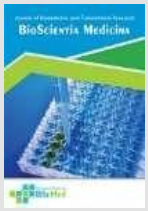

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Early Individualized Medical Nutrition Therapy in Recurrent Luminal B Breast Cancer Complicated by Post-Chemotherapy Febrile Neutropenia and Pancytopenia with Moderate Malnutrition: A Case Report

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ABSTRACT

Background: Breast cancer is the most common malignancy among women, and malnutrition affects approximately 30–50% of hospitalized patients with cancer, independently increasing treatment-related toxicity, prolonging hospitalization, and worsening survival. When cytotoxic chemotherapy precipitates febrile neutropenia and pancytopenia, reduced intake, systemic inflammation, and bone-marrow suppression converge, yet nutritional care is frequently deprioritized during the neutropenic nadir.

Case presentation: A 58-year-old woman with recurrent left breast cancer (luminal B subtype), previously treated with mastectomy and three chemotherapy cycles, presented with two weeks of anorexia, fever (40.6 °C), vomiting, diarrhea, and 3 kg weight loss. Anthropometry showed a body mass index of 17.6 kg/m^2 and a mid-upper-arm circumference of 23 cm; laboratory testing confirmed febrile neutropenia (absolute neutrophil count 0.05 ×10^3/µL) with pancytopenia and hypoalbuminemia (2.7 g/dL). She met three phenotypic and two etiologic Global Leadership Initiative on Malnutrition (GLIM) criteria, establishing moderate malnutrition. Alongside supportive care and granulocyte colony-stimulating factor, individualized medical nutrition therapy targeting 2,100 kcal/day and 90 g protein/day was delivered as a soft oral diet with a peptide-based oral nutritional supplement and vitamin B12. Oral intake rose from 50% or less to 86% by day three and 93% by day four; over six days, weight, appetite, physical function, and all hematologic indices recovered, and she was discharged stable.

Conclusion: Early, individualized medical nutrition therapy initiated before severe malnutrition develops can support concurrent nutritional, immune, and hematologic recovery in breast cancer patients with post-chemotherapy hematologic toxicity. Routine nutritional screening and prompt, protein-focused intervention should be integrated into oncology care.

1. Introduction

Breast cancer remains the most frequently diagnosed malignancy among women worldwide and a leading cause of cancer death, imposing a growing clinical and public-health burden as incidence continues to rise and survivorship lengthens.[1] In many countries cancer has become the second most common cause of death after cardiovascular disease, and breast cancer consistently represents the single largest share of new female cancer diagnoses. The disease is biologically heterogeneous: gene-expression profiling distinguishes hormone-receptor-driven luminal tumors from basal-like phenotypes, and the luminal subtypes, defined largely by estrogen-receptor expression and proliferative activity, dictate both prognosis and the intensity of systemic therapy a patient is likely to receive over the course of the illness.[1] Patients with recurrent disease, in particular,

accumulate the cumulative metabolic costs of repeated cytotoxic exposure superimposed on the catabolic drive of the tumor itself.

Malnutrition is one of the most prevalent and least addressed comorbidities in oncology. Prospective application of the Global Leadership Initiative on Malnutrition (GLIM) framework shows that malnutrition is common and that its measured prevalence varies with the screening tool used, ranging from roughly one third to more than half of acutely hospitalized patients.[2] In oncology the burden is especially high, and malnutrition or nutritional risk is widely reported to affect a large share of patients with cancer, frequently cited in the range of 30–50% depending on tumor type, stage, and treatment phase.[3] The clinical consequences are not cosmetic. A large multicenter observational cohort applying GLIM-defined malnutrition across cancer patients demonstrated that malnourished patients have significantly worse survival, with malnutrition independently associated with increased mortality.[4] It has been estimated that a meaningful fraction of cancer deaths are attributable to the consequences of malnutrition rather than to tumor progression alone, underscoring that nutritional status is a modifiable determinant of outcome rather than a passive marker of disease severity.

