

PREVALENCE OF VITAMIN D DEFICIENCY AND ITS ASSOCIATION WITH DISEASE CHARACTERISTICS IN PATIENTS WITH CHRONIC OBSTRUCTIVE PULMONARY DISEASE AT GOVERNMENT MEDICAL COLLEGE AND HOSPITAL

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Received : 01/09/2026
Received in revised form : 27/09/2026
Accepted : 10/10/2026

Keywords:

Chronic Obstructive Pulmonary Disease; Disease Severity; Pulmonary Function Tests; Vitamin D Deficiency; Vitamin D.

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DOI: 10.47009/jamp.2026.8.5.127

Source of Support: Nil,
Conflict of Interest: None declared

Int J Acad Med Pharm
2026; 8 (5); 801-806



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ABSTRACT

Background: Vitamin D deficiency is common in patients with chronic obstructive pulmonary disease (COPD) and may be associated with disease severity. This study evaluated the prevalence of vitamin D deficiency and its association with COPD severity according to GOLD spirometric stage and clinical classification. **Aims and Objectives:** To determine the prevalence of vitamin D deficiency among patients with COPD and assess its association with GOLD spirometric stage and GOLD clinical classification. **Materials and Methods:** This cross-sectional study included 80 patients with COPD aged ≥ 40 years attending Government Medical College and Hospital, Cuddalore District, from August 2023 to January 2025. COPD was confirmed by post-bronchodilator $FEV_1/FVC < 0.70$. Serum 25-hydroxyvitamin D was measured by chemiluminescent immunoassay and classified as deficient (< 20 ng/mL), insufficient ($20-29$ ng/mL), or sufficient (≥ 30 ng/mL). Associations were assessed using chi-square, Pearson's correlation, and Spearman's rank correlation. **Results:** The mean age was 60.11 ± 11.43 years, with 53 (66.3%) males. Mean serum vitamin D was 18.34 ± 10.16 ng/mL; 52 (65.0%) were deficient, 20 (25.0%) insufficient, and 8 (10.0%) sufficient. GOLD stage 2 was predominant (48, 60.0%), followed by stage 3 (24, 30.0%). Vitamin D status was significantly associated with GOLD stage ($\chi^2=60.577$, Spearman's $\rho=-0.577$; $p<0.001$) and GOLD clinical classification ($\chi^2=64.370$, $\rho=-0.628$; $p<0.001$). **Conclusion:** Vitamin D deficiency was highly prevalent among patients with COPD and was significantly associated with higher GOLD spirometric stage and GOLD clinical classification. Vitamin D deficiency may therefore serve as an associated marker of COPD disease severity.

INTRODUCTION

Chronic obstructive pulmonary disease (COPD) remains one of the leading causes of illness and death worldwide. According to the Global Burden of Disease Study 2021, COPD affected approximately 213 million people and caused nearly 3.72 million deaths globally, making it the fourth leading cause of death.^[1] In India, a recent meta-analysis including more than 27,000 adults estimated the overall prevalence of spirometry-confirmed COPD at 13%, rising to 15% in studies that used GOLD diagnostic criteria and to 27% in hospital-based populations.^[2] The Global Initiative for Chronic Obstructive Lung

Disease (GOLD) provides a widely accepted framework for assessing COPD using spirometric severity and clinical classification based on symptom burden and exacerbation history, which helps guide diagnosis, prognosis, and treatment.^[3]

Vitamin D is best known for its role in calcium homeostasis and bone health, but research over the past two decades has demonstrated its additional roles in immune regulation, inflammation, and respiratory muscle function.^[4] Despite abundant sunlight in many parts of the world, vitamin D deficiency remains a widespread public health problem. A large pooled analysis of nearly 7.9 million people from 81 countries found that 47.9%

had serum 25-hydroxyvitamin D levels below 50 nmol/L, while 15.7% had levels below 30 nmol/L.⁵ This problem is particularly pronounced in South Asia, where a systematic review reported a pooled vitamin D deficiency prevalence of 68% among adults, with India showing a prevalence of approximately 67%.⁶ Earlier Indian studies have also reported deficiency rates of 70–100% in the general population despite year-round sunlight exposure.^[7] Patients with COPD may be at increased risk of vitamin D deficiency because of reduced outdoor physical activity, advancing age, smoking, and corticosteroid exposure. A study by Janssens et al. reported vitamin D deficiency in 31% of smokers with normal lung function, compared with 60% of patients with GOLD stage 3 COPD and 77% of those with GOLD stage 4 disease, suggesting an association between vitamin D status and COPD severity.^[8] Vitamin D receptors are expressed on airway epithelial and immune cells, where vitamin D contributes to regulation of innate and adaptive immunity, modulation of inflammatory responses, and antimicrobial defence in the lungs.^[9,10] Given the high burden of both COPD and vitamin D deficiency in India, and the limited local data examining the association between vitamin D status and COPD severity, particularly according to GOLD spirometric stage and clinical classification, the present study was undertaken to determine the prevalence of vitamin D deficiency among patients with COPD and to assess its association with disease severity.

