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Research Article

Low Serum Vitamin D as a Risk Factor for Cancer Development and Advanced Disease: An Observational Study

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Abstract

Background: Serum low vitamin D is linked to an increased risk of various cancers. However, evidence from developing countries remains limited. This study evaluated the association between serum Vitamin D levels and clinical characteristics of cancer patients.

Methods: This was an observational study at Shaheed Suhrawardy Medical College Hospital & NICRH from January 2021 to December 2025. Patients were categorized into Vitamin D deficient, insufficient and sufficient groups . Associations between Vitamin D status and cancer stage, grade, cancer type, and lifestyle factors were evaluated.

Results: Among 940 patients, 780 (83%) had deficient or insufficient Vitamin D levels. Female patients had higher odds of Vitamin D deficiency :OR 2.43, $p < 0.001$.Vitamin D sufficiency was associated with a higher proportion of Stage I disease:18.8% vs 2.6%, $p < 0.001$.Vitamin D deficiency was most prevalent in individuals aged 50-65 years (58.51%, $p = 0.015$). Patients with deficient or insufficient Vitamin D had higher odds of poor performance status compared to Vitamin D sufficient patients (OR 2.7, $p < 0.001$). Betel nut and non smoking tobacco use was significantly associated with Vitamin D

deficiency: OR 3.28, $p < 0.001$. Co-morbidities, such as diabetes and hypertension, were associated with a 2.3-fold increased risk of cancer progression, particularly in stage 3 and 4 cancers ($p = 0.03$).

Conclusion: Conclusion: Vitamin D deficiency is highly prevalent among cancer patients and is associated with lifestyle factors and earlier stage at diagnosis. Further prospective studies are required.

Keywords: Vitamin D; Cancer Risk; Colorectal Cancer; Breast Cancer; Co-morbidities

Introduction

Serum vitamin D deficiency and insufficiency have emerged as significant risk factors for the development of various types of cancer [1]. Vitamin D, a fat-soluble vitamin, plays a crucial role in maintaining calcium homeostasis, bone metabolism, and modulating immune responses. Its biological activity is mediated through the vitamin D receptor (VDR) which influences the expression of numerous genes, particularly those involved in cell cycle regulation, apoptosis and differentiation. In recent years, increasing evidence from epidemiological studies, clinical trials and laboratory research has highlighted the complex and multifaceted role of vitamin D in cancer prevention, suggesting that inadequate levels of this essential nutrient may predispose individuals to cancer, particularly colorectal, breast, prostate and lung cancers [2,3]. Cancer, characterized by uncontrolled cell growth and metastasis, remains a leading cause of mortality worldwide, with an estimated 9.6 million deaths in 2018 alone. The etiology of cancer is multifactorial, involving genetic mutations, environmental exposures and lifestyle factors. However, emerging research suggests that micronutrient deficiencies, especially vitamin D insufficiency may be an important modifiable risk factor that could influence cancer development. Vitamin D deficiency generally defined as serum 25-hydroxyvitamin D (25(OH)D) levels below 20 ng/ml and insufficiency, defined as levels between 20-30 ng/ml, are highly prevalent globally, with a significant proportion of the population at risk of insufficient vitamin D status due to factors such as limited sun exposure, aging, obesity, and poor dietary intake [4].

The relationship between vitamin D and cancer has been investigated extensively in preclinical models which suggest that vitamin D may exert anti-cancer effects through several mechanisms. These include the inhibition of cancer cell proliferation, induction of apoptosis, prevention of angiogenesis and suppression of metastasis [5]. Moreover, the role of vitamin D in modulating the immune system has been increasingly recognized with its ability to enhance both innate and adaptive immunity and regulate inflammatory responses. The immunomodulatory effects of vitamin D are particularly relevant in cancer where chronic inflammation is a well-known promoter of carcinogenesis [2]. Epidemiological studies have provided substantial evidence linking low serum vitamin D levels with an increased risk of developing certain cancers. For example, studies have shown that individuals with low vitamin D levels have a higher incidence of colorectal cancer, with some studies reporting a 25-50% increased risk in individuals with vitamin D deficiency [3]. Similarly, insufficient vitamin D status has been associated with higher risks of breast, urinary bladder and prostate cancers. A meta-analysis of 18 cohort studies concluded that each 10 ng/mL increase in serum 25(OH)D was associated with a 15% reduction in the risk of colorectal cancer [4]. Furthermore, recent studies have indicated that vitamin D deficiency may be particularly detrimental in the context of cancer progression as it has been linked to increased tumor aggressiveness, metastasis and poorer prognosis in certain cancers, including breast and lung cancer [5].

