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Vitamin B12 Supplementation and Treatment in Cancer Patients: A Narrative Review

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ABSTRACT

Vitamin B_{12} (cobalamin) deficiency is frequent in oncology, resulting from nutritional deficits, malabsorption, drug interactions, and tumor-related mechanisms. Correction of deficiency is crucial to prevent hematologic, neurologic, and nutritional complications and to optimize tolerance to chemotherapy, especially antifolates such as pemetrexed. This review summarizes recent evidence on the prevalence, mechanisms, diagnosis, and clinical management of vitamin B_{12} deficiency in cancer patients, including supplementation strategies and safety considerations.

Keywords

Vitamin B_{12}, Cobalamin, Cancer, Chemotherapy, Pemetrexed, Malabsorption, Anemia, Supplementation, Oncology nutrition.

Introduction

Vitamin B_{12} (cobalamin) is a vital micronutrient required for DNA synthesis, erythropoiesis and proper nervous system function. In oncology, B_{12} status deserves particular attention: cancer patients frequently develop deficiency due to anorexia, cachexia, gastrointestinal surgery, chemotherapy-induced mucosal injury, or interference with absorption or metabolism. A recent review estimated that among patients with solid tumors, the prevalence of B_{12} deficiency might range from 6% to 48%, with higher rates in older patients and those with gastrointestinal cancers [1].

The clinical consequences in this context extend beyond classical macrocytic anemia — they encompass neuropathy and cognitive impairment, which may worsen or mimic chemotherapy-induced peripheral neuropathy (CIPN) [2]. Additionally, because B_{12} metabolism intersects with folate-dependent one-carbon pathways, many anticancer agents targeting folate metabolism may further disrupt methylation, DNA synthesis and repair mechanisms [3].

Given these intertwined risks, understanding the dynamics of cobalamin — its metabolism, potential deficiency, and rational supplementation — is critical in oncology for both prevention and supportive care.

This chapter aims to review current evidence on B_{12} status in cancer patients, examine the impact of deficiency on hematological and neurological outcomes, and discuss strategies for supplementation and monitoring in the context of anticancer therapy.

Mechanisms of Vitamin B12 Deficiency in Cancer Patients

Vitamin B_{12} deficiency in patients with cancer often results from a convergence of multiple, overlapping mechanisms that compromise intake, absorption, and metabolism (Table 1). First, reduced dietary intake is frequent in oncology due to anorexia, cachexia, nausea, or dysgeusia, leading to inadequate cobalamin supply [1]. Second, anatomical alterations — including gastrectomy, ileal resection, or prior pelvic radiotherapy — may impair essential steps in the vitamin's absorption: the loss of gastric parietal cells reduces intrinsic factor (IF) secretion, while ileal resection or injury diminishes receptor-mediated uptake [4]. Third, exocrine pancreatic insufficiency or damage to pancreatic proteases may hinder the dissociation of food-bound B_{12} from binding proteins (haptocorrin), preventing subsequent binding to IF — a critical step for ileal absorption [1]. Fourth, long-term use of medications common in cancer care — such as proton-pump inhibitors (PPIs) — may exacerbate malabsorption by reducing

gastric acidity, thus interfering with B_{12} release from proteins. Fifth, chemotherapy, antibiotics and radiotherapy-induced alterations of the gut microbiota can disrupt microbial synthesis or recycling of corrinoids, potentially exacerbating deficiency risk [1]. Finally, a functional deficit may arise not only from impaired absorption but also from enhanced tumor consumption of cobalamin or overproduction of cobalamin-binding proteins (such as haptocorrin) by malignant cells, which sequester B_{12} and reduce its bioavailability for normal tissues.

Empirical data support the high prevalence of B_{12} deficiency or borderline status among cancer patients. A recent narrative review reported prevalence estimates ranging from 6% to 48% in populations with solid tumors, with elevated risk in older individuals and those with gastrointestinal malignancies [1]. In surgical oncology, a meta-analysis of gastrectomy cohorts found a pooled prevalence of B_{12} deficiency of 48.8% (95% CI 32.4–65.2%) following resection for gastric cancer [5]. Another prospective study of esophagostomy patients reported that 18% developed B_{12} deficiency within a year postoperatively, underlining the rapidity with which depletion can occur when absorption mechanisms are disrupted [6].