Aim & Objectives

Aim

1. To investigate the prevalence of vitamin D deficiency in patients with COPD, and its correlation with disease severity as assessed by GOLD spirometric staging and GOLD clinical classification.

Objectives

1. To determine the prevalence of vitamin D deficiency among patients with COPD
2. To assess the association between vitamin D status and severity of COPD as classified by GOLD spirometric stage
3. To assess the association between vitamin D status and GOLD clinical classification (Groups A–D)

MATERIALS AND METHODS

This cross-sectional study was conducted over a period of 18 months, from August 2023 to January 2025, among patients with COPD attending the outpatient and inpatient services of Government Medical College and Hospital, Cuddalore District.

Study Population

A total of 80 patients with a confirmed diagnosis of COPD were included in the study. Participants were recruited from the outpatient and inpatient services of

the tertiary care hospital after fulfilling the predefined inclusion and exclusion criteria.

Inclusion & Exclusion Criteria

Patients aged ≥ 40 years with a confirmed diagnosis of COPD based on post-bronchodilator spirometry showing $FEV_1/FVC < 0.70$ were included in the study. Only patients who provided written informed consent were enrolled.

Patients with chronic comorbidities that could alter calcium or vitamin D metabolism, including chronic kidney disease and parathyroid disorders, Patients who had received vitamin D supplementation during the preceding six months, Patients who were acutely ill with respiratory infection or had other acute comorbidities that could interfere with the study results were excluded.

Sample Size Calculation

The sample size was determined based on the prevalence of vitamin D deficiency reported in previous studies among patients with COPD. The required sample size was calculated using a standard statistical formula for estimating a proportion, considering an appropriate confidence level, expected prevalence, and allowable error. Based on the calculation, a total sample size of 80 COPD patients was selected for the study.

Methods

All eligible participants were enrolled after applying the inclusion and exclusion criteria. Data were collected using a structured interview, clinical examination, and laboratory investigations.

Demographic and lifestyle information including age, sex, smoking history, pack-years, and body mass index (BMI). Clinical assessment included COPD duration, number of exacerbations and hospitalizations during the preceding year, symptom burden, and functional status. COPD severity was assessed using spirometric parameters and GOLD classification. Spirometry was performed to obtain forced expiratory volume in one second (FEV_1), forced vital capacity (FVC), and FEV_1/FVC ratio. GOLD spirometric staging and clinical classification were recorded for each participant.

Serum vitamin D was assessed by measuring 25-hydroxyvitamin D [$25(OH)D$] using a chemiluminescent immunoassay. Vitamin D status was classified as deficient (< 20 ng/mL), insufficient ($20–29$ ng/mL), or sufficient (≥ 30 ng/mL).

Additional clinical assessments included modified Medical Research Council (mMRC) dyspnoea score, COPD Assessment Test (CAT) score, St. George's Respiratory Questionnaire (SGRQ), six-minute walk distance, resting oxygen saturation, and exercise oxygen saturation. Relevant comorbidities, including hypertension, diabetes mellitus, osteoporosis, cardiovascular disease, depression/anxiety, sleep apnea, and history of tuberculosis, were also recorded.

Statistical Analysis

Data were analyzed using appropriate statistical software. Continuous variables were expressed as mean $\pm$ standard deviation (SD), and categorical

variables as frequencies and percentages. Chi-square test, Pearson's correlation coefficient, and Spearman's rank correlation coefficient were used for statistical analysis. A p value <0.05 was considered statistically significant.

Ethical Approval

The study was approved by the Institutional Human Ethics Committee of Government Medical College and Hospital, Cuddalore District (IHEC/1123/2023) on 21.07.2023. The Institutional Ethics Committee was registered under EC/NEW/INST/2020/1249.

Written informed consent was obtained from all participants prior to enrolment.

