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Original Research Article

ASSOCIATION BETWEEN VITAMIN D DEFICIENCY AND OBESITY: A HOSPITAL-BASED MATCHED CASE-CONTROL STUDY IN TAMIL NADU

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ABSTRACT

Background: Vitamin D deficiency and obesity are major global public health challenges. Evidence suggests a significant association between the two, but data from South Indian adults are scarce. This study aimed to assess the association between vitamin D deficiency and obesity in a tertiary care setting in Tamil Nadu.

Materials and Methods: A hospital-based matched case-control study was conducted from August to December 2024 at MAPIMS Hospital. We enrolled 166 participants: 83 obese cases (BMI ≥25 kg/m²) and 83 age and sex matched normal-BMI controls (BMI 18.5-22.9 kg/m²). Serum 25-hydroxyvitamin D [25(OH)D] levels were measured. Data on demographics, lifestyle, diet, and comorbidities were collected via a validated questionnaire. Vitamin D deficiency was defined as serum 25(OH)D <20 ng/mL. Statistical analyses included Chi-square tests, t-tests, ANOVA, and multivariable logistic regression.

Results: Vitamin D deficiency was significantly more prevalent in the obese group (77.4%) than in controls (22.6%) (p<0.001). Obese individuals had nine-fold higher degrees of deficiency (OR: 9.0, 95% CI: 4.43-18.32). Mean serum 25(OH)D was significantly lower in obese participants (24.44 ± 25.12 ng/mL) versus controls (49.92 ± 26.89 ng/mL) (p<0.001). Obese subjects also had significantly less daily outdoor exposure and lower fish intake (p<0.001). In multivariable analysis, obesity, reduced outdoor exposure, higher systolic blood pressure, and an adverse lipid profile were independent predictors of vitamin D deficiency.

Conclusion: This study demonstrates a strong association between obesity and vitamin D deficiency among adults in Tamil Nadu. The findings highlight the need for routine vitamin D assessment and management in obese populations, with emphasis on lifestyle modifications.

Keywords: Vitamin D, Obesity, India, Case-control, 25-hydroxyvitamin D, Adults.

INTRODUCTION

Vitamin D deficiency is a pervasive global health issue, with an estimated one billion individuals affected, contributing to a spectrum of skeletal and extra-skeletal disorders.[1] Its role has expanded beyond classical calcium and bone metabolism to include modulation of immune function, cardiovascular health, and glucose metabolism.[2] Concurrently, obesity has reached epidemic proportions worldwide, acting as a major risk factor for chronic diseases such as diabetes, hypertension, and cardiovascular disorders. A compelling body of epidemiological research indicates a significant, bidirectional association between these two prevalent conditions.[3] The mechanisms underlying this association are multifactorial and may operate in both directions. Obesity may lead to lower circulating

vitamin D levels through volumetric dilution and sequestration of the fat-soluble vitamin in expanded adipose tissue, reducing its bioavailability.[4] Lifestyle patterns associated with obesity, notably reduced outdoor physical activity, further limit the cutaneous synthesis of vitamin D. Conversely, vitamin D insufficiency may influence metabolic pathways, potentially affecting adipogenesis, insulin sensitivity, and systemic inflammation, thereby contributing to weight gain and metabolic dysfunction.[5] While observational studies consistently report an inverse correlation, establishing causality is complex. Bidirectional Mendelian randomization analyses support the possibility of causal relationships operating from obesity to lower vitamin D status and vice versa.[3,6]

The Indian population presents a critical context for this interaction. The subcontinent bears an exceptionally high burden of vitamin D deficiency, with prevalence estimates ranging from 70% to 100% in various studies.[7] This is attributed to a combination of factors: dietary insufficiency due to low consumption of fortified foods and fatty fish, cultural practices and clothing that limit skin exposure to sunlight despite abundant sunshine, and darker skin pigmentation.[7,8] Simultaneously, India is experiencing a rapid rise in obesity rates due to urbanization, nutritional transition, and sedentary lifestyles. However, robust data exploring the specific link between obesity and vitamin D status in adult South Indian populations remain limited. A stark illustration comes from a study of obese Asian Indian children, which reported universal vitamin D deficiency.[9] This underscores the necessity for focused research in adult populations to quantify the strength of this association and identify contributing factors unique to this demographic.