The intersection of malnutrition with chemotherapy-induced hematologic toxicity is clinically consequential and comparatively understudied. Febrile neutropenia and pancytopenia are among the most serious complications of cytotoxic chemotherapy; neutropenia typically reaches its nadir within roughly one to two weeks of treatment, and febrile neutropenia, defined by fever in the setting of a critically reduced absolute neutrophil count, carries appreciable morbidity and mortality. A growing body of evidence indicates that poor nutritional status and low skeletal-muscle mass are associated with a higher incidence of chemotherapy-related adverse events, including febrile neutropenia and treatment delays, across hematologic and solid tumors.[5,6] In breast cancer specifically, loss of muscle mass during chemotherapy is common and has been linked to greater toxicity and impaired outcomes, so that the nutritional and hematologic dimensions of recovery are tightly coupled rather than independent.[7,8]

The biology of breast cancer is relevant to its nutritional consequences. Hormone-receptor expression and proliferative indices separate the luminal A and luminal B subtypes, and luminal B tumors, which combine hormone sensitivity with higher proliferative activity, generally warrant more intensive systemic therapy. Patients with recurrent disease, such as the woman described here, frequently accumulate exposure to multiple lines of cytotoxic treatment, each of which carries its own gastrointestinal and hematologic toxicity and each of which adds incrementally to the catabolic burden borne by an already vulnerable host. Lifestyle and dietary factors are increasingly recognized to influence outcomes after a breast cancer diagnosis; healthy dietary patterns rich in vegetables, fruit, whole grains, legumes, and unsaturated fats, with limited saturated fat and red meat, have been associated with improved overall survival, while structured nutrition and exercise interventions can improve the capacity to complete planned chemotherapy.[9,10] Nutritional status, in other words, is woven through the entire breast cancer trajectory, from the tolerance of acute treatment to long-term survivorship.

Several mechanisms explain why patients with cancer are vulnerable to malnutrition. Reduced intake arises from primary anorexia of central origin and is aggravated by an array of secondary, often treatable, nutrition-impact symptoms: nausea, vomiting, diarrhea, mucositis, taste and smell alterations, early satiety, pain, and drug side effects. Superimposed systemic inflammation drives a catabolic state characterized by accelerated proteolysis, insulin resistance, and loss of skeletal muscle, frequently reflected in falling serum albumin and rising inflammatory markers. The result is a self-reinforcing cycle in which inflammation suppresses appetite, reduced intake depletes substrate, and progressive muscle loss further impairs immune competence and the capacity to withstand the next cycle of therapy.[3]

Despite robust guideline support for early nutritional intervention, structured nutritional care is still inconsistently delivered during acute, high-risk phases of treatment such as the neutropenic nadir, when clinical attention is understandably focused on infection control and transfusion support. This case addresses that gap. We describe a patient with recurrent luminal B breast cancer who presented during a profound post-chemotherapy nadir with the simultaneous burdens of febrile neutropenia, pancytopenia, and GLIM-defined moderate malnutrition, and who received an early, individualized, predominantly oral medical nutrition therapy strategy delivered in parallel with infectious and hematologic support. The aim of this report is to illustrate the rationale, composition, and short-term clinical and hematologic response to nutrition therapy initiated before severe malnutrition developed. Its novelty lies in documenting the practical, bedside integration of a protein-focused oral regimen, built around a peptide-based oral nutritional supplement and micronutrient repletion, into the management of the malnutrition-myelosuppression dyad in a resource-conscious setting, and in demonstrating that meaningful recovery is achievable even when daily intake never reaches 100% of the prescribed target.

2. Case Presentation

A 58-year-old woman was referred to the clinical nutrition service by the surgical oncology team with a working diagnosis of recurrent left breast cancer (luminal B subtype) complicated by febrile neutropenia and pancytopenia. Her principal complaint was a marked decrease in appetite that had begun approximately two weeks earlier, following her third cycle of chemotherapy, and had worsened over the preceding week. The anorexia was accompanied by fever, nausea, repeated vomiting (more than five episodes per day), watery diarrhea (more than three episodes per day), and an unintentional weight loss of 3 kg over the previous two months.

Her oncologic history was notable for recurrent breast cancer of the luminal B subtype. She had undergone mastectomy in March 2021 and had subsequently received three cycles of a doxorubicin-based cytotoxic chemotherapy regimen between June and July 2025. Her family history was significant for cancer on both the paternal and maternal sides, and her mother had been diagnosed with aplastic anemia, a background of potential relevance to her own marked marrow vulnerability.

