all $P > 0.05$). The T2DM group had significantly higher fasting glucose, HbA1c, serum creatinine, and uACR (all $P \leq 0.001$). The median uACR in the T2DM group was 86.5 mg/g (IQR 29.6–771.0), reflecting a wide range of albuminuria. Among T2DM patients, 20 (33.3%) had normoalbuminuria, 20 (33.3%) microalbuminuria, and 20 (33.3%) macroalbuminuria (this equal distribution arose from convenience sampling, not deliberate stratification).

Table 1 summarises the demographic, clinical, and biochemical characteristics of both groups.

Table 1: Demographic, clinical and biochemical characteristics of patients with type 2 diabetes mellitus and healthy controls

Variable	T2DM patients (n = 60)	Controls (n = 60)	P value
Demographic characteristics			
Age, years	62.5 $\pm$ 8.23	61.5 $\pm$ 6.57	0.72
Sex, male, n (%)	37 (61.8)	32 (53.4)	0.449
Sex, female, n (%)	23 (38.5)	28 (46.6)	
Body mass index, kg/m ²	29.1 $\pm$ 5.8	26.8 $\pm$ 1.37	0.13
Albuminuria status†			
Normoalbuminuria, n (%)	20 (33.3)	—	
Microalbuminuria, n (%)	20 (33.3)	—	
Macroalbuminuria, n (%)	20 (33.3)	—	
Metabolic and biochemical findings			
Fasting blood sugar, mg/dL	205.0 $\pm$ 52.0	102.6 $\pm$ 10.7	<0.001
HbA1c, %	8.70 $\pm$ 1.60	5.64 $\pm$ 0.69	<0.001
Creatinine, mg/dL	1.6 $\pm$ 1.0	0.75 $\pm$ 0.23	0.001
Albumin/creatinine ratio, median (IQR)	86.5 (29.6–771.0)	22.4 (10.6–26.8)	<0.001‡
Biomarker			
VCAM-1, ng/mL	29.6 $\pm$ 12.36	25.6 $\pm$ 4.45	0.029*

Data are presented as mean $\pm$ SD, median (IQR), or n (%). P values were calculated using an independent-samples t-test unless otherwise indicated. Sex distribution and albuminuria status were compared using the chi-square test. † Albuminuria status assessed only in the T2DM group. ‡ Mann–Whitney U test. Statistically significant at $P < 0.05$.

Figure 1 shows the comparison of mean VCAM-1 levels between groups.

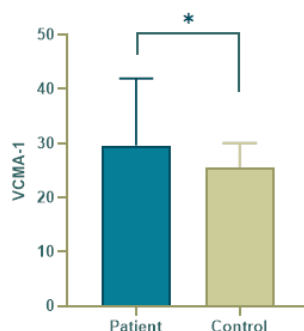


Figure 1. Comparison of mean VCAM-1 among study groups.

VCAM-1 and trace elements

Serum VCAM-1 was significantly higher in T2DM than in controls (29.6 ± 12.36 vs 25.6 ± 4.45 ng/mL, $P = 0.029$). Serum zinc was significantly

lower in T2DM (99.4 ± 18.01 vs 107.9 ± 14.02 µg/dL, $P = 0.028$). Serum iron did not differ (92.9 ± 22.6 vs 88.7 ± 16.5 ng/dL, $P = 0.444$). Table 2 presents the trace element comparisons.

Table 2: Comparison of trace element concentrations between the study groups

Variable	Type 2 diabetes (n = 60)	Control (n = 60)	P value
Iron, ng/dL	92.9 ± 22.6	88.7 ± 16.5	0.444
Zinc, µg/dL	99.4 ± 18.01	107.9 ± 14.02	0.028*

Data are presented as mean $\pm$ SD. Independent-samples t test. Statistically significant at $P < 0.05$

VCAM-1 across albuminuria subgroups

VCAM-1 did not differ significantly across normoalbuminuria (31.77 ± 7.2 ng/mL), microalbuminuria (27.6 ± 12.6), and

macroalbuminuria (29.3 ± 15.8) ($P = 0.57$). The numerically highest level was in normoalbuminuric patients. Table 3 shows the distribution.

