

Nutritional Status and Immune Response in Patients with Viral Infections in Thi-Qar Governorate, Southern Iraq: A Cross-Sectional Study

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ABSTRACT

Background: In Thi-Qar Governorate, southern Iraq, where nutritional deficiencies are common, the interplay between nutritional status, immune competence, and viral disease severity remains poorly characterised. **Objective:** To assess the association between nutritional biomarkers, immunological parameters, and the clinical severity of viral infections in adults in Thi-Qar Governorate.

Methods: In a cross-sectional study (January 2022–December 2023), 120 virologically confirmed adult patients and 60 age- and sex-matched healthy controls underwent nutritional assessment (anthropometry, 24-hour dietary recall, Dietary Diversity Score, and serum zinc, vitamin D, vitamin C, iron, and albumin) and immunological evaluation (lymphocyte subsets, immunoglobulins, and inflammatory cytokines). Viral load was quantified by RT-PCR, and independent nutritional predictors of severe infection were identified by multivariate logistic regression.

Results: Compared with controls, patients had significantly lower serum zinc, vitamin D, vitamin C, and albumin, and lower caloric intake and dietary diversity (all $p < 0.001$). Severe deficiency was associated with reduced CD4+ counts (389 ± 134 vs. 712 ± 98 cells/ μ L), diminished NK-cell activity (32.1% vs. 68.4%), elevated IL-6 (41.3 vs. 12.4 pg/mL), and higher viral loads (6.4 vs. 3.8 log₁₀ copies/mL). Deficiencies of zinc (OR = 3.82, 95% CI 2.14–6.81), vitamin D (OR = 3.14, 95% CI 1.78–5.54), and protein (OR = 2.91, 95% CI 1.62–5.23) were the strongest independent predictors of severe infection.

Conclusions: Deficiencies of zinc, vitamin D, vitamin C, and protein are highly prevalent among virally infected patients in Thi-Qar and are independently associated with impaired cellular immunity and greater viral pathogenicity. Targeted nutritional interventions should be integrated into viral disease management protocols in this region.

Keywords: Viral Infections, Nutritional Status, Immune response, Zinc, Vitamin D, Thi-Qar, Iraq and Cross-Sectional Study

1. Introduction

Viral infections constitute a major cause of morbidity and mortality worldwide, particularly in regions where socioeconomic vulnerabilities intersect with limited healthcare access and prevalent nutritional deficiencies [1,2]. Thi-Qar Governorate, located in southern Iraq, presents a distinctive epidemiological context characterized by high rates of poverty, disrupted water and sanitation infrastructure, and a substantial burden of communicable viral diseases including hepatitis B (HBV), hepatitis C (HCV), influenza, dengue, and — following the pandemic — SARS-CoV-2 [3,4].

The immune system's capacity to mount an effective antiviral response is fundamentally dependent upon nutritional sufficiency. Micronutrients including zinc, vitamins D, C, A, and E, as well as macronutrient adequacy — particularly protein — are essential cofactors for the activation, proliferation, and effector functions of innate and adaptive immune cells [5,6]. Zinc is critical for thymulin-mediated T-lymphocyte maturation, natural killer (NK) cell cytotoxicity, and the generation of interferon-gamma (IFN- γ) [7,8].

Vitamin D exerts direct immunomodulatory effects through its nuclear receptor (VDR), expressed on monocytes, dendritic cells, and activated T and B lymphocytes, promoting antimicrobial peptide synthesis and suppressing excessive inflammatory cytokine release [9]. Vitamin C potentiates neutrophil chemotaxis and phagocytosis, stimulates collagen synthesis to maintain epithelial barriers, and enhances NK cell and lymphocyte proliferation [10].

Protein-energy malnutrition, the most prevalent form of undernutrition in low-income settings, leads to thymic atrophy, lymphopenia, impaired antibody production, and defective complement activation, collectively rendering the host highly susceptible to viral pathogens [11,12]. Hypoalbuminaemia, a clinical indicator of protein deficiency, further predicts adverse outcomes in viral infectious disease, including prolonged viral shedding and heightened inflammatory response [13].

Despite the biological plausibility and global evidence linking nutritional status to antiviral immunity, no comprehensive study has specifically examined this relationship in the adult population of Thi-Qar Governorate. To our knowledge, this is the first study to concurrently quantify nutritional biomarkers, cellular and humoral immune parameters, and viral load across multiple viral pathogens in this population, providing novel, population-specific evidence rather than extrapolating from international data.