On anthropometric assessment, her body weight was 48 kg and her height was 165 cm, yielding a body mass index (BMI) of 17.6 kg/m^2, within the underweight range. Her mid-upper-arm circumference (MUAC) measured 23 cm, consistent with reduced muscle mass. On physical examination she appeared weak but was fully alert (compos mentis). Her blood pressure was 91/59 mmHg, heart rate 99 beats/min, respiratory rate 20 breaths/min, and axillary temperature 40.6 °C. The conjunctivae were pale, and there was edema of the left upper extremity consistent with post-mastectomy lymphedema. The remainder of the examination was within normal limits.

Laboratory investigation on admission confirmed the clinical impression of profound post-chemotherapy marrow suppression with electrolyte and protein derangements, as detailed in Table 1. There was severe leukopenia with a total white-cell count of 0.43 $\times 10^3/\mu L$ and a critically low absolute neutrophil count of 0.05 $\times 10^3/\mu L$, normocytic anemia with a hemoglobin of 10.0 g/dL, and thrombocytopenia with a platelet count of 77 $\times 10^3/\mu L$, together constituting pancytopenia in the setting of fever. Renal function was preserved (urea 29 mg/dL, serum creatinine 0.64 mg/dL) and random blood glucose was 127 mg/dL. Notable abnormalities included hyponatremia (sodium 126 mmol/L), hypokalemia (potassium 3.3 mmol/L), and hypoalbuminemia (albumin 2.7 g/dL), the last of which reflected the combined effect of reduced intake and systemic inflammation.

On the basis of the history, examination, and laboratory findings, the patient was diagnosed with recurrent left breast cancer (luminal B subtype) complicated by febrile neutropenia, pancytopenia, and moderate malnutrition. The surgical oncology team initiated supportive and infection-directed care comprising an intravenous fluid loading dose of

Ringer's lactate 500 mL followed by a maintenance balanced fluid infusion at 20 drops/min, intravenous paracetamol 1 g three times daily, intravenous ondansetron 4 mg three times daily, intravenous dexamethasone 10 mg three times daily, and a granulocyte colony-stimulating factor preparation once daily for two days to accelerate neutrophil recovery. Oral sucralfate syrup was given three times daily for gastrointestinal protection. To correct the cytopenias she received a transfusion of two units of packed red cells and platelet concentrate.

Table 1. Laboratory findings on admission.

Parameter	Result	Reference range	Interpretation
White blood cell count (×10³/μL)	**0.43**	**4.0–10.0**	**Leukopenia**
Absolute neutrophil count (×10³/μL)	**0.05**	**1.5–8.0**	**Severe neutropenia**
Hemoglobin (g/dL)	**10.0**	**12.0–16.0**	**Anemia**
Platelet count (×10³/μL)	**77**	**150–400**	**Thrombocytopenia**
Random blood glucose (mg/dL)	127	<140	Normal
Urea (mg/dL)	29	10–50	Normal
Serum creatinine (mg/dL)	0.64	0.6–1.1	Normal
Sodium (mmol/L)	**126**	**135–145**	**Hyponatremia**
Potassium (mmol/L)	**3.3**	**3.5–5.1**	**Hypokalemia**
Albumin (g/dL)	**2.7**	**3.5–5.2**	**Hypoalbuminemia**

Notes: Reference ranges reflect the reporting laboratory. Pancytopenia in the presence of fever (40.6 °C) constituted febrile neutropenia.

In parallel, the clinical nutrition service designed an individualized medical nutrition therapy plan, summarized in Table 2. The energy target was 2,100 kcal/day with a protein target of 90 g/day, a fat allocation of 47 g/day, and a carbohydrate allocation of 330 g/day, with a pragmatic short-term goal of achieving at least 80% of the prescription. Nutrition was delivered orally as a soft diet supplemented with a peptide-based oral nutritional supplement (Peptisol) at 200 kcal twice daily, fruit juice, and ginger water to mitigate nausea, together with intravenous vitamin B12 500 μg twice daily to support hematopoietic recovery. Oral intake improved steadily: on the first day of care the patient met approximately 60% of her target (1,260 kcal), rising to 86% (1,806 kcal) by the third day and to a maximum of 93% (1,953 kcal) by the fourth day, as also shown in Table 2.

Table 2. Individualized medical nutrition therapy prescription and daily energy intake achieved.

