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Serum Zinc Levels in Type 2 Diabetes Mellitus and Their Association with Diabetic Retinopathy and Glycemic Control

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Abstract

Background: Zinc is involved in a variety of enzymatic and antioxidant pathways, and hyperglycemia and oxidative stress are thought to contribute to diabetic microvascular injury in the retina. The biological plausibility of the association between circulating zinc and severity of DR and glycemic status is clinically relevant but not enough to conclude one way or the other. **Objectives**: To evaluate the serum zinc concentration in adults with type 2 diabetes mellitus, to compare the serum zinc concentration among the different grades of diabetic retinopathy and healthy controls, and to investigate the association between serum zinc and glycated hemoglobin (HbA1c). **Methods:** This hospital-based case-control study was carried out in the ophthalmic outpatient department of Al-Sader teaching hospital in Najaf, and Ibn-Alhaitham eye teaching hospital in Baghdad from August 2016 to March 2017. Patients with type 2 diabetes mellitus (T2DM) (40 without retinopathy, 25 with non-proliferative diabetic retinopathy (NPDR), and 25 with proliferative diabetic retinopathy (PDR)) and age- and sex-matched healthy controls were recruited as the sample. The level of zinc in serum was determined by the colorimetric 5-Br-PAPS method, while diabetic participants had their level of HbA1c measured. Minimal p values of < 0.05 were considered statistically significant and were reported in the source records, including ANOVA, chi-square testing, and Pearson correlation.

Results: Mean serum zinc was 87.1±13.9 µg/dL in controls, 63.8±15.0 µg/dL in diabetic participants without retinopathy, 59.8±13.9 µg/dL in NPDR, and 48.3±12.0 µg/dL in PDR. The group comparisons reported revealed that zinc levels in diabetic groups were lower than in the controls ($p<0.01$). The direct comparisons to the diabetes without retinopathy group were non-significant for NPDR ($p=0.311$) and PDR ($p=0.101$). Of diabetics, 68/90 (75.6%) were considered to have low zinc. We found that 26/40 (65.0%) did not have retinopathy, 18/25 (72.0%) had NPDR, and 24/25 (96.0%) had PDR; the reported odds ratio of PDR compared with no retinopathy is 0.08 (95% CI 0.01–0.63; $p=0.004$), but the coding/reference direction needs to be verified. Low zinc was recorded on 13/29 (44.8%) with optimal glycemic control, 18/24 (75.0%) with suboptimal control, and 37/37 (100%) with poor control ($p<0.01$).
Conclusion: The result indicates low mean serum Zn in diabetic participants; PDR shows the lowest mean serum Zn, and there is a strong descriptive trend between low Zn and poor glycemic control.

1. Introduction

Diabetes mellitus Type 2 is a chronic metabolic disorder that is caused by inadequate insulin production, inadequate insulin sensitivity, or a combination of both, which leads to hyperglycemia. Long-term hyperglycemia leads to injury in several organs, such as the retina. Diabetic retinopathy is a microvascular complication which can develop into proliferative DR with ischemic changes and pathological new vessel formation, which can threaten vision [1–5].

Diabetic retinopathy is a complex disorder. Clinical factors such as duration of diabetes and glycemic exposure are significant; oxidative stress, advanced glycation end products, altered cellular signalling and vascular endothelial growth factor pathways are associated with retinal microvascular damage [5-8]. The mechanisms involved in these studies allow investigation of antioxidant-related micronutrients, but do not provide proof that altering the concentration of these micronutrients will prevent or cure retinal disease.

Zinc is an essential trace mineral that is required for the function of many enzymes and transcription factors, immune function, cellular signalling, and antioxidant protection. It also plays a role in insulin synthesis, storage and secretion. The association of zinc with diabetes and the role of zinc as a regulator in metabolic physiology is complex and can be affected by dietary intake, disease duration, renal function, inflammatory status, medication therapy and many other factors [9–13].

