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Original Research Article

ASSOCIATION BETWEEN VITAMIN D DEFICIENCY AND INFLAMMATORY BOWEL DISEASE (IBD)

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ABSTRACT

Background: Inflammatory Bowel Disease (IBD) is are immune-mediated disease characterized by episodes of intestinal inflammation and relapse. Recent evidence has suggested that vitamin D plays an important role in immune regulation and gut mucosal integrity. Vitamin D deficiency is prevalent in cases of IBD, which contributes to disease severity. The current study aimed to determine the association of vitamin D deficiency with IBD for potentially improving the clinical management of these cases.

Materials and Methods: This study was conducted in n=45 cases with confirmed diagnosis of Crohn's Disease (CD), or Ulcerative Colitis (UC) based on ECCO guidelines. Lab investigations included estimation of Serum 25-hydroxyvitamin D [25(OH)D] using chemiluminescence immunoassay (CLIA). Complete blood count (CBC), erythrocyte sedimentation rate (ESR), and C-reactive protein (CRP) to assess systemic inflammation. Serum calcium, phosphorus, albumin, and alkaline phosphatase were also measured to evaluate calcium-phosphorus metabolism.

Results: Vitamin D deficiency (<20 ng/mL) was found in 62.2% of cases, particularly among Crohn's disease patients 72% of cases compared to ulcerative colitis 50% of cases. Mean vitamin D levels were significantly lower in cases with active disease (15.8 ± 5.2 ng/mL) versus remission (22.9 ± 7.1 ng/mL, p=0.003). Deficiency correlated inversely with CRP (r=–0.65) and ESR (r=–0.58), and positively with albumin and calcium. Logistic regression identified active disease, Crohn's disease, low BMI, and elevated CRP as independent predictors. Corticosteroid use was significantly associated with deficiency (p=0.02).

Conclusion: Vitamin D deficiency was commonly prevalent in cases of IBD. The deficiency was more prevalent in cases of Crohn's disease as compared to Ulcerative colitis. The association was significant in active disease states, systemic inflammation, poor nutritional status, as well as corticosteroid use. Therefore, monitoring and managing the vitamin D levels should be an integral part of comprehensive care for patients with irritable bowel diseases.

Keywords: Vitamin D, Irritable Bowel Diseases (IBD), Crohn's disease (CD), Ulcerative colitis (UC).

INTRODUCTION

Inflammatory bowel disease (IBD) is are chronic relapsing inflammatory disorder of the gastrointestinal tract with having multifactorial etiology. They include Crohn's disease (CD) and ulcerative colitis (UC). There appears to be interaction between genetic predisposition, environmental triggers, microbial dysbiosis, and immune dysregulation. In recent years, there has been growing interest in determining the role of micronutrients, particularly Vitamin D, in the pathogenesis and progression of IBD. We know vitamin D for its role in calcium and bone metabolism; however, there is evidence that it acts as a crucial immunomodulatory hormone with wide effects on innate and adaptive immune responses.[1]

The biological effects of vitamin D on the immune cells appear to be due to the existence of the vitamin D receptor, which is present in T cells, B cells, dendritic cells, and macrophages. It modulates immune responses by suppressing pro-inflammatory cytokine production, which includes TNF-α, IL-6, and IL-17, and also promotes the regulatory functions of T cells. This is essential for maintaining intestinal immune tolerance.[2] Because in IBD there is chronic mucosal inflammation and immune dysregulation against intestinal microbiota, vitamin D's immunoregulatory role has emerged as an important area for research

Several observational studies have found that patients with IBD, especially those with active disease or damage to the small bowel, often experience vitamin D deficiency.[3] Factors that can cause people to lack vitamin D include difficulty in processing vitamins, eating less, spending less time in the sun, chronic inflammation, and the use of corticosteroids. Low vitamin D levels can increase disease activity, relapse more often, reduce quality of life, and expose the person with IBD to more hospitalizations and surgery.[4] Furthermore, a lower level of serum 25-hydroxyvitamin D [25(OH)D] is linked to a low response to biologic therapy and an increased risk of colorectal cancer and osteoporosis.[5] Recent evidence has shown a bidirectional relationship between vitamin D status and IBD. Some studies have shown that IBD can cause or exacerbate vitamin D deficiency through impaired absorption and increased catabolism. While other studies have found that low levels of vitamin D precede the development of IBD and contribute to its pathogenesis. Few cohort studies have also shown that people with lower baseline levels of vitamin D have a higher risk of developing IBD over time, making it a potential modifiable risk factor.[6] Vitamin D supplementation for the treatment of IBD has been studied in a few studies. Some other studies have suggested that correcting vitamin D deficiency could reduce disease activity, enhance mucosal healing, and improve the quality of life of both Crohn's disease and Ulcerative colitis patients.[7] Despite these findings, the optimal targets for vitamin D and its supplementation remain unclear, and no uniform guidelines have been found. Therefore, with this background, we in the current study aimed to determine the association between vitamin D deficiency and IBD in a cohort presenting to our hospital.