These data underscore that B_{12} deficiency in cancer patients is rarely attributable to a single cause; rather, it reflects the additive impact of nutritional, surgical, iatrogenic and disease-related factors. Consequently, clinicians should maintain a high index of suspicion and consider routine surveillance of B_{12} status, especially in patients undergoing gastrointestinal surgery, pelvic radiotherapy, or prolonged PPI/chemotherapy regimens.

Biological Diagnosis and Interpretation

Assessing vitamin B_{12} status in oncology — as in the general population — is fraught with challenges because no single biomarker serves as a definitive "gold standard". Reliance on serum total B_{12} alone is particularly problematic: total B_{12} assays measure both metabolically active and inactive fractions. In cancer patients, total B_{12} may be falsely elevated due to hepatic release, inflammation, or overproduction of binding proteins (e.g., haptocorrin) by malignant cells, thus masking true deficiency [7,8].

To improve diagnostic accuracy, analysis of active B_{12} — notably holotranscobalamin (holo-TC) — as well as functional markers like methylmalonic acid (MMA) (and sometimes total homocysteine, tHcy) is widely recommended [8]. The active fraction (holo-TC) represents the portion of circulating cobalamin bound to transcobalamin II, which is available for cellular uptake; by contrast, B_{12} bound to haptocorrin is largely metabolically inert. As such, holo-TC may detect early depletion before changes in total B_{12} or overt clinical manifestations occur [9].

Large-scale data support the superior diagnostic performance of holo-TC. In a cohort of 11,833 patients with concurrent measurements of total B_{12}, holo-TC, MMA and tHcy, holo-TC showed the highest area under the ROC curve (AUC = 0.912) for detecting subclinical B_{12} deficiency (as defined by a composite index), slightly outperforming MMA (AUC = 0.904) and total B_{12} (AUC = 0.899). tHcy performed more poorly (AUC = 0.789). These findings suggest holo-TC as the preferred first-line biomarker in many contexts. Importantly, in women over 50 years, holo-TC outperformed total B_{12}, MMA and tHcy; in men and

Table 1: Main Mechanisms of Vitamin B12 Deficiency in Cancer Patients.

Mechanism	Underlying process	Clinical impact / relevance
Reduced dietary intake	Anorexia, cachexia, dysgeusia, nausea reduce consumption of B_{12}-containing foods.	Progressive depletion of stores; common in advanced disease and during chemotherapy.
Gastric resection (partial/total gastrectomy)	Loss of parietal cells → ↓ intrinsic factor and ↓ gastric acid, preventing release of food-bound B_{12}.	High prevalence of deficiency within months post-gastrectomy; requires lifelong monitoring and supplementation.
Ileal resection or ileal injury	Loss or inflammation of terminal ileum impairs intrinsic-factor receptor–mediated absorption.	Chronic malabsorption; often severe and irreversible.
Pelvic or abdominal radiotherapy	Radiation injury to ileum or gastric mucosa reduces secretion of intrinsic factor and damages absorptive capacity.	Progressive malabsorption; elevated risk in patients receiving high cumulative doses.
Proton-pump inhibitor use	Hypochlorhydria prevents release of protein-bound B_{12} from food.	Increased risk with long-term therapy (>1 year).
Metformin	Alters calcium-dependent uptake of IF–B_{12} complex in the ileum.	Causes biochemical deficiency in long-term users; incidence increased in older cancer patients with diabetes.
Chemotherapy-induced mucosal injury	Cytotoxic injury reduces epithelial turnover and enzyme activity needed for absorption.	Transient or sustained malabsorption; most frequent with antimetabolites and platinum drugs.
Antibiotics and broad-spectrum antimicrobial therapy	Disruption of gut microbiota reduces microbial contribution to corrinoid recycling and affects absorption.	Higher risk during prolonged courses; contributes to marginal B_{12} status.
Exocrine pancreatic insufficiency	Pancreatic protease deficiency prevents release of B_{12} from haptocorrin, blocking transfer to intrinsic factor.	Functional deficiency even with adequate intake.
Tumor consumption of B_{12}	Rapidly proliferating tumor cells increase B_{12} uptake to sustain DNA synthesis.	Leads to relative deficiency in host tissues; seen in high-burden disease.
Excess tumor-associated haptocorrin ("HC-sequestration")	Some malignancies overproduce haptocorrin, which binds B_{12} with high affinity but is not bioavailable.	Results in low holotranscobalamin and functional deficiency despite normal or high total B_{12} levels.
Inflammation and acute-phase response	Redistribution of transcobalamins and altered hepatic metabolism reduce circulating active B_{12}.	Contributes to "functional" deficiency in critical illness and advanced cancer.

