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The Role of Vitamin D in Inflammatory Bowel Disease: Clinical Relevance and Therapeutic Potential

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ABSTRACT

Background:
Inflammatory bowel disease (IBD), including Crohn's disease and ulcerative colitis, is characterized by chronic relapsing inflammation of the gastrointestinal tract. Increasing evidence suggests that vitamin D plays an important role in immune regulation and intestinal barrier integrity.

Aim:
The aim of this review was to evaluate the clinical relevance of serum vitamin D levels in patients with inflammatory bowel disease and to assess the potential role of vitamin D supplementation.

Material and methods:

A narrative review of current literature concerning vitamin D and IBD was conducted using clinical studies, systematic reviews, meta-analyses, and experimental studies retrieved from databases including PubMed and Scopus.

Results:
Vitamin D deficiency is highly prevalent among patients with IBD and has been associated with increased disease activity, higher inflammatory burden, greater risk of relapse and poorer quality of life. Low serum 25(OH)D levels correlate with inflammatory markers such as C-reactive protein and fecal calprotectin. Experimental and clinical studies suggest that vitamin D

influences intestinal inflammation through modulation of immune responses, maintenance of epithelial barrier function, and regulation of gut microbiota. Available evidence indicates that vitamin D supplementation may improve selected clinical outcomes, particularly in patients with Crohn's disease in remission, although current data remain heterogeneous.

Conclusions:

Vitamin D appears to be a clinically relevant and modifiable factor in IBD management. Routine assessment and correction of vitamin D deficiency may represent a useful adjunct to standard therapy; however, further randomized controlled trials are required to establish optimal supplementation strategies.

Keywords:

vitamin D; inflammatory bowel disease; Crohn's disease; ulcerative colitis; supplementation; inflammation

1. Introduction

Inflammatory bowel disease (IBD), including Crohn's disease and ulcerative colitis, comprises a group of chronic, relapsing inflammatory disorders of the gastrointestinal tract [1,2]. Ulcerative colitis is limited to the colon and primarily affects the mucosa, whereas Crohn's disease may involve any part of the gastrointestinal tract and is characterized by transmural inflammation that may lead to strictures, fistulas, and abscesses.

The pathogenesis of IBD is multifactorial and involves genetic susceptibility, dysregulated immune responses, intestinal microbiota, and environmental factors [1–3]. Chronic inflammation contributes to progressive intestinal damage and may result in complications such as surgery, colorectal dysplasia, impaired bone health, and reduced quality of life.

In recent years, increasing attention has been directed toward modifiable factors that may influence the clinical course of IBD. Among them, vitamin D has emerged as a clinically relevant factor because of its immunomodulatory properties and its role in maintaining intestinal barrier integrity [4,5].

Vitamin D deficiency is common in patients with IBD and has been associated with increased disease activity, higher inflammatory burden, and greater risk of relapse [6–8]. Although a direct

causal relationship has not been definitively established, current evidence suggests that vitamin D status may have clinical relevance in the supportive management of IBD.

The aim of this review is to summarize current evidence regarding the relationship between serum vitamin D levels and inflammatory bowel disease, with particular emphasis on mechanisms of action, clinical relevance, and the potential role of vitamin D supplementation.

2. Research materials and methods

This narrative review was prepared on the basis of currently available literature concerning vitamin D and inflammatory bowel disease (IBD). Publications indexed in PubMed, Scopus, and Google Scholar were analyzed, with particular emphasis on studies published within the last decade. The reviewed literature included clinical studies, observational studies, systematic reviews, meta-analyses, and experimental studies related to Crohn's disease and ulcerative colitis.
The literature search was conducted using the following keywords and their combinations: "vitamin D", "25(OH)D", "inflammatory bowel disease", "Crohn's disease", "ulcerative colitis", "vitamin D deficiency", "immune regulation", "gut microbiota", and "supplementation". The selected publications were evaluated with regard to the association between serum vitamin D levels and disease activity, inflammatory markers, intestinal barrier function, immune response, and clinical outcomes in patients with IBD. Particular attention was paid to studies assessing the potential effects of vitamin D supplementation on disease course and relapse risk.
The collected data were reviewed and summarized to present the current state of knowledge regarding the clinical relevance and potential therapeutic role of vitamin D in inflammatory bowel disease.

3. Vitamin D metabolism and immunological functions

Vitamin D is a fat-soluble hormone involved not only in calcium and phosphate homeostasis but also in regulation of immune responses [5,9]. It is obtained through cutaneous synthesis under ultraviolet B radiation and from dietary sources in the form of vitamin D_2 or vitamin D_3 [9].
Vitamin D undergoes two hydroxylation steps before becoming biologically active. The first takes place in the liver, where vitamin D is converted into 25-hydroxyvitamin D [25(OH)D], the main circulating form used to assess vitamin D status. The second occurs primarily in the kidneys, where 25(OH)D is transformed into the active metabolite 1,25-dihydroxyvitamin D [$1,25(OH)_2D$, calcitriol] [9,10].