The role of zinc in eye physiology and oxidative stress has also been mentioned. There have been studies on trace elements in diabetic complications like diabetic retinopathy in experimental and clinical literature, but the results are not always uniform across different populations and study designs [14-20]. Low serum zinc levels may represent a biomarker of metabolic status or disease burden but not be a primary cause of retinal damage. Moreover, serum zinc does not reflect the availability of zinc in the retina.

This study compared the serum zinc levels in diabetic patients (with and without DR) and normal control subjects in Najaf and Baghdad (Iraq). The primary aims were to compare the serum zinc level among the study groups and to evaluate the correlation between serum zinc and retinopathy category and HbA1c.

2. Materials and Methods

2.1 Study design and setting

The study is a case-control study done between August 2016 and March 2017 in Al-Sader Teaching Hospital in Najaf and in Ibn-Alhaitham Eye Teaching Hospital in Baghdad, Iraq, in the ophthalmic outpatient department.

2.2 Participants and groups

A total of 90 patients, aged above 30 years, with clinically diagnosed type 2 diabetes mellitus, were included in the study, with 40 healthy voluntary controls matched with the diabetes group according to age and sex. Of the diabetic participants, 40 did not have diabetic retinopathy, 25 had non-proliferative diabetic retinopathy, and 25 had proliferative diabetic retinopathy. No distinction was made between the sexes and socioeconomic status of the participants. A detailed recruitment flow was not provided in the source documents, nor was there a formal sample size calculation.

2.3 Eligibility and ophthalmic assessment

The participants included diabetic patients who had type 2 diabetes mellitus and/or NPDR or PDR and were diagnosed based on the WHO criteria in the source thesis. After mydriasis, slit-lamp bio microscopy and a 78-diopter condensing lens were used to examine both eyes. Renal failure, diuretics and malabsorption or chronic diarrhea was recorded as exclusion criteria, as was pregnancy or lactation.

2.4 Blood was collected from each individual, and laboratory measurements were performed.

Participants had a peripheral venous blood sample taken (5 mL). The sample was split into two tubes, one plain for the serum zinc and one EDTA tube for the HbA1c. Zinc was quantified in serum by the 5-bromo-PAPS colorimetric method with a kit obtained from an Egyptian biotechnology company. The reference values reported in the source were 72.6 - 127 µg/dL for males and 70.6 - 114 µg/dL for females. HbA1c was quantitated by a quantitative colorimetric method with a kit from Stanbio Laboratories. HbA1c was only measured in the diabetic participants.

2.5 Glycemic categories and statistical analysis.

The source defined optimal, suboptimal and poor glycemic control as HbA1c ≤6.8%, 6.8–7.6% and ≥7.6%, respectively. The cut-points overlap at 6.8% and at 7.6% as described in the original, and the category labels and rules for the cut-points should be verified against the original data before publishing. The data were analyzed in SPSS version 21. All continuous variables were summarized as mean ±sd and all means compared using ANOVA, categorical associations compared using chi-square and relationships between continuous variables compared using Pearson correlation. The level of statistical significance was considered $p<0.05$. The Pearson correlation coefficient and its precise p-value are not given in the source files.

3. Results

3.1 Participant characteristics

For analysis, there were 130 subjects; 90 of them had type 2 diabetes mellitus, and 40 were healthy controls. Mean ages were similar across groups (56.7±8.2 years in controls, 55.8±5.2 years in diabetes without retinopathy, 58.0±7.1 years in NPDR, and 58.7±7.6 years in PDR; $p=0.370$). No significant difference was also observed for sex distribution ($p=0.766$). Mean duration of diabetes was higher in each group of diabetic patients—8.1±4.7 years in patients without retinopathy, 10.8±5.0 years in NPDR and 12.0±4.5 years in PDR (reported $p<0.01$).

