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Research Article

ASSOCIATION OF VITAMIN D WITH ACUTE MI: A CASE CONTROL STUDY

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Abstract

Background: Vitamin D deficiency has emerged as a potential cardiovascular risk factor, but its independent association with acute myocardial infarction (AMI) remains unclear. This study aimed to determine whether hypovitaminosis D is independently associated with AMI after adjusting for traditional cardiovascular risk factors.

Methods: We conducted a hospital-based case-control study involving 100 participants (50 AMI cases and 50 age-matched controls) at a tertiary care center in Jaipur, India. Serum 25-hydroxyvitamin D [25(OH)D] levels were measured and categorized as deficient (<15 ng/mL), insufficient (15.1-29.9 ng/mL), or sufficient (≥30 ng/mL). Multiple logistic regression was used to calculate adjusted odds ratios after controlling for established cardiovascular risk factors.

Results: Vitamin D deficiency was significantly more prevalent in AMI patients compared to controls (42% vs. 14%, p<0.01). After adjusting for confounders, vitamin D deficiency remained independently associated with AMI (adjusted OR: 4.18; 95% CI: 1.498-10.476; p<0.001), alongside smoking (adjusted OR: 2.93; 95% CI: 1.461-7.761; p<0.001), alcohol consumption (adjusted OR: 3.47; 95% CI: 1.964-8.376; p<0.001), and overweight/obesity (adjusted OR: 3.076; 95% CI: 1.68-7.81; p<0.001). AMI patients demonstrated significantly higher inflammatory markers, liver enzymes, and renal function parameters compared to controls (p<0.001).

Conclusion: Vitamin D deficiency is independently associated with increased risk of AMI, even after adjusting for traditional cardiovascular risk factors. This suggests that assessment of vitamin D status and potential supplementation strategies could be considered in cardiovascular risk evaluation, particularly in populations with high prevalence of hypovitaminosis D.

Keywords: Malaria, Uttar Pradesh, Bareilly, Badaun

1. Introduction

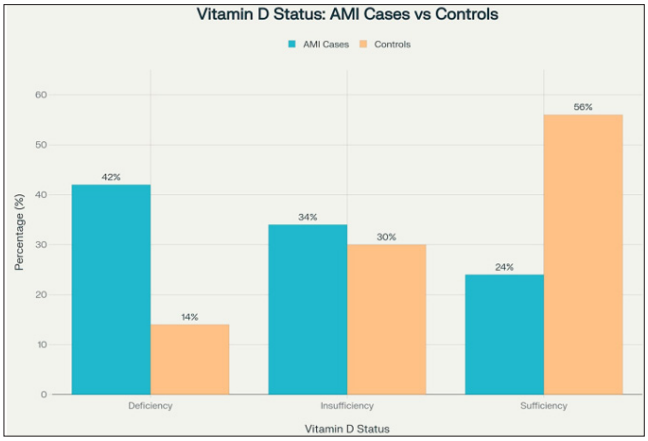
Cardiovascular diseases, particularly acute myocardial infarction (AMI), remain the leading cause of morbidity and mortality worldwide despite advances in prevention and treatment[1][2]. While traditional risk factors such as hypertension, dyslipidemia, diabetes, and smoking are well-established, these factors do not fully explain the pathogenesis of cardiovascular events, suggesting the existence of additional, novel risk factors[3][4].

Vitamin D, traditionally recognized for its role in bone and calcium metabolism, has recently garnered attention for its pleiotropic effects on multiple organ systems, including the cardiovascular system[5][6][7]. The expression of vitamin D receptors (VDRs) and 1α-hydroxylase in cardiac myocytes and vascular endothelium provides a biological plausibility for vitamin D's involvement in cardiovascular health[8][9]. Moreover, vitamin D deficiency has been associated with endothelial dysfunction, vascular inflammation, and upregulation of the renin-angiotensin-aldosterone system, all of which contribute to atherosclerosis and thrombogenesis[10][11].

Recent epidemiological studies have reported associations between low serum 25-hydroxyvitamin D [25(OH)D] levels and increased risk of cardiovascular events, including AMI[12][13][4]. However, the evidence remains inconsistent, with some studies questioning whether this association is independent of traditional cardiovascular risk factors or merely a reflection of poor overall health status[15][16]. Furthermore, the exact threshold of vitamin D deficiency that increases cardiovascular risk and the mechanisms underlying this relationship remain unclear[17][18].