The biological activity of vitamin D is mediated through the vitamin D receptor (VDR), which is expressed in numerous tissues, including intestinal epithelial cells and immune cells such as macrophages, dendritic cells, and T lymphocytes [5,10,11]. Activation of VDR influences transcription of genes involved in immune regulation, epithelial integrity, and inflammatory signaling pathways.

Vitamin D plays an important role in innate immunity by stimulating the production of antimicrobial peptides, including cathelicidin and defensins, and by enhancing phagocytosis and autophagy [10,11]. In adaptive immunity, vitamin D suppresses pro-inflammatory Th1 and Th17 responses while promoting regulatory T-cell activity and anti-inflammatory cytokine production [5,10,11]. Experimental studies suggest that impaired vitamin D signaling may contribute to intestinal barrier dysfunction and exaggerated mucosal inflammation, mechanisms considered important in the pathogenesis of inflammatory bowel disease [5,11].

4. Vitamin D deficiency and clinical relevance in IBD

Vitamin D deficiency is highly prevalent in patients with inflammatory bowel disease and occurs more frequently than in the general population [6,7]. Most studies report decreased serum 25(OH)D concentrations in both Crohn's disease and ulcerative colitis, with lower levels more commonly observed in patients with Crohn's disease and small bowel involvement [7,8].

The development of vitamin D deficiency in IBD is multifactorial. Chronic intestinal inflammation, malabsorption, reduced dietary intake, diarrhea, and previous intestinal resections may all contribute to impaired vitamin D status. Reduced sunlight exposure and seasonal variability additionally influence serum vitamin D concentrations [12,13].

Several studies have demonstrated an association between low serum vitamin D levels and increased disease activity in IBD [14,15]. Lower 25(OH)D concentrations have been linked to elevated inflammatory markers, including C-reactive protein and fecal calprotectin, as well as higher clinical activity scores [14–16]. Patients with vitamin D deficiency were also shown to have a higher risk of relapse, hospitalization, and surgical intervention [8,16].

Vitamin D appears to influence multiple mechanisms involved in IBD pathogenesis, including regulation of immune responses, maintenance of intestinal barrier integrity, and modulation of gut microbiota composition. Experimental studies suggest that impaired vitamin D signaling may contribute to exaggerated intestinal inflammation and mucosal damage.

Some evidence indicates that vitamin D supplementation may improve selected clinical outcomes,

particularly in patients with Crohn's disease in remission [17,18]. Meta-analyses have shown a potential reduction in relapse risk and improvement in inflammatory parameters following supplementation; however, currently available evidence remains heterogeneous [16,19,20]. Although optimal serum 25(OH)D concentrations in patients with IBD have not been clearly established, levels above 30 ng/mL are generally considered sufficient [5,9]. Routine assessment and correction of vitamin D deficiency may therefore represent a useful supportive element in the management of inflammatory bowel disease.

5. Mechanism linking vitamin D and intestinal inflammation

Vitamin D influences intestinal inflammation through multiple interconnected mechanisms involving epithelial barrier integrity, modulation of gut microbiota, regulation of pro-inflammatory cytokines, and control of T lymphocyte responses [2,4,5,11]. These pathways are directly involved in the pathogenesis and progression of inflammatory bowel disease.

Intestinal barrier function

Vitamin D plays a critical role in maintaining intestinal epithelial integrity. Activation of the vitamin D receptor (VDR) strengthens tight junction complexes between epithelial cells, limiting translocation of luminal bacteria and antigens into the mucosa [4,5,11].
At the cellular level, vitamin D regulates the expression of tight junction proteins, including occludin and claudins, which are essential for maintaining epithelial cohesion and barrier stability [4,11]. In addition, vitamin D promotes the production of antimicrobial peptides in the mucus layer, helping to limit direct contact between intestinal microbiota and the epithelial surface.
Experimental studies have shown that enhanced VDR signaling is associated with improved barrier function and reduced epithelial injury in colitis models, whereas impaired vitamin D signaling increases susceptibility to mucosal damage and intestinal inflammation [5,11].

Gut microbiota

Vitamin D status has been linked to differences in gut microbiota composition and diversity. Higher vitamin D levels are associated with increased abundance of beneficial bacteria and a more favorable microbial balance, whereas deficiency may contribute to dysbiosis and pro-inflammatory microbial profiles [21].
Conversely, vitamin D deficiency is associated with dysbiosis, characterized by reduced microbial

diversity and increased prevalence of pro-inflammatory bacterial species. Some studies suggest that vitamin D may influence overall microbial composition and promote bacterial profiles associated with anti-inflammatory effects.
Seasonal variations in vitamin D levels have also been shown to correlate with changes in microbiota composition in patients with IBD, supporting a dynamic relationship between vitamin D status and the intestinal microbial environment.