Table 3: Comparison of VCAM-1 based on albuminuria status in T2DM patients

Variable	Normoalbuminuria (n = 20)	Microalbuminuria (n = 20)	Macroalbuminuria (n = 20)	P value
VCAM-1 (ng/mL), mean $\pm$ SD	31.77 ± 7.2	27.6 ± 12.6	29.3 ± 15.8	0.57

One-way ANOVA

Lipid profile

Total cholesterol was similar between groups (148.6 ± 43.4 vs 147.5 ± 30.5 mg/dL, $P = 0.914$). Triglycerides (133.8 ± 66.8 vs 86.2 ± 41.4 , $P = 0.026$), LDL-C (83.7 ± 30.6 vs 67.5 ± 29.9 , $P = 0.031$), and VLDL-C (26.7 ± 15.9 vs 17.7 ± 8.8 , P

$= 0.026$) were significantly higher in T2DM, while HDL-C was significantly lower (29.6 ± 9.1 vs 40.7 ± 11.7 , $P < 0.001$).¹⁶ Table 4 summarises the lipid profile.

Table 4: Comparison of lipid profile between the study groups

Variable (mg/dL), mean $\pm$ SD	Type 2 diabetes (n = 60)	Control (n = 60)	P value
Total cholesterol	148.6 ± 43.4	147.5 ± 30.5	0.914
Triglycerides	133.8 ± 66.8	86.2 ± 41.4	0.026*
LDL-C	83.7 ± 30.6	67.5 ± 29.9	0.031*
HDL-C	29.6 ± 9.1	40.7 ± 11.7	<0.001*
VLDL-C	26.7 ± 15.9	17.7 ± 8.8	0.026*

Independent-samples t test. Statistically significant at $P < 0.05$

Correlations with glycaemic markers

Triglycerides and VLDL-C correlated positively with both fasting glucose ($r = 0.317$, $P = 0.013$) and HbA1c ($r = 0.294$, $P = 0.028$). LDL-C correlated positively with HbA1c ($r = 0.290$, $P =$

0.034) but not with fasting glucose ($r = 0.203$, $P = 0.141$). Total cholesterol and HDL-C showed no significant correlations (16). Figure 2 presents these correlations as a heat map.

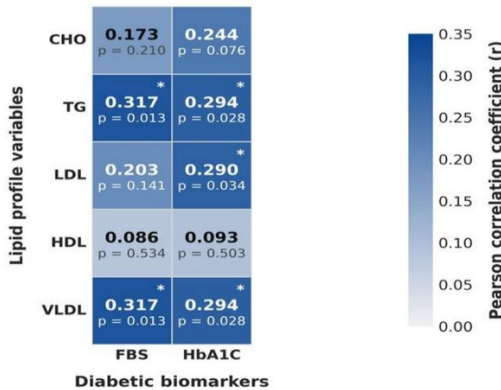


Figure 2: Heat map showing Pearson correlation coefficients between lipid profile variables and diabetic biomarkers

Sex differences in VCAM-1

Within the T2DM group, males had significantly higher VCAM-1 than females (32.4 ± 14.5 vs 25.4

± 6.3 ng/mL, $P = 0.033$). Table 5 shows the comparison.

Table 5: Comparison of VCAM-1 by sex within the type 2 diabetes mellitus group

Variable	Male (n = 37)	Female (n = 23)	P value
VCAM-1 (ng/mL), mean $\pm$ SD	32.4 $\pm$ 14.5	25.4 $\pm$ 6.3	0.033*

Independent-samples t test. Statistically significant at $P < 0.05$

ROC analysis for VCAM-1

The area under the curve was 0.607 ($P = 0.05$). At a cut-off of 27.1 ng/mL, sensitivity was 55.0%, specificity 66.6%, positive predictive value 76.7%

and negative predictive value 51.0%. This indicates poor-to-moderate discriminative ability.¹⁵ Figure 3 shows the ROC curve.

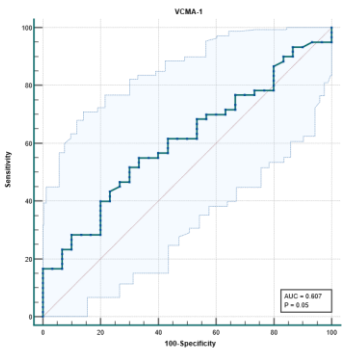


Figure 3: Receiver operating characteristic curve of VCAM-1 for discriminating patients with type 2 diabetes mellitus from healthy controls
(AUC = 0.607, diagonal reference line included.)