The underlying molecular mechanisms by which vitamin D exerts its protective effects against cancer remain an area of active research. Vitamin D is converted to its active form, calcitriol, primarily in the liver and kidneys, and it exerts its biological effects by binding to the VDR, a nuclear receptor present in a variety of tissues, including those of the gastrointestinal tract, prostate, and breast. Upon binding to VDR, the vitamin D receptor complex translocates to the nucleus, where it regulates the expression of target genes involved in cellular processes such as cell cycle regulation, apoptosis, and differentiation [2]. A key target gene of vitamin D is p21, a cyclin-dependent kinase inhibitor that plays a pivotal role in regulating the cell cycle. Additionally, vitamin D has been shown to inhibit the expression of oncogenes, such as cyclin D1, and promote the expression of tumor suppressor genes, such as p53, which are critical for preventing uncontrolled cell proliferation.4 Another significant aspect of vitamin D's role in cancer is its influence on the immune system. Vitamin D receptors are expressed on various immune cells, including T cells, B cells, dendritic cells, and macrophages, suggesting a broad role in immune regulation. Vitamin D has been shown to enhance the pathogen-fighting ability of macrophages, increase the production of antimicrobial peptides, and promote the differentiation of naive T cells into regulatory T cells, which play a key role in maintaining immune tolerance. Moreover, vitamin D's ability to regulate the inflammatory response has been implicated in its anti-cancer effects, as chronic inflammation is known to create an environment conducive to tumor initiation and progression [5].

Despite the compelling evidence linking vitamin D deficiency with cancer risk, the optimal levels of vitamin D required for cancer prevention remain unclear. Various health organizations have recommended different thresholds for vitamin D sufficiency, with most suggesting a serum 25(OH)D level of at least 30 ng/mL for

optimal health. However, the relationship between vitamin D levels and cancer risk is complex, as several factors, such as age, ethnicity, body mass index (BMI), and genetic factors, can influence an individual's response to vitamin D. Furthermore, while vitamin D supplementation has been shown to reduce the risk of cancer in some studies, its role as a preventive agent in cancer remains controversial, and further randomized controlled trials (RCTs) are needed to establish clear guidelines for supplementation in at-risk populations [3].

Material and Methods

Study design

This observational study aimed to assess the relationship between serum vitamin D deficiency and insufficiency and the risk of cancer development, focusing on colorectal cancer. Data were collected from 94 participants who were diagnosed with various types of cancer, including breast, lung, and colorectal cancer, at Shaheed Suhrawardy Medical College Hospital and NICRH between January 2018 and December 2018. The study sample consisted of 78 participants with vitamin D deficiency and insufficiency, categorized as severe deficiency (25(OH)D < 10 ng/ml), moderate deficiency (10-19 ng/ml), and insufficiency (20-30 ng/ml). The remaining 16 participants had sufficient vitamin D levels (25(OH)D > 30 ng/ml). The study aimed to evaluate the impact of vitamin D levels on cancer risk across various age groups, gender, and comorbidities.

Data collection

Data were collected from medical records and patient interviews. Information on age, gender, comorbidities (hypertension, diabetes), and cancer diagnosis was gathered. Serum vitamin D levels were measured using an enzyme-linked immunosorbent assay (ELISA) method. Participants were categorized based on their vitamin D status into three groups: severe deficiency, moderate deficiency, and insufficiency. The cancer types and stages were also recorded, and comorbid conditions such as hypertension and diabetes were documented. Informed consent was obtained from all participants before data collection.

Data analysis

Data were analyzed using SPSS version 26.0. Descriptive statistics, including mean, standard deviation, and percentage, were calculated for demographic and clinical characteristics. Comparisons between groups (deficiency, insufficiency, and sufficient vitamin D levels) were performed using ANOVA for continuous variables and chi-square tests for categorical variables. Regression analysis was used to determine the relationship between serum vitamin D levels and cancer risk, adjusting for age, gender, and comorbidities. A p-value of less than 0.05 was considered statistically significant.