Study Objectives: This study was therefore designed to examine the association between vitamin D deficiency and obesity among adults attending a tertiary care hospital in Tamil Nadu, South India, and to elucidate the lifestyle and clinical factors that contribute to this relationship.

MATERIALS AND METHODS

Study Design and Setting

A hospital-based, matched case-control study was conducted at the Department of General Medicine, Melmaruvathur Adhiparasakthi Institute of Medical Sciences (MAPIMS) Hospital, located in Tamil Nadu, India. The study took place over a five-month period, from August 2024 to December 2024. The study protocol was reviewed and approved by the Institutional Ethics Committee of MAPIMS, with the approval number 481(08)2024. The research was carried out following the ethical guidelines set forth in the Declaration of Helsinki.

Study Participants and Matching Criteria

Participants were selected from the outpatient section of the General Medicine department. The study focused on adults who were between 18 and 65 years old. Individuals with obesity were identified as cases, using the World Health Organization (WHO) Asia-Pacific body mass index (BMI) standard, which considers a BMI of 25 kg/m² or higher as obese. Controls were people who had a normal BMI, defined as ranging from 18.5 to 22.9 kg/m², based on the same criteria. An important part of the study design involved matching: each control was paired with one case, considering two main factors that could affect the results age (within ±2 years) and gender. This one-to-one matching helped reduce the impact of these factors on the findings.

Inclusion and Exclusion Criteria

Possible participants had to give written consent before joining the study. People who were pregnant or breastfeeding were not allowed to take part. We also did not include people with medical conditions that could greatly affect how their body processes or maintains vitamin D. This included conditions like chronic kidney disease, liver failure, and problems with nutrient absorption such as celiac disease or inflammatory bowel disease. Hyperparathyroidism and certain types of infections like sarcoidosis or tuberculosis were also reasons for exclusion. Additionally, individuals taking medications that can interfere with vitamin D metabolism, such as anticonvulsants like phenobarbital or phenytoin, corticosteroids, antifungal drugs like ketoconazole, and weight loss medications were not enrolled. To prevent any influence from recent vitamin D or calcium use, people who had taken any of these supplements within the three months before the study began were also excluded.

Sample Size Calculation and Justification

Before we started the study, we calculated how many volunteers we would need. Based on previous research, we had a good reason to suspect a link between vitamin D deficiency and obesity. To have a strong chance (80%) of confirming this link, while keeping the risk of a statistical error low (5%), we determined we needed 83 people in each group one group with obesity and one without. In total, 166 participants took part in this study, which is a standard and reliable approach for this type of research.

Trained research staff collected all data using a standardized, multi-component approach to ensure consistency and reliability.

1. Structured Interview and Survey

A semi-structured questionnaire, piloted prior to the study, was administered through in-person interviews. It gathered information across five key domains:

- **Sociodemographics:** Age, sex, occupation, and educational attainment.
- **Lifestyle Factors**: Daily duration of sun exposure (in minutes), along with the type, frequency, and duration of habitual physical exercise.

- **Dietary Patterns:** Regular consumption frequency of natural dietary sources of vitamin D (e.g., fatty fish, eggs) and fortified products (e.g., milk, breakfast cereals). Use of calcium or other nutritional supplements was also recorded.
- **Personal Habits:** Current and past use of tobacco products (smoked or smokeless) and alcohol, as well as routine sunscreen application.
- **Medical History:** Presence of any physician-diagnosed comorbid conditions, including hypertension, diabetes mellitus, dyslipidemia, hypothyroidism, or cardiovascular disease. A review of current medications was concurrently performed.

2. Anthropometric and Clinical Assessments

Standardized techniques and calibrated instruments were used for all physical measurements:

- **Height and Weight:** Measured to the nearest 0.1 cm using a stadiometer and to the nearest 0.1 kg on a calibrated digital scale, respectively, with participants in light clothing and without shoes. Body Mass Index (BMI) was derived using the standard formula: weight (kg) / [height (m)] ^{2}.
- **Blood Pressure:** Assessed on the right arm using a mercury sphygmomanometer after the participant had been seated at rest for a minimum of five minutes. The average of two separate readings was documented for analysis.
- **Obesity Classification:** Following WHO Asia-Pacific guidelines, obesity was defined as a BMI ≥ 25 kg/m^2.