Logistic regression

In univariable binary logistic regression, VCAM-1 was not a statistically significant predictor of diabetes (OR = 1.070, 95% CI 0.989–1.157, $P = 0.091$). Multivariable adjustment was not performed because data on medication use,

blood pressure, smoking, and other confounders were not collected.

Discussion

This study compared 60 Iraqi adults with T2DM to 60 healthy controls and found a consistent

pattern of metabolic, renal, endothelial, and trace-element disturbances. Patients with T2DM had poorer glycaemic control, higher uACR and creatinine, higher VCAM-1, lower zinc, and an atherogenic lipid profile. However, VCAM-1 did not differ across albuminuria categories, showed only modest ROC characteristics, and was not a significant predictor in univariable logistic regression (11, 12, 15).

Interpretation of renal and glycaemic findings

The elevated HbA1c and uACR in the diabetes group are consistent with the known pathophysiology of diabetic kidney disease. Albuminuria in T2DM is a clinically meaningful marker of cardiorenal risk (4). Our finding of a wide uACR range (IQR 29.6–771.0) reflects real-world heterogeneity. The results align with previous Iraqi studies showing renal impairment clustering with metabolic stress (11).

VCAM-1 and endothelial activation

The significantly higher VCAM-1 in T2DM is biologically plausible and consistent with diabetes-associated endothelial activation (5, 6). Hyperglycaemia promotes endothelial dysfunction via inflammatory mediators. Our findings are supported by earlier Iraqi and international studies (12, 15). Nevertheless, the absence of a significant gradient across albuminuria categories, the modest AUC of 0.607, and the non-significant logistic regression ($P = 0.091$) indicate that VCAM-1 should not be overinterpreted as a robust independent diagnostic marker. Mechanistic plausibility does not automatically translate into strong clinical classification performance (15). Possible explanations include the multifactorial nature of endothelial activation, the heterogeneity of albuminuria, and reduced statistical power after subgroup stratification.

Zinc findings

The significantly lower serum zinc in T2DM is compatible with both Iraqi and international evidence (7, 8, 11). Zinc participates in antioxidant defence and insulin signalling. Lower zinc may accompany a higher-risk metabolic and renal phenotype. However, serum zinc is an acute-phase reactant that falls during inflammation; without measurement of inflammatory markers such as C-reactive protein, we cannot determine whether lower zinc reflects true deficiency versus inflammatory redistribution. This is a major limitation (7). By contrast, iron did not differ between groups, suggesting that trace-element disturbances are not uniform.

Dyslipidaemia and glycaemic control

The lipid profile – higher triglycerides, LDL-C, VLDL-C, and lower HDL-C with similar total cholesterol – is the classic phenotype of diabetic dyslipidaemia (16). The positive correlations of triglycerides and VLDL-C with both fasting glucose and HbA1c, and of LDL-C with HbA1c, suggest that worse glycaemic control tracks with a more atherogenic lipid pattern (16). The correlation coefficients were modest, reflecting the multifactorial nature of lipid abnormalities.

Sex difference in VCAM-1

The higher VCAM-1 in male patients is an exploratory finding that may reflect sex differences in endothelial function or residual confounding. It should be interpreted cautiously.

Comparison with Iraqi studies

Our findings converge with three local studies: higher VCAM-1 in T2DM (Alsaedy & Al-Rubaei, Baghdad) (12), lower zinc in diabetic nephropathy (Al-Fartosy et al, Basrah) (11), and the association of poor glycaemic control with atherogenic lipids (Al-Asadi & Al-Naama, Basrah) (16). This consistency strengthens the local plausibility of our results (13).

Strengths

The simultaneous examination of glycaemia, renal function, dyslipidaemia, endothelial activation, and trace elements within one case-control framework is a key strength. The inclusion of ROC and regression analyses prevents overstatement of VCAM-1's clinical utility (15).