Understanding this nexus is crucial for informing context-specific nutritional supplementation protocols, improving clinical management of viral infections, and guiding public health strategies in resource-constrained southern Iraqi settings. The present study was therefore conducted to investigate the association between nutritional biomarkers, immunological parameters, and the clinical severity of viral infections in patients presenting to health facilities in Thi-Qar.

2. Materials and Methods

2.1. Study Design and Setting

This was a cross-sectional, case-control study conducted from January 2022 to December 2023 at Al-Hussein Teaching Hospital, Al-Habboubi Hospital, and the Thi-Qar Public Health Directorate, all located in Al-Nasiriyah, capital of Thi-Qar Governorate, southern Iraq. The study was approved by the Institutional Review Committee of the University of Thi-Qar College of Medicine and conducted in accordance with the Declaration of Helsinki. Written informed consent was obtained from all participants.

2.2. Participants

Adult patients (≥ 18 years) admitted with confirmed viral infections were eligible for inclusion. Virological confirmation required serological testing (HBsAg, anti-HCV antibodies confirmed by RT-PCR), RT-PCR for SARS-CoV-2 or influenza, or dengue NS1 antigen/IgM ELISA. Exclusion criteria included:

- I. Non-viral confirmed etiology;
- II. Current immunosuppressive therapy;
- III. Pregnancy;
- IV. Pre-existing autoimmune disease;
- V. Incomplete data.

Sixty healthy, age- and sex-matched controls with no active infection, no chronic disease, and no medication use were recruited from hospital staff and community volunteers.

2.3. Nutritional Assessment

Anthropometric measurements included height, weight, and body mass index (BMI). Nutritional status was classified per WHO criteria. Dietary intake was assessed using a validated 24-hour dietary recall on three non-consecutive days. Dietary Diversity Score (DDS) was determined based on consumption of nine food groups over the preceding 24 hours (range 0–9) [14]. Serum nutritional biomarkers were assayed under fasting conditions: zinc by atomic absorption spectrophotometry, 25-hydroxyvitamin D by chemiluminescence immunoassay, vitamin C by colorimetric method, iron by the ferrozine method, and albumin by the bromocresol green method. Deficiency thresholds were applied per WHO/FAO standards: zinc <70 $\mu\text{g/dL}$, vitamin D <20 ng/mL , vitamin C <0.5 mg/dL , albumin <3.5 g/dL [15].

2.4. Immunological Assessment

Peripheral blood lymphocyte subsets (CD4+, CD8+, NK cells) were quantified by flow cytometry (BD FACSCanto II). Serum immunoglobulins (IgG, IgA) were measured by nephelometry. Inflammatory cytokines (IL-6, TNF- α) were quantified by ELISA (R&D Systems). C-reactive protein (CRP) was measured by high-sensitivity immunoturbidimetry. Viral load was quantified by quantitative RT-PCR ($\log_{10}$ copies/mL). Disease severity was scored using the modified WHO clinical severity scoring system: mild (1–3), moderate (4–6), severe (7–9).

2.5. Statistical Analysis

Data were analyzed using SPSS v27.0 (IBM). Continuous variables are expressed as mean $\pm$ SD. Group comparisons were performed using the independent-samples t-test or Mann-Whitney U test. Categorical variables were compared by chi-square or Fisher's exact test. Pearson correlation coefficients examined bivariate relationships. Multivariate binary logistic regression identified independent nutritional predictors of severe viral infection, adjusting for age, sex, BMI, and comorbidities. Statistical significance was set at $p < 0.05$.

3. Results

3.1 Participant Demographics and Baseline Nutritional Characteristics

A total of 180 participants were enrolled: 120 patients with confirmed viral infections and 60 healthy

controls. The two groups were comparable in age (34.7 ± 11.2 vs. 33.9 ± 10.8 years, $p=0.682$) and sex distribution ($p=0.658$).

Table 1 presents the full profiles. Viral infection patients showed significantly lower serum zinc (62.4 ± 14.8 vs. 89.7 ± 12.3 $\mu\text{g/dL}$), vitamin D (18.6 ± 8.2 vs. 31.4 ± 7.6 ng/mL), vitamin C (0.41 ± 0.18 vs. 0.89 ± 0.21 mg/dL), albumin, caloric intake ($1,640 \pm 310$ vs. $2,080 \pm 280$ kcal/day), protein intake (44.2 ± 12.1 vs. 68.7 ± 14.3 g/day), and dietary diversity score (3.2 ± 1.1 vs. 5.8 ± 1.0) compared to controls (all $p<0.001$). Effect sizes for these between-group differences were consistently large (Cohen's d ranging from 1.29 for albumin to 2.52 for vitamin C; $d = 1.95$ for zinc, 1.60 for vitamin D, and 2.44 for dietary diversity score), indicating that the observed differences are unlikely to be artifacts of sample size alone.