3. Laboratory Analysis

Following a 10–12 hour overnight fast, a 5 mL venous blood sample was collected aseptically from each participant.

- **Sample Processing:** Blood was allowed to clot, then centrifuged to separate serum, which was aliquoted and stored at -80°C until batch analysis.
- **Biochemical Assays:** Serum 25-Hydroxyvitamin D [25(OH)D]: The primary circulating marker of vitamin D status was quantified using a standardized chemiluminescence immunoassay (CLIA).
- **Glycated Hemoglobin (HbA1c):** Measured via high-performance liquid chromatography (HPLC) to evaluate intermediate-term glycemic control.
- **Fasting Lipid Profile:** Concentrations of total cholesterol, Low-Density Lipoprotein (LDL) cholesterol, High-Density Lipoprotein (HDL) cholesterol, and triglycerides were determined using enzymatic colorimetric methods on an automated analyzer.
- **Vitamin D Status:** Participants vitamin D status was classified based on serum 25(OH)D concentration. For the primary study analysis, individuals with levels below 30 ng/mL (<75 nmol/L) were categorized as "Vitamin D Deficient." This group was compared against a combined "Normal" category encompassing all participants with higher levels (insufficient and sufficient).

Statistical Analysis Plan

Data entry and statistical analysis were performed using IBM SPSS Statistics software (version 25.0). Descriptive statistics were computed for all variables. Continuous variables were summarized as mean ± standard deviation (SD), while categorical variables were expressed as frequencies and percentages (%). For bivariate analyses, the Chi-square test (or Fisher's exact test where appropriate) was used to compare categorical variables between cases and controls. The independent samples t-test was used to compare means of continuous variables between the two groups. To compare means across the three vitamin D status categories (deficient, insufficient, sufficient), one-way Analysis of Variance (ANOVA) was employed, followed by post-hoc Tukey tests for pairwise comparisons where the overall ANOVA was significant. The primary analysis to determine the association between obesity and vitamin D deficiency used conditional logistic regression, appropriate for the matched design. The strength of association was expressed as an odds ratio (OR) with a 95% confidence interval (CI). To identify independent predictors of vitamin D deficiency, a multivariable binary logistic regression model was constructed. Variables with a p-value <0.10 in univariable analysis or deemed clinically relevant were entered into the model using a stepwise (forward likelihood ratio) method. The results of the final model are presented as adjusted odds ratios (aOR) with 95% CIs. A two-tailed p-value of less than 0.05 was considered statistically significant for all tests.

RESULTS

Baseline Characteristics of the Study Population

The study enrolled 166 participants, of whom 83 were obese and 83 were age- and sex-matched normal-weight controls. On average, participants were 41.8 years old, with a body mass index (BMI) of 25.47 kg/m^2. The group was predominantly female, making up 55.4% of participants. Participants reported spending an average of about half an hour (32 minutes) outdoors daily. Across the entire group, the average vitamin D (25(OH)D) blood level was 37.18 ng/mL. A concerning finding was the widespread prevalence of poor vitamin D status. Only about a quarter of participants had sufficient levels. The majority fell into the suboptimal range, with 41.0% classified as deficient and another 33.7% as insufficient. Systemic hypertension (38.0%) and diabetes mellitus (32.5%) were the most common health conditions, followed by dyslipidemia (20.5%) and hypothyroidism (10.8%). In terms of diet, consumption of vitamin D-fortified foods was low, reported by only 21.7% of participants, while 29.5% took calcium supplements. Lifestyle habits were also assessed: over half of the participants (56.6%) never used sunscreen, 60.8% were non-smokers, and 57.2%

did not drink alcohol. A notable 41.6% reported not engaging in any regular physical activity.

Primary Association: Vitamin D Status and Obesity

The analysis revealed a strong link between obesity and vitamin D deficiency. Of the 91 participants with vitamin D deficiency, the vast majority (72.5%) were from the obese group, while only 27.5% were controls. Looking at it another way, 77.4% of obese participants were deficient, compared to just 22.6% of normal-weight participants. This difference was highly statistically significant. After accounting for the matched study design, obesity was associated with a nine-fold increase in the odds of having vitamin D deficiency. In simpler terms, vitamin D deficiency was found to be 2.64 times more common in obese individuals than in those with a normal BMI.