MATERIALS AND METHODS

This prospective observational study was conducted in the Department of Gastroenterology, Government Medical College and Hospital, Sangareddy, Telangana. Institutional Ethical approval was obtained for the study based on the Helsinki declaration of 1964 for human research with all its amendments till date. Written consent was obtained from all the participants of the study after explaining the nature of study in vernacular language. The sample was collected by convenience sampling method.

Inclusion Criteria

1. Patients with a confirmed diagnosis of Crohn's Disease (CD) or Ulcerative Colitis (UC) based on ECCO guidelines [8].
2. Patients in active disease or remission phase of disease (classified using CDAI for CD and Mayo score for UC) [9].
3. Aged 18 years and above
4. Males and Females
5. Voluntarily willing to participate in the study

Exclusion Criteria

1. Patients with known disorders affecting vitamin D metabolism (e.g., chronic kidney disease, hyperparathyroidism).
2. History of vitamin D supplementation in the past 3 months.
3. Coexistent chronic liver disease, malignancy, or malabsorptive conditions other than IBD.
4. Pregnant or lactating women.

Study Population: Based on the inclusion and exclusion criteria, patients of both Crohn's disease (CD) and ulcerative colitis (UC), based on clinical presentation, endoscopic findings, radiological imaging, and histopathological confirmation. Patients were recruited from the outpatient and inpatient services of the department. Demographic details, including age, sex, body mass index (BMI), and duration of illness, were recorded. Clinical data regarding disease type (CD or UC), extent, duration, activity status, medication history, and previous surgeries were obtained from medical records and patient interviews.

Assessment of Disease Activity

- In Crohn’s disease, disease activity was assessed using the Crohn’s Disease Activity Index (CDAI).
- In ulcerative colitis, disease activity was graded using the Mayo Clinic Score.
- Patients were categorized into active disease or remission based on standard cut-off scores.

Laboratory Investigations required blood samples to be collected after an overnight fast.

The following parameters were estimated in each case:

1. Serum 25-hydroxyvitamin D [25(OH)D] using chemiluminescence immunoassay (CLIA).
2. Complete blood count (CBC), erythrocyte sedimentation rate (ESR), and C-reactive protein (CRP) to assess systemic inflammation.
3. Serum calcium, phosphorus, albumin, and alkaline phosphatase were also measured to evaluate calcium-phosphorus metabolism.

Vitamin D status was classified as:

1. Deficient: <20 ng/mL
2. Insufficient: 20–29 ng/mL
3. Sufficient: ≥30 ng/mL

4. as per Endocrine Society Clinical Practice Guidelines.[10]

Statistical Analysis: All the available data were refined, segregated, and uploaded to an MS Excel spreadsheet and analyzed by SPSS version 23 (IBM Corp., Armonk, NY) in Windows format. The continuous variables were represented as frequency, mean, standard deviation, and percentages. The categorical variables were calculated by Pearson's Chi-square test was used to compare categorical variables, while an independent t-test or Mann–Whitney U test was used for continuous variables, depending on the normality of distribution. The values of p (<0.05) were considered significant.