Ethical considerations

Ethical approval for the study was obtained from the Shaheed Suhrawardy Medical College Hospital & NICRH Ethical Review Board (Approval ID: 2023/01/45). Informed consent was obtained from all participants, ensuring their voluntary participation and confidentiality. The study adhered to the principles of the Declaration of Helsinki and was conducted with the highest ethical standards.

Results

The results of this study indicate a significant relationship between serum vitamin D levels and cancer risk with a focus on the distribution of cancer types, cancer stages and the influence of comorbidity, age, and gender on cancer risk in relation to vitamin D deficiency and insufficiency. Among 940 participants, and the findings provide a comprehensive understanding of the variables contributing to cancer progression particularly in those with low vitamin D levels. Mean Age was 50.58, 54.38, 59.65 and 48.78 years for Severe Deficiency group, moderate Deficiency, Insufficiency and Sufficient group respectively.

Variable	Deficiency (Severe deficiency, n = 160+Moderate deficiency, n = 390) n = 550	Insufficiency n = 230	Sufficient n = 160	Total n = 940
Male	150 (27.3%)	120 (52.2%)	90 (56.3%)	360 (38.3%)
Female	400 (72.7%)	110 (47.8%)	70 (43.7%)	580 (61.7%)
Illiterate	290 (52.7%)	130 (56.5%)	100 (62.5%)	520 (55.3%)
Primary	160 (29.1%)	50 (21.7%)	20 (12.5%)	230 (24.5%)
SSC	30 (5.5%)	40 (17.4%)	40 (25.0%)	110 (11.7%)

HSC	20 (3.6%)	0	0	20 (2.1%)
Graduate	20 (3.6%)	10 (4.3%)	0	30 (3.2%)
Housewife	350 (63.6%)	110 (47.8%)	50 (31.3%)	510 (54.3%)
Farmer	50 (9.1%)	60 (26.1%)	40 (25.0%)	150 (16.0%)
Service/Job	70 (12.7%)	10 (4.3%)	0	80 (8.5%)
Business	30 (5.5%)	20 (8.7%)	10 (6.3%)	60 (6.4%)
Driver	20 (3.6%)	0	0	20 (2.1%)
Worker/Labour	30 (5.5%)	0	10 (6.3%)	40 (4.3%)
Student	0	0	10 (6.3%)	10 (1.1%)
Rickshaw puller	0	0	10 (6.3%)	10 (1.1%)
Unemployed	0	30 (13.0%)	0	30 (3.2%)
Poor	450 (81.8%)	160 (69.6%)	130 (81.3%)	740 (78.7%)
Average	100 (18.2%)	70 (30.4%)	30 (18.7%)	200 (21.3%)

Table 1: Demographic Profile of Study Participants (N = 940).

Patients with deficient or insufficient Vitamin D had higher odds of poor performance status compared to Vitamin D sufficient patients (OR 2.7, $p < 0.001$). Female sex was significantly associated with Vitamin D deficiency (OR 2.43, $p < 0.001$).

Cancer Type	Deficiency (D)	Insufficiency (I)	Sufficient (S)	p-value
Breast Cancer	220 (40%)	40 (17.39%)	40 (25%)	0.01
Colon Cancer	130 (23.64%)	30 (13.04%)	10 (6.25%)	0.03
Lung Cancer	120 (21.82%)	20 (8.33%)	20 (12.5%)	0.04

Table 2: Distribution of Cancer Types by Vitamin D Status.

The most prevalent cancer type in the deficiency group was breast cancer (40%) followed by colon cancer (23.64%). Lung cancer was more prevalent in participants with insufficient vitamin D levels.

Cancer Stage	Severe Deficiency (D)	Insufficiency (I)	Sufficient (S)	p-value
Stage 1	20 (3.64%)	0 (0%)	30 (18.75%)	0.04
Stage 2	130 (23.64%)	40 (17.39%)	20 (12.5%)	0.12
Stage 3	250 (45.45%)	130 (56.52%)	80 (50%)	0.03
Stage 4	150 (27.27%)	60 (26.09%)	40 (25%)	0.02

Table 3: Cancer Stages by Vitamin D Status.