Component	Prescription/value
Macronutrient and energy prescription	
Energy target	2,100 kcal/day (~35 kcal/kg target body weight)
Protein	90 g/day (~1.5 g/kg target body weight)
Fat	47 g/day
Carbohydrate	330 g/day
Short-term intake goal	at least 80% of prescription
Route and composition	
Oral diet	Soft diet
Oral nutritional supplement	Peptide-based formula, 200 kcal twice daily
Adjuncts for symptom control	Fruit juice; ginger water
Micronutrient repletion	Vitamin B12 500 μg IV twice daily
Daily energy intake achieved	
Day 1	60% of target (1,260 kcal)
Day 3	86% of target (1,806 kcal)
Day 4	93% of target (1,953 kcal)

Notes: Target body weight referenced to a body mass index of 22 kg/m² for the patient's height (approximately 60 kg). IV, intravenous.

By the sixth day of admission the patient's clinical condition had stabilized and she was discharged. She was counseled to continue a high-calorie, high-protein diet at home, to continue the peptide-based oral nutritional supplement at 200 kcal twice daily, and to attend the outpatient nutrition clinic for follow-up; an oral vitamin B-complex preparation twice daily was also provided. She was advised to undertake regular moderate-intensity physical activity, defined as 50–75% of maximal heart rate, for three sessions per week of 10–60 minutes each, including resistance and strengthening exercises, in order to rebuild physical function and muscle mass.

3. Discussion

This case illustrates three intertwined clinical problems that are individually common but, in combination, demand deliberate integration of oncologic, hematologic, and nutritional care: a recurrent hormone-receptor-positive breast cancer, a profound post-chemotherapy hematologic complication in the form of febrile neutropenia with pancytopenia, and GLIM-defined moderate malnutrition. Each problem amplifies the others. The tumor and its treatment drive a catabolic, inflammatory state that erodes nutritional reserves; reduced intake and protein depletion impair immune competence and marrow recovery; and the resulting cytopenias prolong the period during which the patient is too symptomatic to eat. The central message of this report is that interrupting this cycle early, with an individualized and predominantly oral nutrition strategy, is both feasible and clinically rewarding even during the acute neutropenic nadir.

The malnutrition–cancer–myelosuppression triad

Cancer-associated malnutrition is not a single entity but the end result of converging mechanisms: anorexia of central origin, treatable nutrition-impact symptoms, malabsorption, and an inflammatory catabolic drive that accelerates loss of skeletal muscle.[3] In this patient all of these elements were present. The post-chemotherapy gastrointestinal toxicity, with nausea, vomiting, and diarrhea, produced a secondary reduction in oral intake superimposed on the primary anorexia, while the underlying recurrent malignancy and the acute febrile neutropenic episode together generated a substantial inflammatory burden, reflected in her hypoalbuminemia. Inflammation contributes to malnutrition both by suppressing appetite and reducing food intake and by deranging metabolism, and it is increasingly recognized as a prognostically important component of the malnutrition syndrome in its own right.[11]

The link between nutritional status and hematologic tolerance of chemotherapy is well supported. In patients with lymphoma receiving cytotoxic therapy, poor pretreatment nutritional indices and low skeletal-muscle mass have been associated with a higher incidence of treatment-related adverse events, including febrile neutropenia, and with shorter treatment duration and worse outcomes.[5,6] The same principle applies in breast cancer, where loss of muscle mass during neoadjuvant chemotherapy is frequent and has been shown to track with greater treatment toxicity.[7,8] Prospective and retrospective studies in breast cancer confirm that loss of skeletal muscle is common and carries adverse clinical implications across the disease trajectory.[7,8] This patient's combination of a low BMI, a reduced MUAC indicating depleted muscle, and a critically low neutrophil count exemplifies the clinical phenotype in which nutritional depletion and marrow failure reinforce one another, and in which nutritional rehabilitation becomes part of, rather than an adjunct to, the management of the hematologic complication.