RESULTS

Among 80 participants, the mean age was 60.11 ± 11.43 years, and 53 (66.3%) were male. The mean BMI was 23.78 ± 4.26 kg/m². Most participants had normal BMI (38, 47.5%) and were former smokers (48, 60.0%). The mean smoking exposure was 27.85 ± 15.19 pack-years. [Table 1]

Table 1: Baseline Demographic Characteristics of Study Participants (N=80)

Variable	Category	n (%) / Mean $\pm$ SD
Age (years), mean $\pm$ SD	—	60.11 $\pm$ 11.43
Age group	40–50 years	20 (25.0%)
	51–60 years	22 (27.5%)
	61–70 years	19 (23.8%)
	71–80 years	19 (23.8%)
Gender	Male	53 (66.3%)
	Female	27 (33.8%)
BMI (kg/m ²), mean $\pm$ SD	—	23.78 $\pm$ 4.26
BMI category	Underweight	10 (12.5%)
	Normal	38 (47.5%)
	Overweight	25 (31.3%)
	Obese	7 (8.8%)
Smoking status	Never	9 (11.3%)
	Former	48 (60.0%)
	Current	23 (28.7%)
Pack-years, mean $\pm$ SD	—	27.85 $\pm$ 15.19

The mean FEV₁ was $58.36 \pm 14.78\%$ predicted, FVC was $96.19 \pm 17.21\%$ predicted, and FEV₁/FVC was 0.606 ± 0.097 . GOLD stage 2 was most common (48,

60.0%), followed by stage 3 (24, 30.0%). GOLD clinical Groups D and B comprised 42 (52.5%) and 24 (30.0%) participants. [Table 2]

Table 2: Pulmonary Function and Disease Severity Profile of Study Participants (N=80)

Variable	Category	n (%) / Mean $\pm$ SD
PFT	FEV ₁ % predicted	58.36 $\pm$ 14.78
	FVC % predicted	96.19 $\pm$ 17.21
	FEV ₁ /FVC ratio	0.606 $\pm$ 0.097
GOLD stage	Stage 1	7 (8.8%)
	Stage 2	48 (60.0%)
	Stage 3	24 (30.0%)
	Stage 4	1 (1.3%)
GOLD clinical classification	Group A	10 (12.5%)
	Group B	24 (30.0%)
	Group C	4 (5.0%)
	Group D	42 (52.5%)

Abbreviations: FEV₁, forced expiratory volume in 1 second; FVC, forced vital capacity; GOLD, Global Initiative for Chronic Obstructive Lung Disease.

The mean COPD duration was 7.99 ± 4.22 years, with 1.70 ± 1.52 exacerbations and 0.35 ± 0.58 hospitalizations in the preceding year. Mean mMRC, CAT, and SGRQ scores were 1.78 ± 0.94 , $14.41 \pm$

7.19 , and 36.16 ± 18.88 , respectively. The mean six-minute walk distance was 371.81 ± 70.62 m. [Table 3]

Table 3: Clinical Characteristics, Symptom Burden and Functional Capacity of Study Participants (N=80)

Variable	Mean $\pm$ SD
COPD duration (years)	7.99 $\pm$ 4.22
Exacerbations in past year	1.70 $\pm$ 1.52
Hospitalizations in past year	0.35 $\pm$ 0.58
mMRC score	1.78 $\pm$ 0.94
CAT score	14.41 $\pm$ 7.19
SGRQ score	36.16 $\pm$ 18.88
Six-minute Walk distance (m)	371.81 $\pm$ 70.62
Resting SpO ₂ (%)	95.44 $\pm$ 1.46
Exercise SpO ₂ (%)	92.56 $\pm$ 2.64

Abbreviations: COPD, chronic obstructive pulmonary disease; mMRC, modified Medical Research Council; CAT, COPD Assessment Test; SGRQ, St. George's Respiratory Questionnaire; SpO₂, peripheral oxygen saturation.