Study Limitations

Several limitations should be considered when interpreting these findings. First, the cross-sectional design precludes any inference about causality or temporal ordering. Second, no formal sample size calculation was performed, and the sample size, while adequate for detecting several between-group differences, becomes limited for subgroup analyses such as albuminuria stratification. Third, convenience sampling was used, which risks selection bias; moreover, the equal distribution of albuminuria subgroups (20 normoalbuminuria, 20 microalbuminuria, 20 macroalbuminuria) is unlikely to occur naturally and likely reflects convenience rather than a true population distribution. Fourth, key potential confounders were not collected, including diabetes duration, medication use (metformin, SGLT2 inhibitors, statins, ACE inhibitors/ARBs), blood pressure, smoking status, and inflammatory markers such as C-reactive protein; their absence leaves residual confounding possible. Fifth, serum zinc is an acute-phase reactant that falls during

inflammation, and without measurement of inflammatory markers, we cannot determine whether lower zinc reflects true deficiency or inflammatory redistribution. Sixth, the estimated glomerular filtration rate (eGFR) was not reported, and we did not adjust for multiple comparisons, which increases the risk of type I error. Seventh, serum zinc has diurnal variation and is affected by meals; although we standardised collection to fasting morning samples, we did not verify recent meal timing beyond the 8-hour fast. Finally, this is a single-centre study, which limits generalisability to other populations or healthcare settings. These limitations argue for cautious interpretation and underscore the need for larger, prospective, multicentre studies with comprehensive covariate adjustment.

Conclusion

This study shows that Iraqi adults with T2DM have a broader pattern of abnormality extending beyond hyperglycaemia: higher albuminuria, higher serum creatinine, higher circulating VCAM-1, lower serum zinc, and an adverse lipid profile. Poorer glycaemic control is associated with a more atherogenic triglyceride-rich lipid pattern. However, VCAM-1 had limited discriminative ability (AUC 0.607) and was not a statistically significant independent predictor in univariable logistic regression (OR = 1.070, 95% CI 0.989–1.157, $P = 0.091$). Therefore, while VCAM-1 may reflect endothelial perturbation in T2DM, this study does not establish it as a clinically useful standalone marker. Larger prospective and multicentre studies with adjustment for confounders are needed.

List of Abbreviations

T2DM: Type 2 diabetes mellitus
 VCAM 1: Vascular cell adhesion molecule 1
 HbA1c: Glycated haemoglobin
 uACR: Urinary albumin to creatinine ratio
 LDL C: Low-density lipoprotein cholesterol
 HDL C: High-density lipoprotein cholesterol
 VLDL C: Very low-density lipoprotein cholesterol
 ROC: Receiver operating characteristic
 AUC: Area under the curve
 OR: Odds ratio
 CI: Confidence interval
 BMI: Body mass index
 eGFR: Estimated glomerular filtration rate
 CRP: C-reactive protein

Declarations

Ethics Approval and Consent to Participate

The study protocol was approved by the Institutional Review Board of the College of

Medicine, University of Babylon (approval number: UOB/COM/2024/1523). All procedures were conducted in accordance with the Declaration of Helsinki. All participants gave written informed consent.

Consent for publication

The authors gave consent for the publication of the work under the Creative Commons Attribution Non-Commercial 4.0 license.

Data Availability

The data supporting the findings are available from the corresponding author upon reasonable request, subject to institutional and ethical requirements.

Competing Interests

The authors declare no competing interests.

Funding

No specific external funding was received.

Author Contributions

THA did the conceptualisation, methodology, laboratory analysis, and wrote the original draft. BHR recruited participants, collected clinical data, conceptualised, supervised, analysed the statistics, reviewed and edited the writing. All authors approved the final version.

Acknowledgements

The authors thank the staff of the Imam Al-Hasan Centre for Diabetes and Endocrinology and Imam Al-Hasan Teaching Hospital, Karbala Health Directorate, for assistance with recruitment and sample collection.

References

- Atkins RC, Zimmet P. Diabetic kidney disease: act now or pay later. *Nephrology Dialysis Transplantation*. 2010 Feb 1;25(2):331-3. <https://doi.org/10.1093/ndt/gfp757>
- Care D. 11. Chronic kidney disease and risk management: standards of care in diabetes—2025. *Diabetes Care*. 2025 Jan 1;48:S239. <https://doi.org/10.2337/dc25-S011>
- Yang DR, Wang MY, Zhang CL, Wang Y. Endothelial dysfunction in vascular complications of diabetes: a comprehensive review of mechanisms and implications. *Frontiers in Endocrinology*. 2024 Apr 5;15:1359255. <https://doi.org/10.3389/fendo.2024.1359255>
- Parveen S, Malmberg M, Arulmurugananthavadivel A, Carlson N, Andersson C, Andersen CF, Jensen J, Anjum DZ, Elmegaard M, Eroglu T, Fosbøl E. Albuminuria in Type 2 Diabetes and Risk of