The patient's hypoalbuminemia merits careful interpretation within this triad. Albumin is a negative acute-phase reactant: its concentration falls during systemic inflammation as hepatic synthesis is redirected toward acute-phase proteins and as capillary permeability increases, so a low albumin in an acutely unwell patient reflects inflammatory burden and illness severity as much as protein intake. It is therefore not a pure marker of nutritional status, and it should not be used in isolation to diagnose malnutrition; the GLIM framework deliberately relies on phenotypic and etiologic criteria rather than on

albumin alone.[12] Nonetheless, in this patient the convergence of a low albumin, a low BMI, reduced muscle mass, and reduced intake painted a coherent picture of combined inflammatory and intake-related malnutrition, and the albumin trend provided a useful, if non-specific, adjunct for tracking resolution of the acute inflammatory insult as recovery proceeded. Composite indices that incorporate albumin alongside lymphocyte count, such as the prognostic nutritional index, formalize this combined signal and carry prognostic value in breast cancer.[13]

Diagnosing malnutrition with the GLIM framework

Accurate diagnosis is the prerequisite for effective intervention. The GLIM framework provides a globally harmonized, two-step approach: nutritional risk is first identified by a validated screening tool, and the diagnosis is then established by the combination of at least one phenotypic criterion (non-volitional weight loss, low BMI, or reduced muscle mass) and at least one etiologic criterion (reduced food intake or assimilation, or disease burden and inflammation).[12] The phenotypic criteria additionally determine severity, distinguishing moderate from severe malnutrition. The full framework is summarized in Table 3.

Table 3. Global Leadership Initiative on Malnutrition (GLIM) diagnostic framework.

Category	Criterion	Description
Phenotypic	Weight loss	>5% within 6 months, or >10% beyond 6 months
	Low body mass index	<20 kg/m^2 if <70 years (Asia <18.5); <22 kg/m^2 if >=70 years (Asia <20)
	Reduced muscle mass	By anthropometry (mid-upper-arm or calf circumference) or by BIA, DEXA, CT, or MRI
Etiologic	Reduced intake or assimilation	<=50% of energy requirements >1 week, reduced intake >2 weeks, or chronic malabsorption
	Inflammation / disease burden	Acute (infection, trauma, sepsis) or chronic (cancer, chronic organ disease, autoimmune)
Diagnosis	Combination required	At least 1 phenotypic plus 1 etiologic criterion
Severity	Moderate malnutrition	Weight loss 5–10% (<=6 months) or 10–20% (>6 months); BMI mildly reduced; moderate muscle loss
	Severe malnutrition	Weight loss >10% (<=6 months) or >20% (>6 months); BMI markedly reduced; severe muscle loss

Notes: BIA, bioelectrical impedance analysis; DEXA, dual-energy X-ray absorptiometry; CT, computed tomography; MRI, magnetic resonance imaging. Adapted conceptually from the GLIM consensus.

In clinical practice the GLIM diagnosis is preceded by a validated screening step. Tools such as the Malnutrition Universal Screening Tool, the Nutritional Risk Screening 2002, or the Malnutrition Screening Tool identify patients at risk, who then proceed to full GLIM assessment. This two-step logic is efficient and scalable, allowing busy oncology services to triage every admission rapidly and reserve detailed assessment for those who screen positive. The present patient would have screened positive on any of these instruments by virtue of recent weight loss combined with markedly reduced intake, and the subsequent GLIM assessment simply confirmed and graded what screening had flagged. Embedding this sequence into routine oncology admission pathways is one of the most practical levers available for closing the persistent gap between the known prevalence of malnutrition and the proportion of patients who actually receive timely nutritional intervention.[2]

Applying this framework to the present patient, as detailed in Table 4, the diagnosis was unambiguous.

She satisfied three phenotypic criteria: non-volitional weight loss exceeding 5% within the preceding six months (a documented loss of approximately 5.8% over two months), a low BMI of 17.6 kg/m^2, and reduced muscle mass inferred from a MUAC of 23 cm. She also satisfied two etiologic criteria: a reduction in food intake to 50% or less of requirements sustained for more than two weeks, and a marked inflammatory burden arising both acutely from the febrile neutropenic episode and chronically from the underlying breast cancer. The presence of multiple phenotypic and etiologic criteria, with a BMI and weight-loss magnitude falling within the moderate bands, established a diagnosis of moderate malnutrition. The decision to grade her as moderate rather than severe was important clinically because it identified a window in which intervention could plausibly arrest further deterioration before severe malnutrition supervened.

Table 4. Application of GLIM criteria to the patient.

