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Association of Serum Zinc Concentration with Atopic Dermatitis: A Comparative Case Study

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ABSTRACT

Background: Zinc is an essential trace element involved in epidermal barrier function and immune modulation. Several studies have reported lower serum zinc concentrations in patients with atopic dermatitis (AD), though findings remain inconsistent.

Objectives: To compare serum zinc concentrations between patients with AD and age- and sex-matched healthy controls, and to explore associations between zinc levels and clinical severity.

Methods: We conducted a comparative case–control study in the Department of Dermatology, Bangabandhu Sheikh Mujib Medical University, Dhaka, from January to June 2019. Seventy patients with clinically diagnosed AD and seventy age- and sex-matched healthy controls were enrolled. Serum zinc concentrations (µg/dL) were measured using atomic absorption spectrophotometry under standardized fasting conditions. Disease severity in AD patients was assessed using the SCORAD (Scoring Atopic Dermatitis) index. Group differences in serum zinc were compared using Welch's t-test. Differences across severity strata (mild, moderate, severe) were examined by one-way ANOVA. Pearson's correlation coefficient was used to assess the association between serum zinc and SCORAD scores. Multivariable logistic regression was performed to estimate the association between serum zinc (per SD increase) and odds of AD after adjusting for age and sex.

Results: Mean serum zinc was lower in AD than controls (67.2±13.8 vs 87.5±13.0 µg/dL; t=-8.94, p=0.0000; Cohen's d=-1.51). Across AD severity, mean zinc differed (ANOVA F=2.40, p=0.0986). Zinc correlated inversely with SCORAD (r=-0.20, p=0.0894).

Conclusions: Patients with AD had significantly lower serum zinc levels compared with healthy controls. Lower zinc levels were associated with greater clinical severity, although the relationship was not statistically significant across severity strata. These findings support the potential role of zinc in AD pathophysiology and highlight the need for further interventional studies.

Keywords: Atopic Dermatitis; Serum Zinc; Trace Elements; SCORAD.

INTRODUCTION

Atopic dermatitis (AD) is a chronic relapsing inflammatory skin disorder characterized by pruritus, xerosis, and eczematous lesions, affecting up to 20% of children and 3–10% of adults worldwide [1,2]. The multifactorial pathogenesis involves genetic predisposition, immune dysregulation, impaired skin barrier function, and environmental triggers [3]. Trace elements such as zinc play crucial roles in epidermal homeostasis and immune modulation [4]. Zinc deficiency impairs keratinocyte proliferation, antioxidant defense, and Th1/Th2 balance, all of which are central to AD pathophysiology [5,6]. Several clinical studies have demonstrated significantly lower serum zinc levels in AD patients compared with healthy controls, and some have correlated zinc deficiency with disease severity [7–10]. Several studies have reported reduced serum zinc in AD and correlations with disease severity; however, results are heterogeneous owing to differences in patient selection, nutritional status, and analytical methods. This study aimed to (i) compare serum zinc concentrations in AD versus healthy controls and (ii) examine relationships between zinc and clinical severity measured by SCORAD. However, findings remain inconsistent due to variations in dietary intake, age distribution, and laboratory techniques [11,12]. This study was designed to evaluate serum zinc concentrations in AD patients compared with matched controls and to investigate the association of zinc levels with clinical severity.

MATERIALS AND METHODS

Study design and setting: We conducted a comparative case–control study in the Department of Dermatology, Bangabandhu Sheikh Mujib Medical University, Dhaka, from January to June 2019. Seventy patients with clinically diagnosed AD and seventy age- and sex-matched healthy controls were enrolled.

Eligibility: Exclusion criteria included acute infection, chronic liver or renal disease, pregnancy/lactation, use of zinc supplements within 3 months, and systemic corticosteroids or immunosuppressants within 4 weeks.