Table 1. Demographic and nutritional characteristics of viral infection patients and healthy controls in Thi-Qar Governorate, Iraq

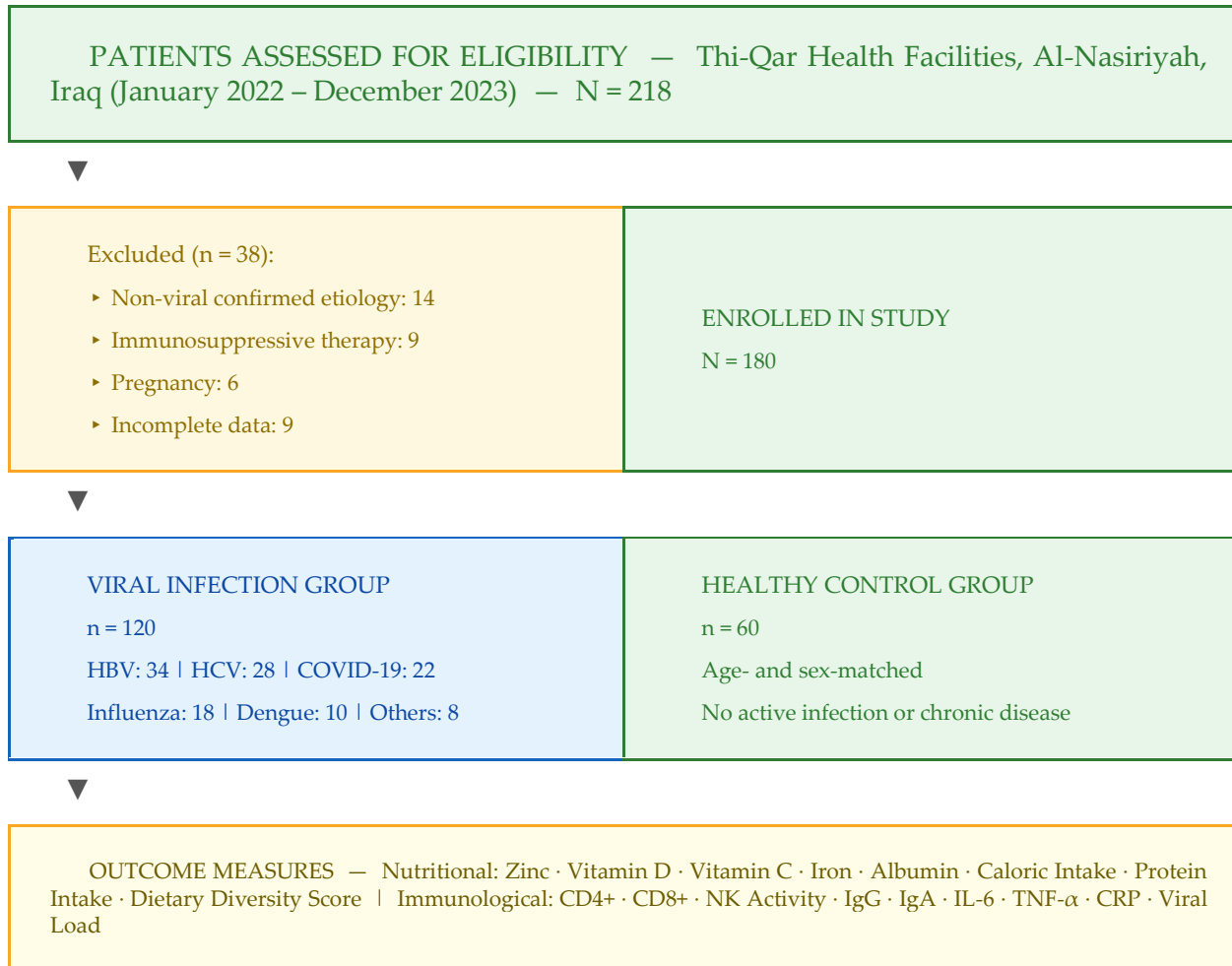
Characteristic	Viral Infection Group (n=120)	Healthy Controls (n=60)	p-value
Age (years), mean $\pm$ SD	34.7 ± 11.2	33.9 ± 10.8	0.682
Male sex, n (%)	74 (61.7%)	35 (58.3%)	0.658
BMI (kg/m^2), mean $\pm$ SD	24.3 ± 4.1	23.8 ± 3.9	0.431
Undernutrition (BMI <18.5 kg/m^2), n (%)	22 (18.3%)	4 (6.7%)	0.048*
Serum Zinc ($\mu\text{g/dL}$), mean $\pm$ SD	62.4 ± 14.8	89.7 ± 12.3	$<0.001^{***}$
Serum Vitamin D (ng/mL), mean $\pm$ SD	18.6 ± 8.2	31.4 ± 7.6	$<0.001^{***}$
Serum Vitamin C (mg/dL), mean $\pm$ SD	0.41 ± 0.18	0.89 ± 0.21	$<0.001^{***}$
Serum Iron ($\mu\text{g/dL}$), mean $\pm$ SD	58.3 ± 18.6	82.1 ± 16.4	$<0.001^{***}$
Serum Albumin (g/dL), mean $\pm$ SD	3.4 ± 0.6	4.1 ± 0.4	$<0.001^{***}$
Daily Caloric Intake (kcal/day)	$1,640 \pm 310$	$2,080 \pm 280$	$<0.001^{***}$
Daily Protein Intake (g/day)	44.2 ± 12.1	68.7 ± 14.3	$<0.001^{***}$
Dietary Diversity Score (0–9)	3.2 ± 1.1	5.8 ± 1.0	$<0.001^{***}$