Category	GLIM criterion	Patient finding	Result
Phenotypic	Weight loss >5% in <=6 months	Weight loss ~5.8% over 2 months	Met
	Low BMI	BMI 17.6 kg/m^2	Met
	Reduced muscle mass	MUAC 23 cm indicating reduced muscle mass	Met
Etiologic	Reduced intake/assimilation	Intake <=50% for 2 weeks	Met
	Inflammation/disease burden	Acute febrile neutropenia; chronic breast cancer	Met
Diagnosis	1 phenotypic + 1 etiologic	3 phenotypic + 2 etiologic criteria	Malnutrition confirmed
Severity	Phenotypic grading	Weight loss and BMI within moderate bands	Moderate malnutrition

Notes: BMI, body mass index; MUAC, mid-upper-arm circumference.

The validity of the GLIM approach for prognostication in oncology is well established. GLIM-defined malnutrition has demonstrated predictive validity for adverse outcomes in solid tumors, including colorectal cancer, and has been validated and shown to be reliable even in critically ill patients with cancer.[14,15] Recent work indicates that the prognostic weight of GLIM is enhanced when phenotypic criteria are combined and when the inflammatory component is formally incorporated, both of which were features of this patient's presentation.[11,16] An important practical point concerns the assessment of muscle mass. Although bioelectrical impedance analysis, dual-energy X-ray absorptiometry, and cross-sectional imaging are the reference methods, anthropometric surrogates such as MUAC remain valuable where these tools are unavailable; comparative work has shown that both anthropometry-based and impedance-based muscle-mass indicators carry prognostic information within the GLIM construct.[17] In settings without ready access to body-composition technology, phase angle and other impedance-derived markers have prognostic value, but a carefully measured MUAC provides a pragmatic and reproducible bedside surrogate that was sufficient to support the diagnosis here.[18]

3.3. Rationale for early, individualized medical nutrition therapy

Guideline recommendations are unequivocal that nutrition therapy should begin before a patient becomes severely malnourished. The European Society for Clinical Nutrition and Metabolism (ESPEN) recommends nutritional intervention to improve oral intake in patients with cancer who can still eat but are malnourished or at risk, encompassing dietary counseling, management of nutrition-impact symptoms, and oral nutritional supplementation, and reserves enteral and then parenteral routes for situations in which oral intake remains inadequate.[3] Medical nutrition is formally indicated when intake is anticipated to fall below approximately 50% of requirements for more than a week, or between 50%

and 75% of requirements for more than two weeks, thresholds this patient had clearly crossed. Initiating therapy at the moderate, rather than severe, stage of malnutrition is consistent with the principle that earlier intervention preserves more functional reserve and is more likely to succeed.

ESPEN frames energy requirements for patients with cancer as broadly similar to those of healthy individuals, on the order of 25–30 kcal/kg/day, with the higher end of the range chosen for younger, underweight, actively treated patients and the lower end for older, bedbound, or obese patients.[3] Protein intake should generally exceed 1 g/kg/day and may be increased to 1.5 g/kg/day, with 1.2–1.5 g/kg/day recommended for older patients with chronic disease and up to 2 g/kg/day considered safe when renal function is normal. In this patient the prescription of 2,100 kcal and 90 g protein is best interpreted in relation to a target rather than actual body weight: referenced to a desirable weight of approximately 60 kg for her height, the energy prescription corresponds to roughly 35 kcal/kg/day and the protein prescription to approximately 1.5 g/kg/day, both of which sit appropriately at the higher, repletion-oriented end of the ESPEN ranges for an actively treated, underweight, catabolic patient with normal renal function. Framing the prescription this way avoids the trap of under-prescribing relative to a depleted current weight while remaining within evidence-based bounds.

The expected benefits of this approach are supported by interventional evidence. A randomized controlled trial showed that whole-course immunonutrition delivered during chemoradiotherapy significantly reduced nausea, vomiting, and severe oral mucositis while better preserving body weight and total protein, and a randomized trial in breast cancer specifically showed that a combined exercise-and-nutrition program improved chemotherapy completion, a direct marker of the capacity to tolerate planned therapy.[9,19] A randomized clinical trial in cancer-related cachexia demonstrated that a structured dietary intervention improved body composition, inflammatory markers, and nutritional status, reinforcing the rationale for treating malnutrition as an actively modifiable target rather than an inevitable accompaniment of advanced disease.[10]