Osteoporosis was present in 42 (52.5%) participants, hypertension in 40 (50.0%), and cardiovascular disease in 39 (48.8%). Diabetes mellitus,

depression/anxiety, sleep apnea, and a history of tuberculosis were present in 29 (36.3%), 30 (37.5%), 10 (12.5%), and 6 (7.5%) participants. [Table 4]

Table 4: Comorbidity Profile of Study Participants (N=80)

Comorbidity	n (%)
Hypertension	40 (50.0%)
Diabetes mellitus	29 (36.3%)
Osteoporosis	42 (52.5%)
Cardiovascular disease	39 (48.8%)
Depression/anxiety	30 (37.5%)
Sleep apnea	10 (12.5%)
History of tuberculosis	6 (7.5%)

The mean serum vitamin D level was 18.34 ± 10.16 ng/mL (range: 5.3–48.3). Vitamin D deficiency was present in 52 (65.0%) participants, insufficiency in 20

(25.0%), and sufficiency in 8 (10.0%). Vitamin D supplementation was reported by 24 (30.0%) participants. [Table 5]

Table 5: Vitamin D Status and Supplementation Among Study Participants (N=80)

Variable	Category	n (%)
Vitamin D status	Deficient (<20 ng/mL)	52 (65.0%)
	Insufficient (20–29 ng/mL)	20 (25.0%)
	Sufficient (≥ 30 ng/mL)	8 (10.0%)
Mean serum vitamin D level	—	18.34 ± 10.16 ng/mL (range: 5.3–48.3)
Vitamin D supplementation	Yes	24 (30.0%)
	No	56 (70.0%)

Among vitamin D-deficient participants, 28 were in GOLD stage 2, 23 in stage 3, and 1 in stage 4. Among vitamin D-insufficient participants, 1, 18, and 1 were in stages 1, 2, and 3, respectively. Of the vitamin D-

sufficient participants, 6 were in stage 1 and 2 in stage 2. The χ^2 test was 60.577 ($p < 0.001$), with Pearson's $r = -0.622$ ($p < 0.001$) and Spearman's $\rho = -0.577$ ($p < 0.001$). [Table 6]

Table 6: Association Between Vitamin D Status and GOLD Stage (N=80)

Vitamin D status	GOLD Stage				Total
	1	2	3	4	
Deficient	0	28	23	1	52
Insufficient	1	18	1	0	20
Sufficient	6	2	0	0	8
Total	7	48	24	1	80

Among vitamin D-deficient participants, 1, 12, 1, and 38 were classified into Groups A, B, C, and D, respectively. Corresponding numbers among vitamin D-insufficient participants were 2, 11, 3, and 4.

Among vitamin D-sufficient participants, 7 were in Group A and 1 in Group B. The χ^2 test was 64.370 ($p < 0.001$), with Pearson's $r = -0.651$ ($p < 0.001$) and Spearman's $\rho = -0.628$ ($p < 0.001$). [Table 7]

Table 7: Association Between Vitamin D Status and GOLD Clinical Classification (N=80)

Vitamin D status	GOLD Group				Total
	A	B	C	D	
Deficient	1	12	1	38	52
Insufficient	2	11	3	4	20
Sufficient	7	1	0	0	8
Total	10	24	4	42	80

DISCUSSION

Vitamin D deficiency was common in the present study, with 52 (65.0%) of 80 participants having serum vitamin D levels <20 ng/mL. The mean serum vitamin D level was 18.34 ± 10.16 ng/mL. This finding is comparable with other COPD populations. Lokesh et al. reported vitamin D deficiency in 64.5% of patients with COPD in a rural South Indian

population and found a higher likelihood of deficiency among COPD patients than controls (adjusted OR 5.05, 95% CI: 1.4–17.8).^[11] Turan et al. reported deficiency in 68.9% of 168 stable COPD patients, with mild, moderate, and severe deficiency in 59.6%, 25.7%, and 14.7%, respectively.^[12] These findings support the high prevalence of vitamin D deficiency observed in our cohort.

The prevalence in our study was higher than that reported by Bouloukaki et al. They also found that vitamin D-deficient patients had higher CAT scores than those without deficiency (15.8 ± 6.7 vs. 11.5 ± 5.2 , $p < 0.001$), and vitamin D deficiency remained associated with CAT ≥ 10 after adjustment (OR 2.3, 95% CI: 1.2–4.4; $p = 0.008$). Deficiency was also associated with depressive symptoms, insomnia, and poorer sleep quality.^[13] Differences in prevalence may be related to variations in population characteristics, nutritional status, smoking exposure, comorbidities, lifestyle, and environmental factors.

In the present study, vitamin D status showed a strong inverse association with GOLD spirometric stage ($\chi^2 = 60.577$, $p < 0.001$; Spearman's $\rho = -0.577$, $p < 0.001$). Of the 24 participants with GOLD stage 3 disease, 23 (95.8%) were vitamin D deficient, and the only participant with GOLD stage 4 disease was also deficient. In contrast, 6 of 7 participants with GOLD stage 1 disease had sufficient vitamin D levels. These findings suggest that lower vitamin D status was associated with greater airflow limitation in our cohort.