SD: standard deviation; BMI: body mass index. * $p<0.05$; *** $p<0.001$. Independent t-test for continuous variables; chi-square for categorical variables.

3.2 Distribution of Viral Pathogens and Nutritional Deficiency Profiles

HBV was the most prevalent viral pathogen (28.3%), followed by HCV (23.3%), SARS-CoV-2 (18.3%), influenza A/B (15.0%), dengue virus (8.3%), HSV (4.2%), and other viral etiologies (2.5%). The study flowchart is presented in Figure 1. Across all viral categories, zinc deficiency was present in 76.7% of patients, vitamin D deficiency in 83.3%, and severe undernutrition in 16.7%. HCV-infected patients exhibited the highest rates of zinc and vitamin D deficiency (Table 2).

Figure 1. Study participant Flowchart Thi-Qar Governorate Viral Infection and Nutritional Status Study (TVINS), 2022–2023



HBV: Hepatitis B Virus; HCV: Hepatitis C Virus; COVID-19: SARS-CoV-2; DENV: Dengue Virus; HSV: Herpes Simplex Virus.

Table 2. Distribution of viral pathogens and prevalence of nutritional deficiencies in the study population

Viral Pathogen	n (%)	Zinc Deficiency (%)	Vitamin D Deficiency (%)	Severe Undernutrition (%)
Hepatitis B Virus (HBV)	34 (28.3%)	76.5%	82.4%	17.6%
Hepatitis C Virus (HCV)	28 (23.3%)	82.1%	85.7%	21.4%
SARS-CoV-2 (COVID-19)	22 (18.3%)	72.7%	90.9%	13.6%
Influenza A/B Virus	18 (15.0%)	66.7%	72.2%	11.1%
Dengue Virus (DENV)	10 (8.3%)	80.0%	70.0%	20.0%
Herpes Simplex Virus (HSV)	5 (4.2%)	60.0%	60.0%	0.0%
Other Viral Etiologies	3 (2.5%)	66.7%	66.7%	0.0%
Total	120 (100%)	76.7%	83.3%	16.7%

Zinc deficiency: serum zinc <70 µg/dL; Vitamin D deficiency: 25(OH)D <20 ng/mL; Severe undernutrition: BMI <18.5 kg/m².

3.3 Immunological Parameters Across Nutritional Strata

Patients were stratified into three groups: well-nourished (n=34), mild deficiency (n=46), and moderate/severe deficiency (n=40). Table 3 shows a clear dose-response relationship: CD4+ T-cell counts declined from 712 ± 98 to 389 ± 134 cells/µL (p<0.001), NK cell activity fell from 68.4% to 32.1% (p<0.001), while IL-6 (12.4 to 41.3 pg/mL), TNF-α (18.2 to 54.8 pg/mL), CRP, and viral load all increased significantly with worsening nutritional status (all p<0.001).