A specific safety consideration when re-feeding a depleted, acutely ill patient is the risk of refeeding syndrome, a potentially dangerous intracellular shift of phosphate, potassium, and magnesium when carbohydrate intake resumes after a period of reduced intake, accompanied by fluid retention and thiamine depletion. This patient had already demonstrated electrolyte derangements on admission, with hyponatremia and hypokalemia, which heightened the importance of cautious initiation, electrolyte monitoring, and gradual escalation of intake rather than abrupt delivery of the full energy target. The observed trajectory, in which intake climbed progressively from 60% on day one to 86% by day three and 93% by day four, is consistent with a prudent, stepwise advancement that balances the imperative to replete against the hazard of overly rapid feeding. The ESPEN framework favors exactly this measured escalation, and the pragmatic short-term goal of at least 80% of requirements reflects the same philosophy.[3]

3.4. Choice of a peptide-based oral nutritional supplement and protein targeting

Oral nutritional supplements are recommended when intake from ordinary food is inadequate, and the evidence for protein-focused supplementation in patients receiving chemotherapy is growing. A randomized controlled trial in patients with cancer receiving chemotherapy found that adding oral nutritional supplementation to dietary guidance attenuated body-weight loss (2.51 versus 3.98 kg), better preserved body mass index, and markedly improved chemotherapy adherence (76.1% versus 58.9%), providing direct support for the protein-focused strategy adopted here.[20] In oncologic surgical populations, a multicenter randomized controlled trial of postoperative oral nutritional supplements reported favorable long-term effects, and observational data show that adherence to supplementation is a key

determinant of its benefit, emphasizing the importance of a palatable, well-tolerated product.[21,22]

The selection of a peptide-based formula in this patient was deliberate and well matched to her gastrointestinal intolerance. Because she presented with diarrhea, nausea, and vomiting, a semi-elemental, peptide-based, generally low-residue formula was preferable to a standard polymeric product: peptide-based diets are more readily digested and absorbed and leave less residue in the gastrointestinal tract. Real-world and cross-sectional analyses of peptide-based enteral formulas report favorable tolerance and a lower burden of feeding intolerance, which is precisely the characteristic required in a patient whose intake was being limited by gastrointestinal symptoms.[23,24] The whey-protein and calcium-caseinate content of such formulas also supplies high-biological-value protein that supports tissue repair, immune function, and the maintenance of serum albumin, all of which are relevant to a patient with hypoalbuminemia and impaired marrow recovery. Increasing protein delivery stimulates muscle protein synthesis, and a high-protein diet is therefore particularly important in malnourished patients requiring nutritional support, complementing the energy provided by the soft oral diet.

Adherence is the hinge on which the benefit of oral nutritional supplementation turns, and it cannot be assumed. Cross-sectional data in patients with cancer at nutritional risk show that real-world compliance with supplementation is frequently suboptimal and is shaped by palatability, symptom burden, taste fatigue, and the practicalities of timing supplements around meals and treatment.[22] Several strategies were available to support adherence in this patient: dividing the supplement into modest twice-daily servings rather than a single large volume; pairing nutrition with active symptom control using antiemetics and gastrointestinal protection; offering fruit juice and ginger water as palatable adjuncts to counter nausea; and framing the plan around an achievable 80% goal rather than an intimidating 100% target. These measures align with the ESPEN emphasis on counseling and symptom management as the foundation of nutritional care, and they help explain why intake rose steadily rather than stalling.[3] The lesson is that prescribing a supplement is only the first step; engineering the conditions for the patient to actually consume it is what converts a prescription into a clinical effect.

Monitoring the trajectory of the prognostic nutritional index and related albumin- and lymphocyte-based composite markers is a useful adjunct in this context, since these indices change predictably with effective nutritional and clinical recovery and have demonstrated prognostic value in breast cancer patients undergoing chemotherapy.[13] In the present case the practical surrogate for nutritional adequacy was the day-by-day proportion of the energy target achieved, supplemented by serial weight and albumin trends, which together provided actionable feedback at the bedside.