Comparison of Lifestyle, Dietary, and Biochemical Parameters

When comparing the obese and control groups, several key differences emerged in modifiable lifestyle factors. Obese participants spent significantly less time outdoors (about 20 minutes per day) compared to controls (about 44 minutes per day). Their weekly fish intake was also lower. As expected from the main finding, average vitamin D levels were much lower in the obese group (24.44 ng/mL) than in the control group (49.92 ng/mL). The obese group also showed a less favorable cardiometabolic profile. They had, on average, higher systolic blood pressure and higher levels of LDL cholesterol, triglycerides, and total cholesterol. Interestingly, their HDL ("good") cholesterol levels were also higher. No significant differences were found in diastolic blood pressure or HbA1c (a marker of long-term blood sugar control) between the two groups.

Analysis Stratified by Vitamin D Status

To see how other factors related to vitamin D levels, we divided participants into three groups based on their vitamin D status: deficient, insufficient, and sufficient. Several measures varied significantly across these groups, including BMI, outdoor time, blood pressure, and all cholesterol markers. Specifically, the vitamin D-deficient group had, on average, a higher BMI, spent less time outdoors, and had higher systolic blood pressure and worse cholesterol levels (higher LDL, triglycerides, and total cholesterol) than the vitamin D-sufficient group. Their fish intake was also lower compared to the other groups.

Multivariable Predictors of Vitamin D Deficiency

We performed an analysis to identify which factors were independently linked to vitamin D deficiency, after accounting for other variables. The results confirmed that obesity was the strongest independent predictor, maintaining its nine-fold increased odds. Less daily time spent outdoors was linked to higher odds of deficiency. Furthermore, a pattern of cardiovascular risk factors was independently associated with deficiency. Higher systolic blood pressure and an adverse lipid profile characterized by elevated LDL, HDL, triglycerides, and total cholesterol all significantly increased the odds of a person being vitamin D deficient. Weekly egg intake did not show a significant association in this final model.

Table 1: Association Between Vitamin D Status and study group (Control vs. Obese)

Serum 25-hydroxy vitamin D	Group		Total	Chi-Square Value	P value	Odds Ratio (OR)
	Control (Normal BMI)	Case (Obese)				
Normal	58	17	75	40.886	<0.001*	9
	77.30%	22.70%	100.00%			
Deficient	25	66	91			
	27.50%	72.50%	100.00%			
Total	83	83	166			
	50.00%	50.00%	100.00%			

*p-value <0.05 – Statistically significant.

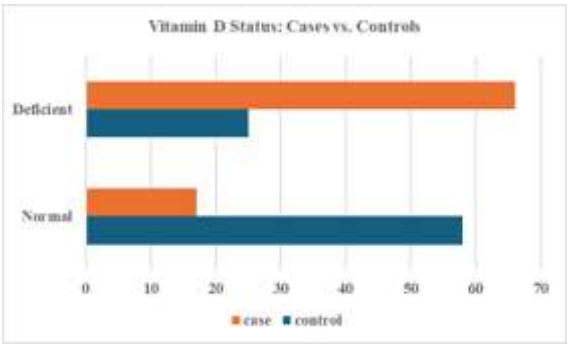

Figure 1: Distribution of Vitamin D status among the study groups

The bar chart clearly illustrates the stark difference in vitamin D status between the two study groups. On one side are the participants with a normal BMI (Controls), and on the other are those classified as obese (Cases). For each group, the chart breaks down their vitamin D health into two key categories: "Normal" (which combines individuals with either sufficient or insufficient levels) and "Deficient" (for those with a clear vitamin D deficiency, defined as serum 25(OH)D levels below 20 ng/mL). This visual comparison instantly highlights where the deficiencies are most

Table 2: Comparison of Lifestyle, Dietary and Biochemical Parameters according to Vitamin D Status