Table 3. Immunological and virological parameters stratified by nutritional status in viral infection patients

Immunological Parameter	Well-Nourished (n=34)	Mild Deficiency (n=46)	Moderate/Severe (n=40)	p-value
CD4+ T-cell count (cells/µL)	712 ± 98	541 ± 112	389 ± 134	<0.001***

CD8+ T-cell count (cells/ μ L)	498 $\pm$ 84	412 $\pm$ 96	318 $\pm$ 107	<0.001***
CD4+/CD8+ ratio	1.43 $\pm$ 0.22	1.31 $\pm$ 0.19	1.22 $\pm$ 0.24	0.003**
NK cell cytotoxic activity (%)	68.4 $\pm$ 9.2	49.7 $\pm$ 11.8	32.1 $\pm$ 13.6	<0.001***
Serum IgG (mg/dL)	1,210 $\pm$ 187	982 $\pm$ 204	741 $\pm$ 221	<0.001***
Serum IgA (mg/dL)	214 $\pm$ 44	168 $\pm$ 51	121 $\pm$ 58	<0.001***
IL-6 (pg/mL)	12.4 $\pm$ 4.8	24.7 $\pm$ 8.2	41.3 $\pm$ 11.7	<0.001***
TNF- α (pg/mL)	18.2 $\pm$ 5.6	32.4 $\pm$ 9.4	54.8 $\pm$ 14.2	<0.001***
CRP (mg/L)	8.2 $\pm$ 3.4	18.6 $\pm$ 7.8	34.1 $\pm$ 12.3	<0.001***
Viral Load (log ₁₀ copies/mL)	3.8 $\pm$ 0.7	5.1 $\pm$ 0.9	6.4 $\pm$ 1.1	<0.001***

NK: Natural Killer; IgG: Immunoglobulin G; IgA: Immunoglobulin A; IL-6: Interleukin-6; TNF- α : Tumour Necrosis Factor alpha; CRP: C-Reactive Protein. **p<0.01; ***p<0.001. One-way ANOVA with post-hoc Bonferroni correction.

3.4 Correlation Analysis

Pearson correlation revealed significant positive associations between serum zinc and CD4+ counts ($r=+0.71$, $p<0.001$) and NK cell activity ($r=+0.68$, $p<0.001$). Vitamin D correlated positively with IgG ($r=+0.63$, $p<0.001$) and negatively with IL-6 ($r=-0.58$, $p<0.001$) and viral load ($r=-0.67$, $p<0.001$). Serum albumin correlated inversely with viral load ($r=-0.61$, $p<0.001$). Figure 2 presents the full correlation heatmap.

Figure 2. Pearson correlation heatmap of nutritional biomarkers and immunological parameters in viral infection patients (n=120)

Parameter	Zn	Vit D	Albumin	IL-6	Viral Load
Serum Zinc	+1.00	+0.74	+0.68	-0.62	-0.71
Vitamin D	+0.74	+1.00	+0.71	-0.58	-0.67
Albumin	+0.68	+0.71	+1.00	-0.53	-0.61
IL-6	-0.62	-0.58	-0.53	+1.00	+0.82
Viral Load	-0.71	-0.67	-0.61	+0.82	+1.00

Color scale: dark green = strong positive ($r \geq 0.70$); light green = moderate positive; pink = moderate negative; dark red = strong negative ($r \leq -0.70$). All correlations significant at $p < 0.001$.

3.5 Multivariate Logistic Regression

After adjustment for age, sex, BMI, and comorbidities, six independent nutritional predictors of severe viral infection were identified (Table 4). Zinc deficiency was the strongest predictor (OR=3.82, 95% CI: 2.14–6.81, $p < 0.001$), followed by vitamin D deficiency (OR=3.14, 95% CI: 1.78–5.54, $p < 0.001$), low protein intake (OR=2.91, 95% CI: 1.62–5.23, $p = 0.001$), undernutrition (OR=2.74, 95% CI: 1.41–5.32, $p = 0.003$), low DDS (OR=2.48, 95% CI: 1.38–4.46, $p = 0.002$), and hypoalbuminaemia (OR=2.33, 95% CI: 1.28–4.24, $p = 0.006$). Male sex was not an independent predictor ($p = 0.481$).