Regulation of pro-inflammatory cytokines

Vitamin D exerts anti-inflammatory effects by modulating cytokine production. It suppresses the expression of key pro-inflammatory mediators involved in IBD pathogenesis, including TNF-α, IL-6, IL-17, and IFN-γ [5,10,11].
These effects are partly mediated through suppression of intracellular inflammatory signaling pathways, leading to reduced activation of macrophages and other immune cells.
At the same time, vitamin D promotes anti-inflammatory pathways and increases production of cytokines such as IL-10, contributing to restoration of immune balance [10,11].

T lymphocyte regulation

Vitamin D has a significant impact on adaptive immune responses, particularly on T lymphocyte differentiation. It suppresses pro-inflammatory Th1 and Th17 responses while promoting the development and activity of regulatory T cells [10,11].
This shift toward a more tolerogenic immune profile helps limit excessive immune activation in response to intestinal microbiota. Vitamin D acts both directly on T cells and indirectly through dendritic cells, reducing their antigen-presenting capacity and favoring anti-inflammatory signaling pathways [10,11].
Reduced expression of the vitamin D receptor has been observed in inflamed intestinal tissue in patients with IBD, suggesting impaired local responsiveness to vitamin D during active disease [5].
Experimental models further support the role of vitamin D signaling in intestinal immune regulation, as vitamin D deficiency and impaired VDR activity are associated with increased susceptibility to colitis and more severe mucosal inflammation.
These mechanisms are closely interconnected, forming a complex regulatory network in which disruption of vitamin D signaling may amplify intestinal inflammation through multiple parallel pathways.

6. Vitamin D supplementation in IBD

Vitamin D supplementation has been evaluated as an adjunctive intervention in patients with inflammatory bowel disease, particularly in those with documented deficiency or increased risk of relapse [21]. Available studies suggest that supplementation is effective in increasing serum 25(OH)D levels and may improve selected clinical and inflammatory outcomes; however, the overall strength of evidence remains limited and heterogeneous [18–20].
Interventional studies and meta-analyses indicate that vitamin D supplementation may reduce the risk of clinical relapse in patients with IBD [21]. Some studies have also demonstrated improvement in inflammatory markers and clinical activity indices following supplementation. The effect appears less certain in ulcerative colitis, where available data are more limited [18,19].
From a clinical perspective, vitamin D should not be considered a treatment capable of inducing remission as monotherapy. Its role is better understood as supportive treatment used alongside standard pharmacological therapy [19]. Correction of deficiency may be particularly relevant in patients with Crohn's disease in remission, recurrent flares, corticosteroid exposure, or low baseline serum 25(OH)D levels.
Dosing strategies vary considerably between studies. In adults, daily doses between 2000 and 4000 IU are among the most commonly used regimens and appear effective in correcting deficiency [17–19]. In patients with more severe deficiency, malabsorption, active disease, or small intestinal involvement, higher doses may be required [6,17]. Daily doses between 2000 and 4000 IU are frequently used in clinical practice, while selected protocols use higher replacement regimens or intermittent high-dose therapy. In pediatric IBD, 2000 IU daily has been studied, while some protocols use high-dose intermittent supplementation under close monitoring [22].
Seasonal variation should also be considered when planning supplementation. Serum 25(OH)D levels tend to be lower during winter and early spring because of reduced ultraviolet B exposure [9,12]. In most patients with IBD, year-round supplementation in a stable dose may be preferable because it is simple, supports adherence, and helps prevent winter decline. However, patients with documented seasonal drops, high-risk features, or persistent deficiency despite regular supplementation may require temporary dose adjustment during autumn and winter months. This is particularly relevant in countries at higher latitudes, where cutaneous vitamin D synthesis is markedly reduced for several months of the year.

The optimal target serum 25(OH)D concentration in IBD remains uncertain. Levels above 30 ng/mL are generally considered sufficient; however, some studies suggest that higher concentrations, approximately 40–60 ng/mL, may be associated with better disease control. At present, there is no universal consensus regarding the ideal therapeutic target [5,9].
Vitamin D supplementation appears to be generally safe when appropriately monitored. Nevertheless, serum 25(OH)D, calcium levels, and clinical status should be assessed during higher-dose regimens, particularly in patients receiving prolonged supplementation.
The main limitations of the available evidence include heterogeneous study populations, small sample sizes, different definitions of relapse and remission, variable baseline vitamin D status, and inconsistent dosing protocols. Most studies focus on patients in remission, whereas evidence regarding the ability of vitamin D to induce remission in active IBD remains insufficient. Larger randomized controlled trials are needed to determine optimal dosing, target serum levels, and the patient groups most likely to benefit [18–20].