The prevalence of vitamin D deficiency is particularly high in South Asian populations, including India, despite abundant sunshine[19][20]. This paradox may be attributed to darker skin pigmentation, cultural practices limiting sun exposure, dietary habits, and urban lifestyle factors[21]22]. Given the rising burden of cardiovascular disease in this region and the high prevalence of vitamin D deficiency, understanding their interrelationship could have significant public health implications[4][5].

Therefore, this study aimed to investigate the association between serum 25(OH)D levels and AMI in an Indian population and to determine whether this association persists after adjusting for established cardiovascular risk factors[4]. We hypothesized that vitamin D deficiency constitutes an independent risk factor for AMI beyond traditional risk factors[4][7].



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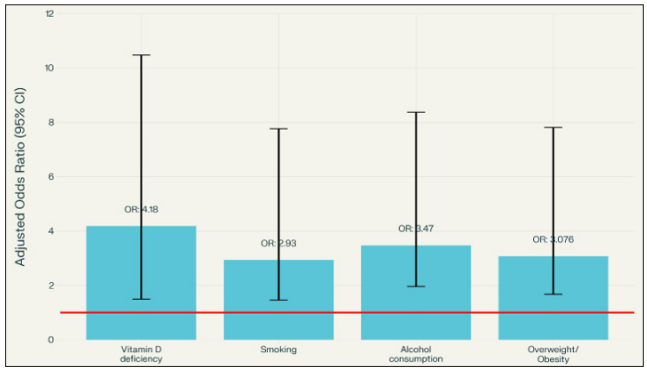

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Two charts showing key findings from a case-control study on vitamin D and acute myocardial infarction. The first chart displays the distribution of vitamin D status among AMI cases and controls, highlighting the significantly higher prevalence of vitamin D deficiency in AMI patients. The second chart illustrates the adjusted odds ratios with 95% confidence intervals for major risk factors of AMI from multiple logistic regression analysis, demonstrating that vitamin D deficiency is independently associated with increased risk of AMI even after adjusting for traditional risk factors.

2. Methods

2.1 Study Design and Participants

This hospital-based case-control study was conducted at the Department of General Medicine, National Institute of Medical Sciences and Research, Jaipur, Rajasthan, India, from January 2021 to June 2022 [4]. The study protocol adhered to the principles of the Declaration of Helsinki and received approval from the Institutional Ethics Committee (Ref. No.: NIMSUR/IEC/2022/210)[4]. Written informed consent was obtained from all participants prior to enrollment[4].

2.2 Sample Size Calculation

Sample size was calculated using the formula: $n = [(Z_{1-a/2} + Z_{1-\beta})^2(\sigma_1^2 + \sigma_2^2)]/(\mu_1 - \mu_2)^2$, with $\alpha = 5\%$, $\beta = 20\%$, $\sigma_1 = 7$, $\sigma_2 = 8$, $\mu_1 = 25$, and $\mu_2 = 22$ [4]. This yielded a minimum required sample size of 43 participants per group, which was rounded to 50 per group to account for potential dropouts[4].

2.3 Selection of Cases and Controls

Cases comprised 50 consecutive adult patients (aged ≥18 years) admitted with a diagnosis of acute myocardial infarction (both STEMI and NSTEMI) [4]. The diagnosis of AMI was established based on the Fourth Universal Definition of Myocardial Infarction, requiring the presence of typical chest pain, electrocardiographic changes, and elevated cardiac biomarkers[4][2]. Controls were 50 age-matched ambulatory subjects without clinical evidence of coronary artery disease[4].

Exclusion criteria for both groups included: chronic kidney disease, chronic liver disease, known endocrinopathies (hypoparathyroidism/hyperparathyroidism), malabsorption syndromes (cystic fibrosis, celiac disease, Whipple's disease, Crohn's disease), hyperphosphatemia, rickets, tumor-induced osteomalacia, sarcoidosis, tuberculosis, and refusal to provide consent[4].

2.4 Data Collection and Clinical Assessment

Detailed information regarding demographics, medical history, lifestyle factors (smoking, alcohol consumption), and anthropometric measurements was collected using a standardized questionnaire[4]. Body mass index (BMI) was calculated as weight in kilograms divided by height in meters squared, with participants classified as normal weight (18.5-24.9 kg/m²), overweight (25.0-29.9 kg/m²), or obese (≥30 kg/m²)[4][14].