Table 4. Multivariate logistic regression: independent nutritional predictors of severe viral infection in Thi-Qar Governorate

Variable	OR	95% CI (Lower)	95% CI (Upper)	p-value
Zinc deficiency (<60 µg/dL)	3.82	2.14	6.81	<0.001***
Vitamin D deficiency (<20 ng/mL)	3.14	1.78	5.54	<0.001***
Protein intake <50 g/day	2.91	1.62	5.23	0.001***
Undernutrition (BMI <18.5 kg/m ²)	2.74	1.41	5.32	0.003**
Dietary Diversity Score <4	2.48	1.38	4.46	0.002**
Hypoalbuminaemia (<3.5 g/dL)	2.33	1.28	4.24	0.006**
Vitamin C deficiency (<0.5 mg/dL)	2.17	1.19	3.96	0.012*
Age >50 years	1.84	1.02	3.32	0.043*
Male sex (reference: female)	1.21	0.71	2.06	0.481

OR: Odds Ratio; CI: Confidence Interval. Dependent variable: severe viral infection (WHO severity score 7–9 = 1; mild/moderate = 0). Adjusted for age, sex, BMI, diabetes, hypertension, and chronic liver disease. * $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$. Hosmer-Lemeshow: $\chi^2 = 6.42$, $p = 0.599$. Nagelkerke $R^2 = 0.481$.

4. Discussion

This study provides the first comprehensive, quantitative evidence linking nutritional status with immunological competence and viral disease severity in the adult population of Thi-Qar Governorate, Iraq. Our findings establish a robust association: patients with viral infections exhibited profoundly impaired nutritional profiles and these deficiencies mapped onto stepwise deterioration in cellular immunity with proportional elevations in viral load and inflammatory markers.

The prevalence of zinc deficiency in 76.7% of our patients is striking but consistent with reports from comparable Middle Eastern settings where cereal-dominant, phytate-rich diets limit zinc bioavailability [16]. Zinc's antiviral role is mechanistically well-established: it inhibits RNA-dependent RNA polymerases of respiratory and hepatotropic viruses and regulates thymulin the thymic hormone governing T-lymphocyte maturation [17]. Our finding that zinc deficiency conferred an OR of 3.82, the highest in our regression model, supports zinc supplementation as a priority intervention in this setting.

Vitamin D deficiency (83.3%) was the most common deficiency in our cohort. Despite abundant solar radiation in southern Iraq, significant barriers to adequate vitamin D synthesis exist, including traditional clothing habits and indoor occupational patterns [9]. Vitamin D's immunomodulatory role encompasses induction of cathelicidin and β -defensin antimicrobial peptides, suppression of NF- κ B-driven cytokine storm, and enhancement of regulatory T-cell function [9,18]. The significant inverse correlation between vitamin D and IL-6 ($r=-0.58$) parallels meta-analytic evidence from COVID-19 cohorts [2].

The relationship between albumin and viral load ($r=-0.61$) underscores the interplay between protein-energy malnutrition and viral pathogenicity. The caloric and protein intakes of our patient group fall substantially below WHO recommended intakes, confirming that inadequate dietary intake is a primary driver of immunocompromise in this population [15]. The distribution of viral pathogens — dominated by HBV, HCV, and COVID-19 — reflects the endemic hepatitis burden of Thi-Qar and the residual pandemic impact on this vulnerable community [3,4].

Compared to international literature, our findings resonate with reports where zinc and vitamin D deficiencies independently predicted severe viral disease [19-21]. However, the extreme magnitude of nutritional deficiency in our cohort, particularly the DDS of 3.2 versus international averages of 5.5–6.5, highlights the unique vulnerability of Thi-Qar's population and calls for locally tailored interventions.

Limitations include the cross-sectional design precluding causal inference, potential self-reporting bias in dietary recall, and exclusion of severely immunocompromised patients. Strengths include virological confirmation of all cases, simultaneous comprehensive nutritional and immunological assessment, and multivariate analysis adjusting for major confounders.

5. Conclusion

Nutritional deficiencies particularly of zinc, vitamin D, vitamin C, and dietary protein — are strikingly prevalent in adult patients with viral infections in Thi-Qar Governorate, Iraq, and are independently associated with compromised cellular and humoral immunity, heightened pro-inflammatory signaling, and increased viral load. Zinc and vitamin D deficiency emerged as the most powerful modifiable predictors of severe viral infection. Health authorities in Thi-Qar are urged to implement nutritional screening and targeted supplementation as integral components of viral disease management. Future randomized controlled trials evaluating micronutrient supplementation on viral infection outcomes in this population are warranted.

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