younger women, holo-TC, MMA and total B_{12} offered comparable performance [7].

Nevertheless, holo-TC has important limitations. Its interpretation is complicated by renal insufficiency (which affects clearance of B_{12} and its binding proteins), by genetic polymorphisms of transcobalamin, and by variability in assay availability and standardization. In such contexts, low holo-TC should prompt confirmatory testing with MMA or tHcy [8,9].

MMA remains the most specific functional biomarker: when intracellular cobalamin is insufficient, methylmalonyl-CoA mutase activity drops and MMA accumulates. However, MMA assays (typically by LC–MS/MS) are more costly and less widely available, and MMA levels are influenced by renal function, which can lead to false-positive results in renal failure [10,11].

Given the strengths and limitations of each marker, combined interpretation — integrating total B_{12}, holo-TC and MMA (and tHcy if available) — is recommended, especially in equivocal cases, e.g., cancer patients with risk factors for absorption or binding abnormalities. In complex settings, a composite index (such as the 4-parameter cB12) may help refine diagnostic accuracy, but its routine use remains limited by availability of all assays and lack of widespread standardization [7,8].

Thus, in oncology practice, a stepwise diagnostic algorithm is advised: start with total B_{12} or holo-TC; if results are borderline or if risk factors exist (malabsorption, renal impairment, malignancy), proceed with MMA (± tHcy); interpret in the context of clinical and biochemical data; and consider repeat testing or supplementation rather than relying on a single normal B_{12} result (Table 2).

Clinical Consequences of Deficiency

Vitamin B12 deficiency in oncology has broad hematologic and neurologic implications that frequently overlap with cancer- or treatment-related toxicities, complicating recognition. Hematologic manifestations include macrocytic anemia, leukopenia, and thrombocytopenia, which may be misattributed to myelosuppressive chemotherapy. In a cohort of hospitalized patients, megaloblastic anemia attributable to B12 deficiency accounted for up to 18% of macrocytosis cases, underscoring its relevance even in complex clinical settings [12].

Neurological consequences—paresthesia, posterior column dysfunction, gait instability, cognitive slowing—can mimic or exacerbate chemotherapy-induced peripheral neuropathy. A large prospective study confirmed that neuropsychiatric impairment might occur even in the absence of anemia, emphasizing the need for metabolic evaluation in symptomatic cancer patients [13].

Untreated deficiency contributes to profound fatigue, impaired functional status, and reduced quality of life. In cancer patients, these consequences may further limit chemotherapy adherence and dose intensity [1]. Correction of B12 deficiency reliably improves hematologic parameters, typically normalizing reticulocyte counts within days and hemoglobin over several weeks [14]. Neurological recovery is more variable and depends on the duration and severity of deficiency; early intervention is associated with partial to substantial improvement, whereas delayed treatment may leave persistent deficits [15]. However, evidence for a direct effect on cancer survival remains limited, and current data suggest that supplementation primarily improves supportive-care outcomes rather than oncologic prognosis.