- Cardiorenal Outcomes. JACC: Advances. 2025 Nov 1;4(11):102254. <https://doi.org/10.1016/j.jacadv.2025.102254>
5. Li J, Zhang Y, Sun X, Guan H, Wang J, Zhao S, Tian C, Han M, Ma K, Li M. Specificity of endothelial cells in endothelial dysfunction of diabetic kidney disease and their crosstalk with neighboring cells: an updated review. Frontiers in Endocrinology. 2025 Dec 17;16:1742296. <https://doi.org/10.3389/fendo.2025.1742296>
6. Hegazy GA, Awan Z, Hashem E, Al-Ama N, Abunaji AB. Levels of soluble cell adhesion molecules in type 2 diabetes mellitus patients with macrovascular complications. Journal of International Medical Research. 2020 Oct;48(4):0300060519893858. <https://doi.org/10.1177/0300060519893858>
7. Ahmad R, Shaju R, Atfi A, Razzaque MS. Zinc and diabetes: a connection between micronutrient and metabolism. Cells. 2024 Aug 15;13(16):1359. <https://doi.org/10.3390/cells13161359>
8. Gembillo G, Visconti L, Giuffrida AE, Labbozzetta V, Peritore L, Lipari A, Calabrese V, Piccoli GB, Torreggiani M, Siligato R, Santoro D. Role of zinc in diabetic kidney disease. Nutrients. 2022 Mar 24;14(7):1353. <https://doi.org/10.3390/nu14071353>
9. Harkin C, Cobice D, Brockbank S, Bolton S, Johnston F, Strzelecka A, Watt J, Kurth MJ, Lamont JV, Fitzgerald P, Moore T. Biomarkers for detecting kidney dysfunction in type-2 diabetics and diabetic nephropathy subjects: a case-control study to identify potential biomarkers of DN to stratify risk of progression in T2D patients. Frontiers in Endocrinology. 2022 Jun 29;13:887237. <https://doi.org/10.3389/fendo.2022.887237>
10. Freitas EP, Cunha AT, Aquino SL, Pedrosa LF, Lima SC, Lima JG, Almeida MG, Sena-Evangelista KC. Zinc status biomarkers and cardiometabolic risk factors in metabolic syndrome: a case control study. Nutrients. 2017 Feb 22;9(2):175. <https://doi.org/10.3390/nu9020175>
11. Al-Fartosy AJ, Awad NA, Alsalmi SA. Clinical markers and some trace elements in patients with type-2 diabetic nephropathy: Impact of insulin resistance. The Journal of Medical Investigation. 2021;68(1.2):76-84. <https://doi.org/10.2152/jmi.68.76>
12. Alsaedy AM, Al-Rubaei ZM. Study of Adhesion Molecules in Diabetes Mellitus Type2 Iraqi Patients with Dyslipidemia. Journal of the Faculty of Medicine Baghdad. 2024 Jul 1;66(2):141-6. <https://doi.org/10.32007/jfacmedbagdad.6622273>
13. Mohammed HR, Othman RB, Alghurabi H, Hussein RM, Al-Obaidi Z, Abdeselem H. Comparison of clinical characteristics, microvascular complications and inflammatory markers in type 2 diabetic patients under insulin versus metformin treatment: A cross-sectional study at Karbala Diabetic Center, Iraq. Medicine. 2024 Nov 1;103(44):e40330. <https://doi.org/10.1097/MD.00000000000040330>
14. Care D. 2. Diagnosis and classification of diabetes: Standards of care in diabetes—2024. Diabetes Care. 2024 Jan;47(Suppl 1):S20-s42. <https://doi.org/10.2337/dc24-S002>
15. McCormick S, Ward GM, Trambas C, MacIsaac RJ. A review of albuminuria testing rates in people with diabetes mellitus: poor guideline adherence. Pathology. 2025 Sep 15. <https://doi.org/10.1016/j.pathol.2025.08.002>
16. Al-Asadi JN, Al-Naama LM. The role of non-HDL cholesterol and atherogenic indices in predicting poor glycemic control among type 2 diabetic patients in Basrah, Iraq. Qatar Medical Journal. 2022;2022(3):32. <https://doi.org/10.5339/qmj.2022.32>