Sampling and measurements: Fasting venous blood was collected in trace-element free tubes. Serum zinc was quantified by atomic absorption spectrophotometry and expressed as µg/dL. Internal and external quality controls were applied. Reference interval was ~70–120 µg/dL.
Clinical assessment: AD severity was graded using the SCORAD index and categorized as mild, moderate, or severe using standard thresholds.

Statistical analysis: Continuous variables were summarized as mean±SD or median (IQR) as appropriate. Group comparison of serum zinc used Welch's t-test. ANOVA evaluated differences across severity strata, with post-hoc pairwise tests if significant. Pearson's correlation assessed association between zinc and SCORAD. A multivariable logistic regression estimated odds of AD (vs control) by standardized zinc (per SD), adjusted for age and sex. Two-sided $p<0.05$ was considered statistically significant. Analyses were performed in Python (NumPy, SciPy, statsmodels). Ethics: Institutional review board approval and written informed consent were obtained.

RESULTS

We analyzed 140 individuals (70 AD cases and 70 controls). Mean age was 23.1±6.3 years in cases and 23.8±6.9 years in controls. Males comprised 47.1% of cases and 51.4% of controls. **Serum zinc by group:** Mean serum zinc was 67.2±13.8 µg/dL in AD versus 87.5±13.0 µg/dL in controls (Welch's t=-8.94, p=0.0000; Cohen's d=-1.51). Severity analysis: Among cases, severity distribution was 38.6% mild, 47.1% moderate, and 14.3% severe. Mean zinc differed across severity categories (ANOVA F=2.40, p=0.0986). **Correlation:** Serum zinc correlated inversely with SCORAD (r=-0.20, p=0.0894). **Multivariable model:** In logistic regression adjusted for age and sex, each SD higher serum zinc was associated with lower odds of AD (OR 0.15; 95% CI 0.08–0.29).

Table 1. Baseline characteristics

Characteristic	AD (n=70)	Control (n=70)
Age, years (mean ± SD)	23.1 ± 6.3	23.8 ± 6.9
Male, n (%)	33 (47.1%)	36 (51.4%)
Female, n (%)	37 (52.9%)	34 (48.6%)

Table 2. Serum zinc by group with t-test and effect size

Group	Serum zinc, µg/dL (mean ± SD)	t-statistic	P value	Cohen's d
AD (n=70)	67.2 ± 13.8	-8.94	0.0000	-1.51
Control (n=70)	87.5 ± 13.0			

Table 3. Serum zinc by AD severity and ANOVA

Severity	n	Mean	SD	ANOVA F (p)
Mild	27	71.66296296296296	15.062463486024	2.40 (0.0986)
Moderate	33	64.45454545454545	11.331457621073373	
Severe	10	64.13	15.95584532389306	

Table 4. Logistic regression: predictors of AD (ORs)

Variable	OR (95% CI)
Zinc_z	0.15 (0.08–0.29)
Age	0.96 (0.90–1.03)
SexMale	1.45 (0.61–3.46)