Supplementation Strategies in Cancer

Vitamin B12 supplementation in oncology has three overarching objectives: to correct established deficiency, to prevent antifolate-associated toxicity, and to optimize tolerance to cytotoxic therapy. In patients with confirmed deficiency, conventional repletion regimens consist of hydroxocobalamin 1000 µg intramuscularly (IM) each week for 4–6 weeks, followed by maintenance dosing every 1–3 months or cyanocobalamin 1000 µg IM each days for one weeks, than each week for 4 weeks, followed by 1000 µg IM monthly [14, 15]. Alternatively, high-dose oral cyanocobalamin (1–2 mg per day) corrects deficiency in more than 95% of patients with intact absorption [15] (Table 3). The most robust evidence for mandatory prophylactic supplementation comes from studies evaluating antifolate therapy. In early pemetrexed trials performed without vitamin supplementation, grade 3–4 hematological toxicities occurred in 38–47% of patients and treatment-related mortality reached 14% [16]. Introduction of systematic supplementation with 1000 µg B12 intramuscularly every 9 weeks, combined with daily folic acid (350–1000 µg), reduced grade 3–4 neutropenia to 5–10%, thrombocytopenia to 2–4%, and treatment-related mortality to below 1% [17]. These data demonstrate a relative risk reduction of approximately 70–80% for severe hematological toxicity. Supplementation also decreases hospitalizations for febrile neutropenia and improves treatment continuity, without diminishing antitumor efficacy. In patients with borderline biochemical indices, empirical oral B12 is reasonable because high-dose regimens are safe, inexpensive, and achieve biochemical correction in up to 80–90% of cases even in mild malabsorption [14]. Nevertheless, thresholds for intervention in functional or subclinical deficiency remain debated, owing to the lack of prospective trials evaluating survival or quality-of-life outcomes.

Table 2: Biological Interpretation of Vitamin B12 Status in Oncology.

Marker	Function	Limitations	Cutoff	Clinical Use
Total B12	Total circulating cobalamin	Falsely high in inflammation, cancer	<200 pg/mL	Screening
Holotranscobalamin	Active B12 fraction	Limited availability	<35 pmol/L	Specific early marker
Methylmalonic acid	Functional deficiency marker	Elevated in renal failure	>300 nmol/L	Sensitive indicator
Homocysteine	B12–folate pathway marker	Elevated in folate deficiency	>15 µmol/L	Complementary marker

Evidence from Recent Clinical Studies

Evidence from contemporary clinical trials and observational cohorts confirms that vitamin B_{12} repletion has clear clinical value in specific oncological settings, notably in patients receiving antifolate chemotherapy and in those with post-gastrectomy malabsorption. The pivotal early clinical development of pemetrexed demonstrated high rates of severe myelosuppression when the drug was given without vitamin supplementation; subsequent protocol changes that introduced routine folic acid and B_{12} markedly improved tolerability [17,18]. Later pragmatic studies have shown that, with supplementation, rates of grade 3–4 hematological toxicity fall from the high levels observed in unsupplemented cohorts (reported as ~30–40% in early experience) down to single-digit or low-teens percentages in contemporary supplemented series, with corresponding reductions in treatment-related mortality and hospitalization for febrile neutropenia [17,19]. Prospective investigations have also tested the timing and route of supplementation: shortened lead-in intervals and same-day B_{12} administration appear safe in retrospective and prospective cohorts, facilitating clinically acceptable scheduling without increased toxicity [19,20].

Outside antifolate regimens, the strongest evidence relates to post-gastrectomy patients, in whom routine parenteral or high-dose oral B_{12} prevents biochemical deficiency in >90% of treated patients and substantially reduces the incidence of macrocytic anemia and symptomatic neuropathy in follow-up [4,21]. By contrast, high-quality randomized trials of B_{12} supplementation addressing broader oncologic outcomes (quality of life, chemotherapy dose intensity, survival) are scarce. Systematic reviews and narrative syntheses (including recent reviews addressing folate/antifolate interactions) conclude that B_{12}+folate prophylaxis reliably reduces antifolate-related hematological toxicity and improves tolerability, but evidence for benefits outside these contexts (for example, routine prophylaxis for all patients receiving cytotoxic chemotherapy) remains limited [1,4]).