Table 1. Participant Characteristics

Characteristic	Health controls (n=40)	Diabetes no DR (n=40)	NPDR (n=25)	PDR (n=25)	Reported p-value
Age, years, mean ± SD	56.7 ± 8.2	55.8 ± 5.2	58.0 ± 7.1	58.7 ± 7.6	0.370
Male, n (%)	20 (50.0)	24 (60.0)	13 (52.0)	15 (60.0)	0.766
Female, n (%)	20 (50.0)	16 (40.0)	12 (48.0)	10 (40.0)	0.766
Diabetes duration, years, mean ± SD	—	8.1 ± 4.7	10.8 ± 5.0	12.0 ± 4.5	<0.01

Abbreviations: DR, diabetic retinopathy; NPDR, non-proliferative diabetic retinopathy; PDR, proliferative diabetic retinopathy; SD, standard deviation. **Note:** Values are transcribed from the results supplied. The statistical tests used to calculate the p-values were not specified at table level.

3.2 Serum zinc levels by study group

The healthy control group had the highest mean levels of serum zinc, while the lowest levels were seen with the PDR group. According to the source, diabetic subgroups exhibited p<0.01 when compared to controls. The mean zinc in the no-retinopathy group was, however, not significantly different from the NPDR (p=0.311) or PDR (p=0.101) groups. Comparisons should be considered separately and should not be interpreted as being statistically significant on a stage-to-stage basis.

Table 2. Serum Zinc Levels by Study Group

Group	n	Serum zinc, µg/dL (mean ± SD)	Range, µg/dL	Reported comparison
Healthy controls	40	87.1 ± 13.9	65–120	Reference
Diabetes no DR	40	63.8 ± 15.0	40–90	p = 0.01 vs controls
NPDR	25	59.8 ± 13.9	31–80	p = 0.01 vs controls; p = 0.311 vs no DR
PDR	25	48.3 ± 12.0	28–79	p = 0.01 vs controls; p = 0.101 vs no DR

The source tables are confusing whether the p values in the zinc table are for the overall test or for multiple different tests. Before submitting to the analysis plan, the original statistical output should be looked over. Abbreviations: DR, diabetic retinopathy; NPDR, non-proliferative diabetic retinopathy; PDR, proliferative diabetic retinopathy; SD, standard deviation. Note: Comparison values are given as supplied; see the statistical test and any multiple comparison correction before publication.

3.3 A low zinc level and retinopathy category.

The 90 diabetic patients were divided into two groups: 68 (75.6%) patients had low zinc levels, and 22 (24.4%) patients had normal zinc levels. Low zinc was recorded in 26/40 (65.0%) participants without retinopathy, 18/25 (72.0%) with NPDR, and 24/25 (96.0%) with PDR. The table reports an odds ratio of 0.08 (95% CI 0.01–0.63; p=0.004) for the PDR category and 0.72 (95% CI 0.24–2.14; p=0.557) for NPDR. The sign of the odds ratio doesn't appear to be consistent with the description of the proportion of low zinc in PDR, so the results should be coded and interpreted in reference to the original analysis.

Table 3. Low Zinc Level and Retinopathy Category

Retinopathy category	Low zinc, n (%)	Normal zinc, n (%)	Reported OR (95% CI)	Reported p-value
No retinopathy (reference)	26 (65.0)	14 (35.0)	1.00	—
NPDR	18 (72.0)	7 (28.0)	0.72 (0.24–2.14)	0.557
PDR	24 (96.0)	1 (4.0)	0.08 (0.01–0.63)	0.004
Total	**68 (75.6)**	**22 (24.4)**	—	—

OR, odds ratio; CI, confidence interval. Reported values and estimates are presented as they were provided in the results; the direction of coding needs to be checked.

Abbreviations: OR odds ratio; CI, confidence interval; NPDR, non-proliferative diabetic retinopathy; PDR, proliferative diabetic retinopathy.
Note: The reported PDR odds ratio appears inconsistent with the observed proportions of low zinc. Verify outcome and reference-category coding and recalculate the odds ratio and confidence interval from the raw data before publication.

3.4 Serum Zinc Levels:

In each category of retinopathy, there was no significant difference between the mean zinc levels of the two groups of participants with duration of diabetes of ≤10 years and >10 years (p=0.54 for participants without retinopathy, p=0.41 for NPDR, p=0.70 for PDR). In all categories, the mean zinc was significantly reduced in the PDR group compared to the non-retinopathy group both in the short (less than 1 year) and long (1 year or longer) duration strata.