Variable	Group	N	Mean ± SD	F Value	p-value
BMI	Deficient	84	27.95 ± 4.96	23.305	<0.001*
	Insufficient	7	22.33 ± 5.14		
	Sufficient	75	22.98 ± 4.46		
	Total	166	25.47 ± 5.35		
Outdoor Exposure (in minutes)	Deficient	84	25.81 ± 11.60	20.901	<0.001*
	Insufficient	7	40.71 ± 13.30		
	Sufficient	75	38.12 ± 13.43		
	Total	166	32.00 ± 13.96		
Fish Intake (per week)	Deficient	84	0.32 ± 0.60	5.118	0.007*
	Insufficient	7	0.86 ± 1.07		
	Sufficient	75	0.69 ± 0.93		
	Total	166	0.51 ± 0.81		
Serum 25-hydroxyvitamin D	Deficient	84	12.75 ± 4.31	354.656	<0.001*
	Insufficient	7	24.73 ± 2.12		
	Sufficient	75	65.69 ± 18.09		
	Total	166	37.18 ± 28.91		
SBP	Deficient	84	149.19 ± 23.66	33.438	<0.001*
	Insufficient	7	115.71 ± 14.49		
	Sufficient	75	121.20 ± 21.65		
	Total	166	135.13 ± 26.53		
LDL	Deficient	84	1.50 ± 0.32	11.417	<0.001*
	Insufficient	7	1.20 ± 0.34		
	Sufficient	75	1.27 ± 0.32		
	Total	166	1.38 ± 0.34		
HDL	Deficient	84	0.49 ± 0.10	8.530	<0.001*
	Insufficient	7	0.39 ± 0.07		
	Sufficient	75	0.44 ± 0.11		
	Total	166	0.46 ± 0.10		
Triglycerides	Deficient	84	1.73 ± 0.46	8.068	<0.001*
	Insufficient	7	1.46 ± 0.29		
	Sufficient	75	1.47 ± 0.38		
	Total	166	1.61 ± 0.44		
Total Cholesterol	Deficient	84	1.80 ± 0.14	18.603	<0.001*
	Insufficient	7	1.66 ± 0.14		
	Sufficient	75	1.67 ± 0.14		
	Total	166	1.74 ± 0.15		

*p-value <0.05 – Statistically significant

Table 3: Binary Logistic Regression Analysis of Factors Associated with Vitamin D Deficiency

Variable	Coefficient (B)	ODDS Ratio (95% CL)	p-value
Group	-2.19	9.007 (4.43, 18.32)	<0.001*
Egg intake	0.016	1.01 (0.770. 1.34)	0.908
Outdoor Exposure	0.064	0.94 (0.914, 0.963)	<0.001*
SBP	0.042	1.043 (1.028, 1.058)	<0.001*
LDL	1.906	6.73 (2.52, 17.93)	<0.001*
HDL	4.62	101.4 (4.65, 2213.33)	0.003*
Triglycerides	1.342	3.83 (1.77, 8.26)	0.001*
Total Cholesterol	5.76	316.75 (30.13, 333)	<0.001

*p-value <0.05 – Statistically significant

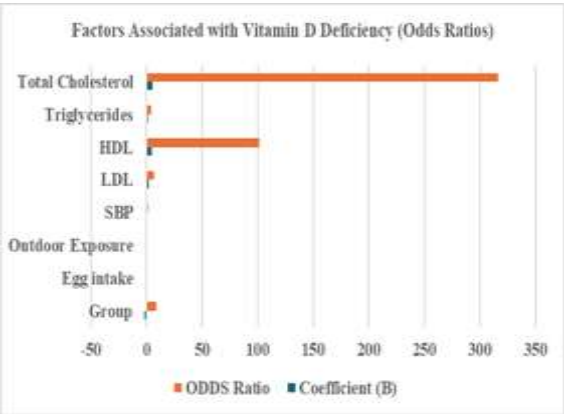

Figure 2: Predictors of Vitamin D Deficiency: Multivariable Logistic Regression Results

Figure 2 presents the crucial predictors of vitamin D insufficiency from our analysis. Using a timber plot, it displays the acclimated odds rate (aOR) and 95 confidence interval for each factor, showing both the strength of the association and its perfection.