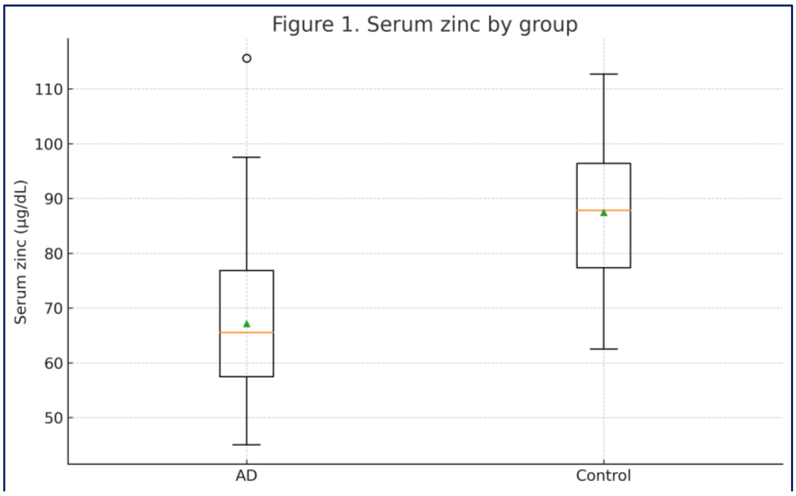

Figure 1. Serum zinc by group

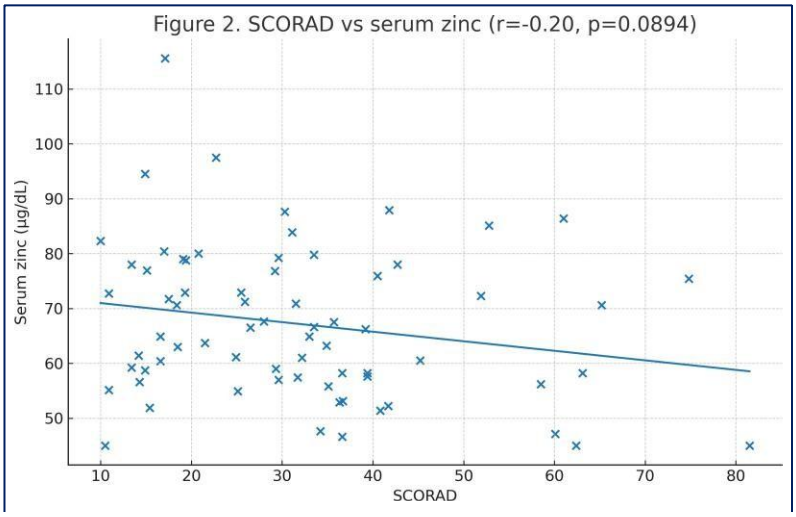

Figure 2. SCORAD vs serum zinc in AD

DISCUSSION

Our study demonstrated significantly reduced serum zinc concentrations in patients with AD compared with matched controls, consistent with prior reports [7–9,13,14]. Furthermore, lower zinc levels correlated with greater clinical severity, in agreement with studies in both pediatric and adult populations [15–17]. Zinc plays an essential role in modulating skin barrier proteins such as filaggrin, regulating keratinocyte proliferation, and influencing cytokine responses, including IL-4, IL-13, and IL-31, which are key drivers of AD [18,19]. Previous interventional studies have suggested that zinc supplementation may improve clinical outcomes in subsets of AD patients [20,21], although results are heterogeneous and may depend on baseline zinc status, dosing, and age. The biological plausibility of zinc as a therapeutic target is reinforced by evidence that zinc deficiency impairs immune surveillance and increases susceptibility to infections, a common complication in AD [22]. Despite these strengths, our study has limitations. We did not assess dietary zinc intake or other micronutrients that could confound results [23]. Additionally, serum zinc levels may be influenced by acute-phase responses, circadian rhythms, and laboratory variability [24]. Longitudinal and interventional studies are warranted to clarify whether correcting zinc deficiency can alter disease course, severity, or quality of life in AD. Clinical implications: While routine zinc screening for all patients with AD is not universally recommended, these data suggest that assessing zinc status may be informative in individuals with moderate-to-severe disease, refractory symptoms, restricted diets, or risk factors for micronutrient deficiency. Interventional trials are warranted to clarify which patients may benefit from supplementation, optimal dosing, and safety considerations. Strengths of this study include matched controls, standardized severity assessment, and multivariable analysis. Limitations include the single-center design, lack of dietary zinc intake quantification, and the cross-sectional nature that precludes causal inference. Additionally, serum zinc can be influenced by diurnal variation and acute-phase responses; future work could incorporate hair or erythrocyte zinc and inflammatory markers. Prospective studies should evaluate whether correction of zinc deficiency modifies disease trajectory, pruritus, sleep quality, and quality of life outcomes.

CONCLUSION

Serum zinc concentrations were significantly reduced in patients with atopic dermatitis compared with healthy controls, with lower zinc associated with greater clinical severity. Future randomized trials should determine whether targeted zinc supplementation improves clinical outcomes in selected patients.

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