Table 4. Serum Zinc Levels by Diabetes Duration and Retinopathy Category

Duration of diabetes	No DR, mean ± SD	NPDR, mean ± SD	PDR, mean ± SD	Reported p across retinopathy categories
≤10 years	62.9 ± 15.8	62.0 ± 13.0	49.3 ± 14.0	0.035
>10 years	66.4 ± 13.3	57.4 ± 15.0	47.4 ± 10.7	0.005
Within-category p-value	**0.54**	**0.41**	**0.70**	—

Abbreviations: DR, diabetic retinopathy; NPDR, non-proliferative diabetic retinopathy; PDR, proliferative diabetic retinopathy; SD, standard deviation.

Note: p-values for within category comparisons have been reproduced as reported. Review the original statistical techniques used to explain the comparison(s) shown as well as the adjustments applied for confounding variables for p-values among retinopathy categories.

The provided table does not include specific statistical tests for comparisons between retinopathy categories, or adjusted analyses.

3.5 Serum zinc and glycemic control.

The less well controlled groups had more hypozincemia. In the optimal-control category, 13/29 (44.8%) had low zinc and 16/29 (55.2%) had normal zinc. Of the 18 respondents in the suboptimal category, 18 (75.0%) had low zinc, and of the 37 respondents in the poor control category, the zinc level was low for all (100%). This reported association was significant statistically (p<0.01). Another negative correlation between serum zinc and HbA1c was also mentioned in the original abstract, but there was no correlation coefficient and exact p-value provided in the provided files.

Table 5. Serum Zinc Levels by Glycemic Control Category

Glycemic control category (as recorded)	Low zinc, n (%)	Normal zinc, n (%)	Total
Optimal (HbA1c ≤6.8%)	13 (44.8)	16 (55.2)	29
Suboptimal (HbA1c ≤6.8–7.6%)	18 (75.0)	6 (25.0)	24
Poor (HbA1c ≥7.6%)	37 (100.0)	0 (0.0)	37
Total	**68 (75.6)**	**22 (24.4)**	**90**

Abbreviations: HbA1c, glycated hemoglobin.

Note: The recorded HbA1c categories overlap at 6.8% and 7.6%. Confirm the intended thresholds against the original study protocol and raw data before finalizing the manuscript.

There is overlap between the source categories. The authors should check the definitions of the categories with the raw data and make sure that the wording is correct throughout the manuscript.

4. Discussion

In the hospital setting, the zinc levels in blood were found to be lower in those with type 2 diabetes as compared to healthy controls. This was also true for mean concentration in non-retinopathy, diabetes without retinopathy and PDR groups. This is a similar trend to that observed in the preceding reports on the changes in status of these trace elements in diabetes, and the studies on the zinc and its correlation with oxidative stress and diabetic retinopathy [11-13,17-20]. In practical terms, however, the study design is observational and cross-sectional and thus does not address the question of whether or not low zinc is a cause of retinal disease or of metabolic disturbance or other differences between the groups. There are two layers of interpretation for the main result. First, the zinc mean reported was lowest among the PDR group, and low zinc was descriptively very common in this group (96%). Second, the source reports that there were no significant differences in mean zinc between the no-retinopathy and NPDR groups (p=0.311) or the no-retinopathy and PDR group (p=0.101). Therefore, the numerical differences, although significant, are not statistically significant in the provided pairwise tests as differences in the mean zinc level between the retinopathy stages. A categorical analysis shows an association for PDR (p=0.004); however, the direction of the odds ratio does not agree with the raw proportions of low zinc. This difference might be either due to reverse coding or to a reference category problem and should not be used to draw a conclusion until it is addressed.

The second finding is that the proportion with low zinc rose from 44.8% in the optimal category to 75.0% in the suboptimal category and 100% in the poor-control category in a clear descriptive relationship. The provided abstract describes the association between serum zinc and HbA1c as inversely related to each other. This is biologically plausible as zinc is involved in some insulin-related processes and antioxidant defense, and hyperglycemia has been shown to lead to increased oxidative stress [9,10,12,17,18]. The strength and confidence in the continuous association are not available in the source materials, however. The categorical trend should not be cited as evidence of the association between zinc deficiency and poor glycemic control.