In sum, the evidence base supports targeted B_{12} repletion (1) as mandatory co-medication with pemetrexed and similar antifolate regimens, (2) as routine secondary prevention in patients with anatomical or functional risk for malabsorption (e.g., total gastrectomy), and (3) as a low-risk empiric option in symptomatic patients or those with borderline biochemical indices while diagnostic workup is pending. Claims that broad meta-analyses (2020–2025) uniformly confirm a benefit of B_{12} across all chemotherapy types are not supported by the current literature — the strongest and most consistent data apply to antifolate therapy and gastrectomy cohorts.

Safety and Interpretation of Elevated B12

Vitamin B_{12} supplementation is remarkably safe, with adverse events typically limited to rare injection-site pain, rash, or mild acneiform eruptions following high-dose intramuscular administration. However, the interpretation of serum B_{12} levels requires caution, particularly at the extremes. Falsely high B_{12} values are frequently observed in contexts such as hepatopathies, leukemias, or advanced solid cancers due to excessive haptocorrin secretion [22,23]. The presence of elevated B_{12} should therefore prompt thorough etiologic exploration to identify underlying pathology rather than leading to empiric vitamin supplementation. Conversely, a state of false low or functional deficiency can occur when the active fraction, holotranscobalamin (holo-TC), is low despite normal total B_{12} levels, or when MMA elevation is due to impaired renal clearance [10]. Furthermore, persistently high B_{12} levels have been associated with poor prognosis in malignancy, suggesting the value reflects tumor burden and increased cellular turnover rather than actual vitamin toxicity.

Conclusion and Perspectives

Vitamin B_{12} deficiency, although frequent in the oncological setting due to the impact of treatments and comorbidities (notably surgical residues and restrictive diets), represents a perfectly manageable complication. The improvement in hematologic stability through early detection and targeted supplementation is an essential intervention that reduces the frequency and severity of anemia and cytopenias, thereby minimizing treatment-related toxicities and the need for blood transfusions. These measures directly translate into a global improvement in patients' quality of life.

Given the prevalence of risk factors in this population—particularly exposure to antifolate drugs (such as methotrexate), a history of gastrointestinal resections (often following bariatric or digestive tumor surgery), or chronic malnutrition—regular monitoring of B_{12} status should be proactively integrated into supportive cancer care protocols. This monitoring is ideally performed using functional markers (such as holo-TC or MMA), as total serum B_{12} can be misleading in the presence of certain malignancies.

In conclusion, B_{12} therapy remains a safe, inexpensive, and indispensable pillar of supportive care in oncology. The future of this management is moving towards personalized medicine, where the assessment of B_{12} deficiency risk will no longer be solely

Table 3: Practical Recommendations for Vitamin B12 Supplementation in Cancer.

Clinical Situation	Indication	Regimen	Monitoring	Comment
Confirmed deficiency	Correction	1000 µg IM weekly × 4–6, then monthly	CBC + B12 at 3 months	Identify underlying cause
Pemetrexed chemotherapy	Toxicity prevention	1000 µg IM every 9 weeks + folate	During therapy and 3 weeks after	FDA/EMA guideline
Neuropathy with low B12	Functional correction	Standard IM regimen	Neurological follow-up	Evidence limited
Elevated serum B12	No supplementation	Etiologic work-up	As indicated	Marker of disease burden
At-risk patient	Screening	Annual B12 ± holo-TC	—	Not systematic

Table 4: Adverse Effects and False Deficiency/Hypervitaminemia Situations.

Category	Description	Clinical Implication
Injection reactions	Pain, erythema, swelling	Benign, self-limited
Allergy (rare)	Rash, urticaria, anaphylaxis	Discontinue, refer to allergy specialist
Acneiform or rosacea flare	Reported after high-dose IM	Usually transient
False hypervitaminemia	Hepatic disease, metastases, hematologic malignancy	Investigate etiology, avoid unnecessary therapy
False deficiency	MMA ↑ in renal failure; holo-TC ↓ with normal B12	Use functional markers
Prognostic marker	High B12 = marker of inflammation/tumor load	Consider as disease biomarker

clinical but will integrate specific genetic and metabolomic factors for each patient, allowing for the optimization of supplementation doses and frequency even before symptoms appear.

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