RESULTS

A total of n=45 patients diagnosed with Inflammatory Bowel Disease (IBD), including 25 with Crohn's Disease (CD) and 20 with Ulcerative Colitis (UC), were included in the study. Table 1 shows the demographic and clinical profile of the patients in the study. The mean age of the cohort was 38.5 ± 12.3 years. Patients with UC were older (mean age 42.1 ± 13.5 years) compared to those with CD (mean age 35.2 ± 10.8 years). The gender distribution appeared to be equal, with males accounting for 53.3% and females 46.7%, and no significant gender predominance was observed within either disease group. The mean body mass index (BMI) for the cohort was 23.4 ± 3.8 kg/m². Patients with UC had having higher mean BMI compared to CD (24.9 ± 4.1 kg/m²) versus (22.1 ± 3.2 kg/m²), respectively. This reflects the more nutritional impact commonly observed in CD, possibly due to small bowel involvement impairing nutrient absorption. The mean disease duration across the cases was 6.2 ± 4.5 years, with CD patients showing a slightly longer disease history (7.1 ± 5.0 years) than UC patients (5.0 ± 3.8 years). The assessment of disease activity revealed that 62.2% of the cases were in the active phase of illness as defined by a Crohn's Disease Activity Index (CDAI) ≥150 for CD or a Mayo score ≥3 for UC. The disease activity was similar in both groups, with 64.0% of CD patients and 60.0% of UC patients having active disease at the time of assessment, while the remaining were in clinical remission. The subclassification of disease in UC cases showed 30.0% had proctitis, 45.0% had left-sided colitis, and 25.0% had extensive colitis. The patients with CD showed more variability in disease distribution, with 56.0% having ileal disease, 28.0% having isolated colonic disease, and 16.0% having ileocolonic involvement. The kind of distribution is commonly known for IBD.

Table 1: Demographic and Clinical Characteristics of the Study Cohort (n=45)

Parameter	***Total (n=45)***	***Crohn's Disease (n =25)***	***Ulcerative Colitis (n =20)***
Age (years)	38.5 ± 12.3	35.2 ± 10.8	42.1 ± 13.5
Gender			
Male	24 (53.3%)	14 (56.0%)	10 (50.0%)
Female	21 (46.7%)	11 (44.0%)	10 (50.0%)
BMI (kg/m^2)	23.4 ± 3.8	22.1 ± 3.2	24.9 ± 4.1
Disease Duration (years)	6.2 ± 4.5	7.1 ± 5.0	5.0 ± 3.8
Disease Activity			
Active (CDAI ≥150 / Mayo ≥3)	28 (62.2%)	16 (64.0%)	12 (60.0%)
Remission	17 (37.8%)	9 (36.0%)	8 (40.0%)
Disease Extent (UC only)			
Proctitis	-	-	6 (30.0%)
Left-sided colitis	-	-	9 (45.0%)
Extensive colitis	-	-	5 (25.0%)
CD Location			
Ileal	-	14 (56.0%)	-
Colonic	-	7 (28.0%)	-
Ileocolonic	-	4 (16.0%)	-

Table 2 shows the distribution of the vitamin D status among the studies. Overall, vitamin D deficiency, defined as serum 25(OH)D levels <20 ng/mL, was observed in a substantial proportion of cases (62.2%) in the study. The deficiency appeared to be more severe in patients with Crohn's disease (CD), where 72.0% were deficient compared to 50.0% in the Ulcerative Colitis (UC) group. Vitamin D insufficiency (20–29 ng/mL) was found to be present in 26.7% of all cases, and a higher proportion of UC patients was affected than CD patients (35.0% vs. 20.0%). A total of 11.1% of the patients had sufficient vitamin D levels (≥30 ng/mL). The UC group (15.0%) showed sufficiency, while the CD group (8.0%) showed Vitamin D sufficiency. The mean serum 25(OH)D concentration appeared to be significantly lower in CD cases (16.2 ± 5.9 ng/mL) than in UC cases (21.3 ± 7.1 ng/mL), indicating a stronger association between vitamin D deficiency and Crohn's disease. These results show a higher prevalence of low vitamin D levels in patients with IBD.

Table 2: Vitamin D Status Across the Cohort

Vitamin D Category	*Total (n=45)*	*Crohn's Disease (n=25)*	*Ulcerative Colitis (n=20)*
Deficient (<20 ng/mL)	28 (62.2%)	18 (72.0%)	10 (50.0%)
Insufficient (20—29 ng/mL)	12 (26.7%)	5 (20.0%)	7 (35.0%)
Sufficient (230 ng/mL)	5 (11.1%)	2 (8.0%)	3 (15.0%)
Mean 25(OH)D (ng/mL)	18.4 ± 6.8	16.2 ± 5.9	21.3 ± 7.1