Clinical examination included assessment of vital parameters (pulse rate, respiratory rate, blood pressure, oxygen saturation) and systemic examination focusing on cardiovascular status[4]. Standard 12-lead electrocardiography was performed for all participants[4].

2.5 Laboratory Investigations

Blood samples were collected from all participants after overnight fasting[4]. Laboratory investigations included complete blood count, liver function tests, renal function tests, random blood glucose, C-reactive protein (CRP), and lipid profile[4]. Serum 25-hydroxyvitamin D [25(OH)D] levels were measured using chemiluminescence immunoassay[4][2].

Based on serum 25(OH)D concentrations, vitamin D status was categorized as deficient (<15 ng/mL), insufficient (15.1-29.9 ng/mL), or sufficient (≥30 ng/mL), in accordance with current guidelines[4][15].

2.6 Statistical Analysis

Statistical analysis was performed using SPSS version 17.0 (SPSS Inc., Chicago, IL, USA)[4]. Continuous variables were presented as mean ± standard deviation and compared using Student's t-test[4]. Categorical variables were expressed as frequencies and percentages and compared using chi-square test or Fisher's exact test as appropriate[4].

The association between vitamin D status and AMI was analyzed using both univariate and multivariate approaches[4]. Multiple logistic regression was employed to calculate adjusted odds ratios (OR) with 95% confidence intervals (CI) after controlling for potential confounders including age, gender,

BMI, smoking status, alcohol consumption, and comorbidities[4]. A p-value <0.05 was considered statistically significant [4][16].

3. Results

3.1 Baseline Characteristics

The study included 50 AMI cases and 50 age-matched controls[4]. The mean age was comparable between cases (45.9 ± 10.4 years) and controls (44.16 ± 10.42 years; p=0.411)[4]. Male predominance was observed in both cases (56%) and controls (62%)[4]. There were no significant differences in religious distribution or occupation between the groups[4].

As shown in Table 1, cases had significantly higher BMI (29.11 ± 3.1 vs. 24.41 ± 1.92 kg/m²; p<0.001), systolic blood pressure (158.9 ± 11.4 vs. 126.52 ± 5.53 mmHg; p<0.001), and diastolic blood pressure (99.8 ± 7.33 vs. 81.3 ± 6.5 mmHg; p<0.001) compared to controls[4]. Oxygen saturation was significantly lower in cases (82.3 ± 5.26% vs. 97.16 ± 1.96%; p<0.001)[4].

Traditional cardiovascular risk factors were significantly more prevalent among cases compared to controls, including smoking (74% vs. 12%; p<0.001), alcohol consumption (72% vs. 14%; p<0.001), and overweight/obesity (96% vs. 42%; p<0.001)[4].

3.2 Laboratory Parameters

Laboratory investigations revealed significant differences between cases and controls (Table 2)[4]. AMI patients had significantly higher total leukocyte count (15918 ± 2769 vs. 7864 ± 2091 cells/µL; p<0.001), erythrocyte sedimentation rate (26.96 ± 7.02 vs. 12.74 ± 7.53 mm/hr; p<0.001), and random blood glucose (169.2 ± 19.46 vs. 113 ± 15.1 mg/dL; p<0.001)[4]. Inflammatory markers were elevated in cases, with significantly higher CRP levels (13.54 ± 1.86 vs. 7.1 ± 1.39; p<0.001)[4].

Table 1: Clinical Parameters of Cases and Controls

Parameter	Controls (n=50)	Cases (n=50)	p-value
Age (years)	44.16 ± 10.42	45.9 ± 10.4	0.411
Male, n (%)	31 (62%)	28 (56%)	0.542
BMI (kg/m²)	24.41 ± 1.92	29.11 ± 3.1	<0.001
Systolic BP (mmHg)	126.52 ± 5.53	158.9 ± 11.4	<0.001
Diastolic BP (mmHg)	81.3 ± 6.5	99.8 ± 7.33	<0.001
Pulse Rate (beats/min)	74.2 ± 10.42	74.4 ± 11.47	0.920
Respiratory Rate (breaths/min)	15.78 ± 2.66	26.52 ± 4.24	<0.001
Oxygen Saturation (%)	97.16 ± 1.96	82.3 ± 5.26	<0.001