Another finding of the study was that mean duration of diabetes was greater in NPDR and PDR groups than in the group without retinopathy. Clinical correlates of retinopathy are also recognized, such as the duration of diabetes, which could also affect the relationship between zinc and retinopathy status. The provided analysis is based on the stratification by duration but does not include multivariable adjustments for either duration, age, sex, renal function in the range of non-excluded subjects, dietary

status, inflammatory markers or medication use. However, it is not possible to conclude if there is an independent association between zinc and retinopathy when potential confounding factors are taken into account.

The advantages of the study are that it compares healthy controls, it involves both NPDR and PDR groups, it uses a specified laboratory method for measuring the serum zinc level, and it measures HbA1c. It also explores a clinically relevant question in two hospital environments in Iraq. The shortcomings are the small sample size of the samples, and its selection at a hospital, which does not mention its sample size calculation; its description is not detailed; it does not have a detailed grading protocol; it is not masked; and the statistical reporting is incomplete. No Pearson correlation coefficient, full model output, and odds-ratio coding are given. The category boundaries for the HbA1c are also overlapped and thus must be corrected with the original dataset. Third, no formal ethics committee information is included in the files provided, only verbal consent information.

These results, when combined, warrant a further study of zinc in type 2 diabetes mellitus, but do not support the suggestion that zinc supplementation is beneficial for prevention/treatment of DR. Zinc supplementation in diabetes has been reported previously [12], although the present study was not a zinc supplementation study and did not measure the time course of changes in retinopathy and cannot prove a therapeutic effect. The associations themselves should not be used as a basis for clinical decisions regarding supplementation unless there is clear evidence indicating a deficiency.

5. Limitations

Observational design and limited sample size should be taken into account when interpreting the results. The study was conducted in two ophthalmic outpatient departments, which limited the generalizability of the study. The documents do not contain any reports of the prospective protocol, sample size calculation, modified regression function, or exact Pearson correlation coefficient or full statistical output. The reported PDR odds ratio seems to be conflicting with the ratios of low zinc and should be checked again. The cut points of the HbA1c categories overlap.

6. Conclusion

The serum zinc level decreased in the diabetic group compared with the healthy controls, and the lowest mean value was seen in the participants with PDR. Furthermore, low Zn was more common among participants who were categorized as having poor glycemic control. No significant mean difference was reported for the comparison between the diabetic patients with and without retinopathy for zinc, however. The odds-ratio coding should be verified because of the apparent categorical association with PDR. The findings suggest an association that deserves further investigation, but not proof of causation or that zinc supplements can prevent DR.

7. Recommendations

Larger (ideally multicenter) cohorts of subjects should be recruited, the grading of retinopathy and the threshold of zinc deficiency should be defined in advance, the entire outputs of the correlation and regression analysis should be reported, and the effects of diabetes duration, glycemic status, renal function, diet, inflammatory status and relevant drugs should be adjusted. Future follow-up studies would be useful to determine if serum zinc is a good predictor of the progression of retinopathy. To identify if supplementation can be beneficial for those who have been clinically diagnosed with zinc deficiency, interventional trials would be necessary. Zinc supplementation should not be touted as a proven intervention for DR prevention or treatment until there is supporting evidence.

Declarations

Ethics approval and consent to participate were obtained.
The study protocol and ethical considerations were reviewed and approved by the Scientific and Research Ethics Committee of the Iraqi Board for Medical Specializations (IBMS) and also by the Ministry of Health, Iraq. All individual participants who were enrolled in the study gave informed consent before participating. The procedures followed were in line with the ethical guidelines set by the Iraqi Ministry of Health research guidelines.

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Conflicts of interest
The authors state that they do not have any competing or financial interests with regard to the work described in this paper.

Data availability
Not all the data collected and analyzed in the present study are publicly available, since some are confidential or protected by institutional regulations, but they are accessible from the corresponding author on reasonable request.

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