Table 3 illustrates the relationship between vitamin D levels and disease activity. A critical analysis of the table shows an inverse relationship between 25(OH)D concentrations and disease activity. The results show that patients with active disease (n = 28) had significantly lower mean vitamin D levels (15.8 ± 5.2 ng/mL) compared to those in remission (22.9 ± 7.1 ng/mL), with a statistically significant difference (p = 0.003). The prevalence of vitamin D deficiency (<20 ng/mL) showed a higher prevalence in the active disease group 78.6%) than in those in remission (35.3%) (p = 0.002). The results of inflammatory markers C-reactive protein (CRP) and erythrocyte sedimentation rate (ESR) were significantly elevated in active disease (CRP: 24.5 ± 12.3 mg/L; ESR: 42.6 ± 18.7 mm/hr) compared to remission (CRP: 5.8 ± 3.1 mg/L; ESR: 18.2 ± 9.4 mm/hr), with p-values <0.001 for both. These findings suggest that lower vitamin D levels are significantly associated with more severe disease in IBD, along with increased inflammatory activity. Showing that there is a potential role of vitamin D as a biomarker of disease activity.

Table 3: Vitamin D Levels vs. Disease Activity

Parameter	Active Disease (n =28)	Remission (n= 17)	p-value
Mean 25(OH)D (ng/mL)	15.8 ± 5.2	22.9 ± 7.1	0.003*
Vitamin D Deficiency	22/28 (78.6%)	6/17 (35.3%)	0.002*
CRP (mg/L)	24.5 ± 123	5.8 ± 3.1	<0.001*
ESR (mm/hr)	42.6 ± 187	18.2 ± 9.4	<0.001*

***Significant**

Table 4 shows the correlation between serum vitamin D levels and important inflammatory and nutritional markers. There was a strong negative correlation found between vitamin D and C-reactive protein (CRP), with a Pearson's correlation coefficient (r) of -0.65 (p < 0.001), which shows that higher inflammation is associated with lower vitamin D levels. The association of vitamin D and ESR showed (r = -0.58, p < 0.001), which showed that there is an inverse relationship between hypovitaminosis D and systemic inflammation. A positive correlation was found to be present between serum albumin (r = 0.42, p = 0.004) and calcium levels (r = 0.38, p = 0.01), implying that better vitamin D status is associated with improved metabolic and nutritional profile. These statistically significant associations support the role of vitamin D not only as a potential marker of inflammation but also as a reflection of overall disease severity and nutritional status in IBD patients.

Table 4: Correlation Between Vitamin D and Inflammatory Markers

Variable	Pearson's Correlation (r)	p-value
CRP (mg/L)	-0.65	<0.001*
ESR (mm/hr)	-0.58	<0.001*
Albumin (g/dL)	0.42	0.004*
Calcium (mg/dL)	0.38	0.01*

Significant

Table 5 shows the results of multivariate logistic regression analysis. The analysis identified independent predictors of vitamin D deficiency in inflammatory Bowel Disease. A critical analysis of the table revealed that active disease was identified as a significant predictor of vitamin D deficiency with an adjusted OR of 4.8 (95% CI: 1.9–12.1, p = 0.001). The cases with Crohn's disease also showed a significant association with vitamin D deficiency (adjusted OR: 2.6, 95% CI: 1.1–6.3, p = 0.03). These results show that there is a fivefold odds of vitamin D deficiency in patients with ongoing inflammation, and patients with Crohn's disease are more likely to be deficient in vitamin D as compared with those with ulcerative colitis. Other factors, such as BMI below 18.5 kg/m², indicating undernutrition, were another significant factor (adjusted OR: 3.1, 95% CI: 1.2–8.0, p = 0.02). Elevated CRP (>10 mg/L) showed a strong association adjusted OR: 5.2, 95% CI: 2.0–13.5, p < 0.001).

Table 5: Multivariate Logistic Regression for Vitamin D Deficiency

Factor	Adjusted OR	95% CI	p-value
Active Disease	4.8	1.9 -12.1	0.001*
Crohn's Disease	2.6	1.1- 6.3	0.03*
BMI <18.5 kg/m.2	3.1	1.2 -8.0	0.02*
CRP 210 mg/L	5.2	2.0 -13.5	<0.00*1

Significant

The assessment of the use of medication with vitamin D status in the cases has been depicted in Table 6. Among the n=45 cases, we found corticosteroid use was significantly higher in the vitamin D-deficient group, 64.3%, compared to those with sufficient vitamin D levels, 20.0%. The values were statistically significant (p=0.02). This could be due to the alteration of calcium and vitamin D metabolism induced by corticosteroids. The use of immunomodulators did not show a significant difference between the deficient group and sufficient group (p = 0.53). Similarly, biologic therapy cases 40% of all cases had nearly equal distribution in two groups (42.9% vs. 40.0%, p = 0.91) with no significant association.