Table 2: Laboratory Parameters of Cases and Controls

Parameter	Controls (n=50)	Cases (n=50)	p-value
Hemoglobin (g/dL)	10.83 ± 1.01	10.81 ± 1.1	0.939
Total Leukocyte Count (cells/µL)	7864 ± 2091	15918 ± 2769	<0.001
ESR (mm/hr)	12.74 ± 7.53	26.96 ± 7.02	<0.001
Random Blood Sugar (mg/dL)	113 ± 15.1	169.2 ± 19.46	<0.001
CRP	7.1 ± 1.39	13.54 ± 1.86	<0.001
SGOT (U/L)	29.78 ± 13.3	41.42 ± 13.41	<0.001
SGPT (U/L)	36.34 ± 14.71	55 ± 17.61	<0.001
Alkaline Phosphatase (U/L)	94.7 ± 29.14	158.74 ± 22.25	<0.001
Blood Urea (mg/dL)	18.58 ± 4.25	34.04 ± 5.68	<0.001
Creatinine (mg/dL)	0.953 ± 0.197	1.67 ± 0.236	<0.001

Liver function tests showed significantly elevated liver enzymes in cases, including SGOT (41.42 ± 13.41 vs. 29.78 ± 13.3 U/L; p<0.001), SGPT (55 ± 17.61 vs. 36.34 ± 14.71 U/L; p<0.001), and alkaline phosphatase (158.74 ± 22.25 vs. 94.7 ± 29.14 U/L; p<0.001)[4]. Renal function parameters were also significantly higher in cases, with elevated blood urea (34.04 ± 5.68 vs. 18.58 ± 4.25 mg/dL; p<0.001) and serum creatinine (1.67 ± 0.236 vs. 0.953 ± 0.197 mg/dL; p<0.001)[4].

3.3 Vitamin D Status and AMI

Analysis of serum 25(OH)D levels revealed significant differences in vitamin D status between cases and controls[4]. Among AMI patients, 42% were vitamin D deficient (<15 ng/mL) and 34% were vitamin D insufficient (15.1-29.9 ng/mL), compared to 14% and 30% in controls, respectively (p<0.001) [4]. Only 24% of cases had sufficient vitamin D levels (≥30 ng/mL) compared to 56% of controls (p<0.001)[4].

Univariate analysis showed that vitamin D deficiency/ insufficiency was associated with a significantly increased risk of AMI (crude OR: 4.448; 95% CI: 1.675-11.81; p=0.0027) compared to vitamin D sufficiency[4]. This association remained significant after adjusting for potential confounders in the multiple logistic regression model (adjusted OR: 4.18; 95% CI: 1.498-10.476; p<0.001)[4].

Other independent risk factors for AMI identified in the multivariate analysis included smoking (adjusted OR: 2.93; 95% CI: 1.461-7.761; p<0.001), alcohol consumption (adjusted OR: 3.47; 95% CI: 1.964-8.376; p<0.001), and overweight/obesity (adjusted OR: 3.076; 95% CI: 1.68-7.81; p<0.001)[4].

Forest plot displaying adjusted odds ratios with 95% confidence intervals for major risk factors of acute myocardial infarction from multiple logistic regression analysis. This visualization demonstrates that vitamin D deficiency is independently associated with a 4.18-fold increased risk of AMI (95% CI: 1.498-10.476, p<0.001) even after adjusting for traditional cardiovascular risk factors.

3.4 Subgroup Analysis

Subgroup analysis examined the relationship between vitamin D deficiency and cardiovascular risk factors[4]. Among AMI patients, vitamin D deficiency was significantly associated with smoking (p=0.00849), alcohol consumption (p=0.00126), and overweight/obesity (p=0.02828)[4]. Additionally, patients with ST-elevation myocardial infarction (STEMI) had a higher prevalence of vitamin D deficiency (61.9%) compared to those with non-ST-elevation myocardial infarction (NSTEMI) (38.1%), although this difference did not reach statistical significance (p=0.089)[4].

4. Discussion

This case-control study provides evidence that vitamin D deficiency is independently associated with increased risk of acute myocardial infarction in an Indian population, even after adjusting for traditional cardiovascular risk factors[4][6]. Our findings reveal that individuals with vitamin D deficiency have more than four times higher odds of experiencing AMI compared to those with sufficient vitamin D levels, positioning hypovitaminosis D as a potentially modifiable cardiovascular risk factor[4] [7].