7. Discussion and clinical implications

The relationship between vitamin D and inflammatory bowel disease is complex and likely multifactorial. Although vitamin D deficiency is highly prevalent in this population, it remains unclear whether it represents a cause, a consequence, or both.
Vitamin D influences several key mechanisms relevant to IBD pathogenesis, including immune regulation, intestinal barrier integrity, and gut microbiota composition [23]. Similar observations regarding the immunomodulatory role of vitamin D in autoimmune diseases have also been reported in recent review papers [24]. Most available evidence is derived from observational studies, and interventional trials have shown heterogeneous results, limiting the ability to establish a clear causal relationship. This highlights the need to interpret existing data with caution and within the context of study design limitations.

Routine assessment of vitamin D status

From a clinical perspective, routine assessment of serum 25(OH)D levels in patients with IBD appears justified, particularly in patients with Crohn's disease, ileal involvement, recurrent disease exacerbations, or previous intestinal surgery [6,12]. Measurement of 25(OH)D provides a simple and non-invasive tool that may complement established biomarkers such as C-reactive protein and fecal calprotectin.

Importantly, interpretation of vitamin D levels should take into account disease activity, as inflammatory states may influence serum biomarkers. Assessment during remission and follow-up after supplementation may therefore provide more reliable information.

Patients at increased risk of deficiency

Certain subgroups of patients with IBD are at particularly high risk of vitamin D deficiency and may require closer monitoring. These include patients with Crohn's disease, especially those with ileal involvement or a history of intestinal resection, due to impaired absorption of fat-soluble vitamins. Additional risk factors include active disease with ongoing inflammation, chronic diarrhea, smoking, and prior disease-related complications.

Seasonal variation should also be considered, as serum 25(OH)D levels typically decrease during winter and early spring due to reduced ultraviolet B exposure. Patients living in higher latitudes may therefore be particularly susceptible to deficiency during these periods. In clinical practice, assessment in early spring—when levels are expected to be lowest—may help identify patients requiring dose adjustment.

Clinical significance and practical relevance

Vitamin D status may have broader clinical implications beyond bone health. Lower serum 25(OH)D levels have been associated with increased disease activity, higher risk of relapse, and greater healthcare utilization, including hospitalizations and surgical interventions. Some evidence also suggests that adequate vitamin D levels may be associated with improved response to biological therapies, particularly anti-TNF agents.
Although vitamin D cannot replace standard pharmacological treatment, it represents a modifiable and easily correctable factor in IBD management. Correction of deficiency is generally safe, inexpensive, and widely accessible, making it a practical component of comprehensive patient care.

Limitations and future directions

Despite these observations, the clinical impact of vitamin D supplementation remains uncertain. The available evidence is limited by heterogeneity in study design, patient populations, baseline vitamin D status, and supplementation protocols. In addition, most studies have focused on patients in remission, and data regarding the role of vitamin D in inducing remission in active disease are scarce.

Further well-designed randomized controlled trials are required to define optimal dosing strategies, target serum levels, and the specific patient populations most likely to benefit from supplementation.

8. Conclusions

Vitamin D deficiency is highly prevalent in patients with inflammatory bowel disease and is consistently associated with increased disease activity, higher risk of relapse, and less favorable clinical outcomes. Although a direct causal relationship has not been definitively established, accumulating evidence suggests that vitamin D plays a relevant role in the pathophysiology of IBD. Vitamin D influences several key mechanisms involved in intestinal inflammation, including modulation of innate and adaptive immune responses, maintenance of epithelial barrier integrity, and regulation of gut microbiota composition. These biological effects provide a strong theoretical basis for its potential role as a supportive factor in disease management.
From a clinical perspective, assessment of serum 25(OH)D levels represents a simple, accessible, and potentially valuable component of routine care. Monitoring may be particularly important in patients with active disease, ileal involvement, prior intestinal surgery, or recurrent exacerbations. Identification and correction of deficiency may contribute to improved overall patient outcomes. Vitamin D supplementation is safe, inexpensive, and effective in restoring adequate serum levels. While it is unlikely to induce remission as monotherapy, available evidence suggests that it may reduce the risk of relapse and improve selected clinical and inflammatory parameters when used as an adjunct to standard therapy.

Despite these promising findings, the current evidence base remains limited by heterogeneity of study designs, variability in supplementation protocols, and inconsistent clinical endpoints. As a result, optimal dosing strategies and target serum concentrations have not yet been clearly established.
In conclusion, vitamin D should be regarded as a modifiable and clinically relevant factor in the management of inflammatory bowel disease. Routine monitoring and individualized supplementation may be justified, particularly in high-risk populations. Future research should focus on identifying patient-specific factors that may predict response to vitamin D supplementation, enabling a more personalized approach to IBD management.

Disclosure

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