DISCUSSION

This matched case-control study provides robust evidence of a strong association between obesity and vitamin D deficiency among adults in Tamil Nadu, South India. The findings are striking: obese individuals had nine-fold higher odds and a 2.64 times greater prevalence of vitamin D deficiency compared to their age- and sex-matched normal-BMI

counterparts. The overall prevalence of deficiency (41%) in our cohort is consistent with the alarmingly high rates reported across India, a country grappling with endemic hypovitaminosis D.[7,10] However, the stark disparity between obese (77.4%) and non-obese (22.6%) individuals appears more pronounced than differences observed in some Western populations,[11] potentially reflecting the synergistic effect of genetic, dietary, and profound lifestyle factors unique to this region.[7,8] The magnitude of the observed odds ratio (OR=9.0) exceeds the pooled estimates (typically ranging from 1.35 to 3.0) from international meta-analyses.[12] This heightened association in our population underscores the potential for the obesity-vitamin D link to be particularly severe in the Indian context. The endemic background deficiency may act as a baseline upon which the metabolic perturbations of obesity exert a powerful additional effect. Our data strongly support the primary pathophysiological mechanism of sequestration, whereby the fat-soluble vitamin D is diluted within and stored by the expanded adipose tissue mass, rendering it less bioavailable in the circulation.[4,13] This is compounded by clear behavioral differences; the significantly lower daily outdoor exposure among obese participants (20.24 vs. 43.76 minutes) directly impairs the body's primary natural source of vitamin D, cutaneous synthesis.[7] Furthermore, our multivariable analysis revealed that vitamin D deficiency was independently associated not only with obesity and low sun exposure but also with a cluster of cardiometabolic risk factors, including higher systolic blood pressure and a more atherogenic lipid profile. This aligns with the conceptual model positioning vitamin D deficiency within the broader constellation of metabolic syndrome.[14,15] The relationship is complex and likely bidirectional: obesity promotes deficiency through sequestration and sedentary behavior, while low vitamin D status may exacerbate metabolic dysfunction by influencing insulin secretion, promoting inflammation, and affecting the renin-angiotensin system.[5,15] Our results thus reinforce the observation that obesity is a critical confounder and effect modifier in the relationship between vitamin D status and cardiovascular disease risk.[16] The clinical and public health implications of these findings are substantial. First, they mandate a proactive approach: routine screening for vitamin D deficiency should be considered a standard component of clinical assessment in obese individuals, given the exceptionally high likelihood of insufficiency. Second, management must be tailored. Standard supplementation doses may be insufficient for obese individuals due to altered pharmacokinetics and a larger volume of distribution; evidence suggests they often require higher, sometimes weight-based, doses to achieve repletion.[17] Third, a powerful and cost-effective strategy lies in integrated lifestyle modification. Counseling that promotes safe, regular outdoor physical activity can simultaneously address the twin problems of obesity and vitamin D deficiency, offering a dual benefit.

Strengths and Limitations: This study has notable strengths, including a matched design that controlled for key confounders (age, sex), comprehensive data collection on lifestyle and clinical factors, use of the gold-standard 25(OH)D assay, and robust multivariable analysis. It provides valuable, region-specific data on an understudied population. Limitations must be acknowledged. The cross-sectional design precludes causal inference regarding the direction of the association. Hospital-based sampling may limit generalizability to the wider community. We did not account for seasonal variation in vitamin D levels, which can be significant. Dietary intake was assessed qualitatively, not quantitatively. We did not evaluate genetic polymorphisms (e.g., in VDR, GC, CYP2R1 genes) that influence vitamin D metabolism. The single-center nature of the study suggests a need for validation in larger, multi-center cohorts.

Future Research Directions: Future studies should adopt longitudinal designs to clarify causality and temporality. Community-based sampling would improve external validity. Intervention trials are urgently needed to establish the optimal dosing, efficacy, and metabolic impact of vitamin D supplementation in obese Indian adults. Research into the role of genetic determinants of vitamin D status in this population is warranted. Investigating seasonal patterns and conducting cost-effectiveness analyses of screening and supplementation programs would provide crucial insights for public health policy and clinical guidelines.

CONCLUSION

This study conclusively demonstrates a strong and significant association between obesity and vitamin D deficiency in a South Indian adult population. Obese individuals were found to have dramatically higher odds and prevalence of vitamin D deficiency compared to those with normal body weight. Given the concurrent epidemics of obesity and vitamin D deficiency in India, these findings carry considerable clinical and public health weight. Vitamin D deficiency should be recognized as a frequent and important comorbidity in obesity. Consequently, integrating routine assessment of vitamin D status into the management of obese patients is warranted. Management strategies should include appropriate, often higher-dose, supplementation coupled with lifestyle counseling focused on increasing safe sun exposure and physical activity. Addressing vitamin D deficiency in this high-risk group offers a pragmatic avenue for improving not only skeletal health but also potentially mitigating associated cardiometabolic risks.

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