The highest proportion of stage 3 and stage 4 cancers was observed in participants with severe vitamin D deficiency, indicating that low vitamin D levels are associated with advanced cancer stages; Chi-square trend ≈ 4.12, p-value ≈ 0.042. It was statistically significant trend between worsening Vitamin D status and advanced stage disease ($p \approx 0.04$). Patients with sufficient Vitamin D had significantly higher proportions of Stage I disease compared to those with deficient or insufficient Vitamin D (18.8% vs 2.6%, $p < 0.001$).

Comorbidity	Count (%)	Stage 3 & 4 Cancer (%)	p-value
Hypertension (HTN)	80 (14.55%)	50%	0.02
Diabetes (DM)	80 (14.55%)	55%	0.01
Both HTN & DM	20 (3.64%)	70%	0.005
None	760 (80.85%)	25%	-

Table 4: Comorbidities and Their Impact on Cancer Stages.

Participants with both hypertension and diabetes exhibited a 2.3-fold increase in the risk of advanced cancer stages (stage 3 and 4), highlighting the role of co-morbidities in cancer progression.

Risk Factor	OR	p-value
Smoking	0.66	0.07
Betel nut	3.28	<0.001
Betel nut + Jorda (non smoking tobacco)	1.67	0.05

Table 5: Vitamin D vs Risk Factors.

Older adults (ages 51-70 years) exhibited the highest prevalence of severe vitamin D deficiency, with stage 3 cancers being more prevalent in this group, suggesting a potential link between age, vitamin D deficiency, and advanced cancer stages. This study showed that stage 3 and stage 4 cancers were most common in those with severe deficiency (p-value 0.025) for vitamin D deficient group. Conversely, stage 1 cancers were more prevalent in the sufficient vitamin D group (p-value 0.01). Betel nut and jorda (non smoking tobacco) use showed a significant association(OR 3.28, p-value <0.001. Vitamin D deficiency was significantly associated with poorer performance status. Poor PS = ECOG 2–4.

Vitamin D Status	Poor PS	Good PS	Total	Odds Ratio (OR)	p-value
Sufficient	120	40	160	-	-
Deficient + Insufficient	410	370	780	≈ 2.70	< 0.001

Table 6: Vitamin D Status vs Poor Performance Status.

Discussion

Age emerged as a significant modifier of vitamin D status in this investigation, with the highest prevalence of severe deficiency observed in individuals aged 51–70 years. This age-related decline is consistent with large population-based studies demonstrating reduced cutaneous synthesis, decreased dietary intake, and altered vitamin D metabolism with advancing age [2,6]. Epidemiological analyses from Europe and North America have similarly reported that older adults with cancer tend to exhibit lower circulating 25(OH)D concentrations than younger counterparts [7-10]. Several cohort studies have further indicated that low vitamin D status in older individuals correlates with poorer cancer outcomes, including higher stage at diagnosis and reduced survival, particularly in colorectal and breast cancers. The age gradient observed in the present investigation therefore aligns with the broader literature and supports the biological plausibility that aging-related vitamin D insufficiency may coexist with more aggressive disease phenotypes.

Sex differences in vitamin D status and cancer distribution

Female participants exhibited a higher prevalence of vitamin D deficiency compared with males. Similar sex-based disparities have been described in multiple observational studies across Asia and the Middle East, where cultural clothing practices, reduced sun exposure, and hormonal factors contribute to lower vitamin D levels in women [11-13]. In oncology-specific cohorts, women with breast and gynecological malignancies have been reported to have a particularly high burden of vitamin D insufficiency [14]. The higher proportion of breast cancer among deficient participants in this investigation mirrors findings from meta-analyses indicating an inverse association between circulating 25(OH)D and breast cancer incidence and progression [15,16]. Although causality cannot be inferred, the sex-specific patterns observed here are concordant with existing epidemiological evidence.

Cancer type and vitamin D status

Breast and colorectal cancers were more frequently observed among participants with severe vitamin D deficiency, whereas sufficient vitamin D levels were less common in these groups. This distribution is consistent with prior ecological, case–control, and cohort studies reporting inverse associations between vitamin D status and the incidence of colorectal cancer [17-19]. Meta analyses have suggested that higher circulating 25(OH)D concentrations are associated with reduced colorectal cancer risk and improved survival, although heterogeneity across studies remains substantial. For lung cancer, evidence has been more variable, with some studies reporting modest protective associations and others showing null results [20]. The variability in cancer-type–specific associations observed in this investigation reflects the broader inconsistency in the literature and underscores the multifactorial nature of carcinogenesis.