Table 6: Medication Use and Vitamin D Status

Medication	Total (n=45)	Deficient (n=28)	Sufficient (n=5)	p-value
Corticosteroids	22 (48.9%)	18/28 (64.3%)	1/5 (20.0%)	0.02
Immunomodulators	30 (66.7%)	20/28 (71.4%)	3/5 (60.0%)	0.53
Biologics	18 (40.0%)	12/28 (42.9%)	2/5 (40.0%)	0.91

DISCUSSION

The current study was done in 45 cases of Inflammatory Bowel Disease (IBD), which included both Crohn's disease (CD) and ulcerative colitis (UC). This study found a significant association between vitamin D deficiency and clinical and inflammatory parameters. A significant proportion of the cases, 62.2%, exhibited vitamin D deficiency. Among these, a higher prevalence of vitamin D deficiency was found in Crohn's disease, 72.0%, compared to Ulcerative colitis, 50% of cases. This showed that CD patients are often susceptible to vitamin D deficiency. Our findings are in agreement with other similar studies, which have reported lower 25(OH)D levels in CD compared to UC, which is due to greater involvement of the small bowel leading to problems with vitamin D absorption. [3,11] The results of this study showed that the mean 25(OH)D level across the cohort was 18.4 ± 6.8 ng/mL. The values were significantly lower in patients with active disease (15.8 ± 5.2 ng/mL) as compared to those with remission 22.9 ± 7.1 ng/mL, p = 0.003). The existence of vitamin D deficiency was more frequent in active disease cases, 78.6%, than in cases in remission, 35.3%, and the p-values were found to be significant. This reinforces the link between low vitamin D and increased disease activity. Similar observations were reported by Ananthakrishnan et al. [4] and Garg et al,[5] who reported vitamin D insufficiency with increased disease severity and exacerbation in IBD.

Inflammatory markers such as C-reactive protein CRP and ESR were found to be significantly elevated in active disease, and there was an inverse correlation between these parameters to serum vitamin D levels (CRP: r = -0.65, p < 0.001; ESR: r = -0.58, p < 0.001). These negative association shows that there is an immunomodulatory role of vitamin D in downregulating pro-inflammatory cytokines such as TNF-α and IL-6.[12] The association of independent predictors of vitamin D deficiency was analyzed by multivariate logistic regression analysis. The results showed that active disease was the strongest predictor of vitamin D deficiency (Adjusted OR = 4.8, p = 0.001), followed by increased CRP (>10 mg/L, OR = 5.2, p < 0.001). Modest association were shown by BMI <18.5 kg/m², OR = 3.1, p = 0.02), and CD diagnosis (OR = 2.6, p = 0.03). These results imply that inflammatory burden and nutritional compromise can act together and contribute to vitamin D levels in IBD patients. A similar observation has been shown by other studies, where they found that chronic inflammation and malabsorption are involved in the pathogenesis of vitamin D deficiency in IBD. [13,14]

A moderate positive association of vitamin D was found between serum albumin (r = 0.42, p = 0.004) and calcium (r = 0.38, p = 0.01). These results illustrate that a broader nutritional and biochemical profile is associated with deficiency. These associations augment the utility of vitamin D as a surrogate marker for nutritional status and systemic disease impact in IBD.[15] The assessment of medication use to vitamin D deficiency pointed out that patients on corticosteroids were more vitamin D deficient than compared on other medications. The corticosteroids are known to cause vitamin D deficiency by altering its metabolism and decreasing intestinal calcium absorption.[16] Few longitudinal studies have found that vitamin D supplementation could reduce relapse rates and disease outcomes, especially in cases of Crohn's disease (CD),[6] which strengthens the concept of vitamin D as a modifiable risk factor for IBD management. While the current study strengthens valuable insights, it has its limitations which including a cross-sectional design and a small sample size, which could limit the generalizability of the results. Additionally, seasonal variations of vitamin D and dietary intake were not taken into account. Therefore, longitudinal studies with a larger cohort will give a correct picture.

CONCLUSION

In conclusion, we found that vitamin D deficiency was commonly prevalent in cases of IBD. The deficiency was more prevalent in cases of Crohn's disease as compared to Ulcerative colitis. The association was significant in active disease states, systemic inflammation, poor nutritional status as well as corticosteroid use. Therefore, monitoring and managing the vitamin D levels should be

integral part of comprehensive care for patients with irritable bowel diseases.

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