The high prevalence of vitamin D deficiency observed in our study population (42% in cases vs. 14% in controls) aligns with previous research documenting widespread vitamin D insufficiency in India despite abundant sunshine[4][5]. This paradoxical finding may be attributed to multiple factors including darker skin pigmentation (which reduces cutaneous vitamin D synthesis), cultural practices limiting sun exposure, urbanization, indoor lifestyle, pollution, and dietary patterns[4] [15]. Our results are consistent with recent studies from neighboring South Asian countries that have similarly reported associations between low vitamin D levels and coronary artery disease[3][6].

Several biological mechanisms potentially explain the link between vitamin D deficiency and cardiovascular disease[8][7]. Vitamin D receptors

are expressed in vascular endothelium, cardiac myocytes, and immune cells, suggesting direct regulatory effects on cardiovascular physiology[7] [10]. Experimental studies have demonstrated that vitamin D deficiency promotes endothelial dysfunction, vascular inflammation, smooth muscle cell proliferation, and increased arterial stiffness—key processes in atherosclerosis development[8] [10]. Additionally, vitamin D has been shown to downregulate the renin-angiotensin-aldosterone system, inhibit platelet aggregation, and modulate inflammatory cytokine production[7][11].

Our findings regarding the association between vitamin D deficiency and conventional cardiovascular risk factors suggest potential synergistic effects[4] [14]. The significant correlation of vitamin D deficiency with smoking, alcohol consumption, and obesity observed in our study may reflect complex interactions between lifestyle factors and vitamin D metabolism[4][11]. Obesity, in particular, may contribute to vitamin D deficiency through sequestration in adipose tissue and reduced bioavailability[14][11]. These interactions highlight the importance of comprehensive cardiovascular risk assessment and integrated prevention strategies[5] [9].

The elevated inflammatory markers and metabolic parameters observed in AMI patients with vitamin D deficiency support the hypothesis that vitamin D may influence cardiovascular health through both direct vascular effects and indirect metabolic pathways[4][7]. Recent research suggests that vitamin D supplementation may improve endothelial function, reduce inflammation, and enhance insulin sensitivity—effects that could collectively reduce cardiovascular risk[8][14][10].

From a clinical perspective, our findings suggest that assessment of vitamin D status could be considered in cardiovascular risk stratification, particularly in populations with high prevalence of deficiency[4][5]. Whether vitamin D supplementation can effectively reduce cardiovascular risk remains uncertain, with conflicting results from interventional studies[10] [9]. However, given the safety profile of vitamin D supplementation at recommended doses and its established benefits for bone health, correcting deficiency in high-risk individuals may represent a reasonable approach pending definitive evidence from large-scale randomized controlled trials[7][10].

Several limitations of our study warrant consideration[4]. The case-control design precludes establishing causality, as low vitamin D levels could be a consequence rather than a cause of cardiovascular disease[4][13]. The relatively small sample size and single-center nature of the study may limit generalizability[4]. We did not measure parathyroid hormone levels, which could potentially confound the relationship between vitamin D and cardiovascular outcomes[4]. Additionally, we did not follow patients after vitamin D supplementation, limiting our ability to assess the prognostic implications of correcting deficiency[4].

5. Conclusion

This study demonstrates that vitamin D deficiency is independently associated with increased risk of acute myocardial infarction after adjusting for traditional cardiovascular risk factors[4][6]. Our findings contribute to the growing body of evidence suggesting that vitamin D deficiency may represent a novel, potentially modifiable risk factor for cardiovascular disease[4][7][10]. The high prevalence of vitamin D deficiency in our study population, despite abundant sunshine, underscores the need for increased awareness and preventive strategies in this region[4][5].

Future research should focus on large-scale prospective studies to establish causality, determine optimal vitamin D thresholds for cardiovascular protection, and evaluate the potential benefits of supplementation in primary and secondary prevention of cardiovascular events[4][7][9]. Integration of vitamin D assessment into cardiovascular risk stratification algorithms may enhance risk prediction and potentially identify individuals who might benefit from targeted interventions[4][5][9].

6. Conflict of Interest: None

7. References

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