Similar findings were reported by Calle Rubio et al., who reported vitamin D deficiency (<20 ng/mL) was present in 63 (54.3%) patients, including 29 (25.0%) with severe deficiency (<12 ng/mL). Deficient patients had lower mean FEV₁ values than those without deficiency ($50.6 \pm 21.3\%$ vs. $60.7 \pm 17.9\%$ predicted; $p = 0.007$), higher CAT scores (median 16 vs. 10; $p = 0.033$), a more frequent exacerbator phenotype (58.7% vs. 24.5%; $p < 0.001$), and more hospitalizations in the previous year (50.8% vs. 3.8%; $p < 0.001$).^[14] These findings are consistent with the direction of our results. However, Turan et al. found no significant correlation between plasma 25-OH-D levels and spirometric COPD stage ($r = -0.087$, $p = 0.275$) and no significant relationship between vitamin D deficiency and severe COPD ($p = 0.808$).¹² Thus, the relationship between vitamin D and COPD severity may vary across populations.

Vitamin D status was also strongly associated with GOLD clinical classification in our study ($\chi^2 = 64.370$, $p < 0.001$; Spearman's $\rho = -0.628$, $p < 0.001$). Deficiency was particularly common in Group D, where 38 of 42 participants (90.5%) were deficient, whereas 7 of 10 participants (70.0%) in Group A had sufficient vitamin D levels. Bouloukaki et al. similarly reported significant differences across GOLD groups A, B, and E ($p = 0.036$), with Group B comprising 62% of vitamin D-deficient patients compared with 44% of those without deficiency.¹³ Although the classification systems differ, these findings support an association between vitamin D status and clinical disease burden.

Previous studies also support an association between vitamin D deficiency and exacerbations. Lokesh et al. reported that deficient patients were approximately three times more likely to have previous-year exacerbations (adjusted OR 3.51, 95% CI: 1.27–9.67).^[11] Zhou et al., found that severe vitamin D deficiency (<10 ng/mL) was associated with a higher

risk of severe exacerbations (HR 2.74, 95% CI: 1.55–4.84; $p < 0.01$).¹⁵ Turan et al. found a weak inverse correlation between plasma 25-OH-D levels and previous-year exacerbations ($r = -0.160$, $p = 0.044$).^[12] In the study by Calle Rubio et al., found deficient patients had a higher median number of exacerbations (1 vs. 0; $p = 0.003$), more patients with ≥ 2 exacerbations (30.2% vs. 7.5%; $p = 0.002$), and a higher exacerbator phenotype (58.7% vs. 24.5%; $p < 0.001$).^[14] Lee et al. also reported that vitamin D insufficiency/deficiency was associated with greater respiratory symptom burden, with higher odds of CAT ≥ 10 (adjusted OR 2.41, 95% CI: 1.20–4.82; $p = 0.013$) and mMRC ≥ 2 (adjusted OR 2.39, 95% CI: 1.08–5.32; $p = 0.032$).^[16]

Calle Rubio et al. also identified low physical activity (OR 3.84, $p = 0.004$), inhaled corticosteroid use (OR 3.21, $p = 0.016$), and systemic corticosteroid cycles (OR 2.15, $p = 0.002$) as factors independently associated with vitamin D deficiency.^[14] These factors were not specifically assessed in our study and therefore cannot be directly compared. Zhu et al., reported that 25(OH)D deficiency was independently associated with increased COPD incidence (HR 1.874, 95% CI: 1.659–2.117) and mortality (HR 1.598, 95% CI: 1.406–1.816) over a median follow-up of 15 years.^[17]

Overall, vitamin D deficiency was highly prevalent in our COPD cohort and showed a strong inverse association with both GOLD spirometric stage and clinical classification. Although previous studies generally support associations between low vitamin D status, poorer lung function, greater symptom burden, and exacerbations, results are not uniform. Because of the cross-sectional design, causality cannot be established. Vitamin D status may therefore be considered an associated marker of COPD disease burden, but not a substitute for spirometric assessment.

CONCLUSION

Vitamin D deficiency was common among patients with COPD in this study, affecting 65.0% of participants. Lower vitamin D status was significantly associated with higher GOLD spirometric stage and GOLD clinical classification. These findings suggest that vitamin D deficiency may be an associated marker of COPD disease severity.

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