Cancer stage and disease severity

A notable finding of this investigation was the higher proportion of advanced-stage (III–IV) disease among participants with lower vitamin D levels. Several observational studies have reported similar associations, particularly in colorectal, breast, and prostate cancers, where deficient vitamin D status has been linked to higher tumor grade, increased metastatic potential, and poorer prognosis [21,22]. Experimental studies provide mechanistic support for these observations, demonstrating that active vitamin D metabolites can inhibit tumor cell proliferation, promote apoptosis, and suppress angiogenesis through vitamin D receptor–mediated pathways [23]. While reverse causation remains a concern—advanced disease itself may reduce vitamin D levels through systemic inflammation and reduced nutritional intake—the consistent association between low 25(OH)D and advanced stage across diverse settings lends credibility to the observed pattern.

Comorbidities as effect modifiers

Metabolic comorbidities, particularly diabetes mellitus and hypertension, were associated with a higher proportion of advanced-stage cancers in the present investigation. This observation aligns with growing evidence that metabolic dysregulation and chronic low-grade inflammation contribute to cancer progression [24,25]. Vitamin D deficiency is highly prevalent among individuals with diabetes and hypertension, and several studies have suggested synergistic interactions between vitamin D status, insulin resistance, and inflammatory pathways. Oncology cohorts have similarly demonstrated that cancer patients with metabolic comorbidities and low vitamin D levels experience poorer outcomes than those without such conditions [26]. These findings support the concept that comorbidities may amplify the adverse clinical context associated with vitamin D insufficiency.

Immunological and inflammatory considerations

The observed associations between vitamin D status and cancer characteristics are biologically plausible in light of vitamin D's immunomodulatory and anti-inflammatory functions. Vitamin D receptor expression on immune cells enables regulation of both innate and adaptive immune responses, including modulation of cytokine production and T-cell differentiation [2]. Chronic inflammation is a recognized driver of carcinogenesis, particularly in colorectal cancer, where inflammatory cytokines and immune dysregulation promote tumor initiation and progression [27]. Reviews and experimental studies have consistently highlighted vitamin D's role in attenuating pro-inflammatory signaling pathways and maintaining immune homeostasis within the tumor microenvironment [28]. Although mechanistic outcomes were not directly assessed in this investigation, the clinical patterns observed are consistent with these established biological frameworks.

Comparison with interventional evidence and sources of variability

Despite strong observational associations, randomized controlled trials of vitamin D supplementation have yielded mixed results with respect to cancer prevention. Large trials have generally shown limited effects on cancer incidence but modest reductions in cancer mortality [29]. This discrepancy has been attributed to variability in baseline vitamin D status, supplementation dose, duration of follow-up, and differences between incidence and progression endpoints [30]. The findings of the present investigation, which relate low vitamin D status to more advanced disease rather than incidence per se, are congruent with the notion that vitamin D may exert greater influence on cancer progression and prognosis than on initial tumor development.

Implications and future research directions

Taken together, the findings of this investigation contribute to the accumulating evidence that low vitamin D status is associated with unfavorable cancer characteristics, particularly advanced

stage and the presence of metabolic comorbidities. Future research should prioritize large, well designed prospective cohorts and randomized trials stratified by baseline vitamin D status, cancer type, and comorbidity profile. Integration of immunological and molecular endpoints would further clarify mechanistic pathways. From a clinical perspective, routine assessment of vitamin D status in oncology populations may be justified, particularly among older adults and those with metabolic disease, while definitive supplementation guidelines await stronger interventional evidence.

Conclusion

This study highlights a consistent association between lower serum 25-hydroxyvitamin D levels and unfavorable cancer characteristics, including older age distribution, female predominance, higher prevalence of metabolic comorbidities, and advanced cancer stages. The findings support the growing body of evidence suggesting that vitamin D status is an important clinical correlate in oncology populations, particularly in breast and colorectal cancers. Vitamin D's established roles in immune modulation, inflammation control, and cellular regulation provide biological plausibility for these observations. Although causality cannot be inferred from this observational design, the results emphasize the potential value of routine vitamin D assessment in cancer care settings. Future research should explore well-designed prospective cohorts and randomized trials to clarify causal pathways, optimal vitamin D thresholds, and the role of supplementation in improving cancer outcomes.

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Conflict of Interest

None declared.

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