3.5. Micronutrient repletion and hematologic recovery

Beyond macronutrient provision, micronutrient status influences hematologic recovery after myelosuppressive chemotherapy. This patient received vitamin B12 to support hematopoiesis during the post-chemotherapy nadir. Vitamin B12 is a necessary cofactor for DNA synthesis in rapidly dividing cells, including hematopoietic precursors, and clinically significant deficiency can aggravate anemia, contribute to ineffective hematopoiesis, and delay recovery of blood counts. Repletion in a patient with profound pancytopenia is therefore a reasonable supportive measure within the broader ESPEN principle that micronutrient deficiencies should be identified and corrected as part of comprehensive nutritional care.[3] The concurrent administration of a granulocyte colony-stimulating factor and transfusion of red cells and platelets were the primary drivers of the rapid hematologic correction observed; the nutritional and micronutrient measures are best understood as complementary, creating the metabolic substrate and cofactors required for the marrow to respond. Disentangling the independent contribution of each component is not possible in a single case, and this is acknowledged as a limitation, but the integrated

strategy was associated with a prompt and complete recovery of all three cell lines.

Effective nutritional care also depends on disciplined, objective monitoring rather than impression alone. In this patient the most actionable bedside metric was the daily percentage of the energy target achieved, recorded prospectively and reviewed each day, which transformed a static prescription into a dynamic feedback loop. Complementary measures included serial body weight, the trend in serum albumin as the acute inflammatory insult resolved, electrolyte surveillance given the admission hyponatremia and hypokalemia and the attendant refeeding risk, and a structured account of functional status as she progressed from a bedbound state to sitting and standing. Where available, repeated measurement of composite nutritional-inflammatory indices and of body composition would have added further granularity, and incorporating such monitoring into routine practice would allow the response to nutrition therapy to be quantified rather than merely observed.[13,18]

A note of caution about micronutrient supplementation in oncology is warranted. While correcting documented or clinically suspected deficiencies is appropriate, routine high-dose supplementation with antioxidant vitamins during active cytotoxic treatment is not recommended, because of theoretical concerns that high-dose antioxidants could blunt the oxidative mechanisms through which some chemotherapeutic agents exert their effect. ESPEN advises providing micronutrients in amounts approximating recommended daily allowances and reserving higher doses for specific, identified deficiencies.[3] The vitamin B12 given to this patient is best understood within this targeted framework, as support for hematopoiesis during a period of profound marrow stress rather than as indiscriminate megadose supplementation, and the transition at discharge to a standard oral vitamin B-complex preparation is consistent with this measured approach.

3.6. Clinical response and the meaning of less than 100% of target

The patient's response over six days was clinically meaningful across nutritional, symptomatic, functional, and hematologic domains, as summarized in Table 5. Her body weight rose modestly from 48 kg to 49 kg and her BMI from 17.6 to 18.0 kg/m^2; while small, an early reversal of weight loss during an acute catabolic illness is a favorable signal. Energy intake improved from 50% or less of requirements to more than 80%, and the constellation of nausea, vomiting, diarrhea, and fever resolved, with a corresponding improvement in appetite. Physical function improved from a bedbound state to sitting and standing, an important patient-centered gain. Most strikingly, the hematologic indices recovered comprehensively: the white-cell count rose from 0.43 to 12.9 ×10^3/μL, hemoglobin from 10.0 to 12.2 g/dL, platelets from 77 to 292 ×10^3/μL, and the absolute neutrophil count from a critical 0.05 to 11.51 ×10^3/μL.

Table 5. Clinical and hematologic parameters before and after nutritional intervention.

Parameter	Before intervention	After intervention
Body weight (kg)	48	49
Body mass index (kg/m^2)	17.6	18.0
Energy intake	<=50% of requirement	>80% of requirement
Clinical symptoms	Weak, febrile, anorexia, nausea, vomiting, diarrhea	Well, afebrile, improved appetite, no nausea or vomiting, normal bowel habit
Physical function	Bedbound	Able to sit and stand
White blood cell count (×10^3/μL)	0.43	12.9
Hemoglobin (g/dL)	10.0	12.2
Platelet count (×10^3/μL)	77	292
Absolute neutrophil count (×10^3/μL)	0.05	11.51

Notes: Values recorded at first nutrition consultation and after six days of individualized medical nutrition therapy with concurrent supportive care.

An instructive feature of this case is that the patient never reached 100% of her prescribed energy target, achieving a maximum of 93% on day four, yet still experienced significant clinical improvement. This observation carries a practical lesson: the goal of medical nutrition therapy in the acutely ill patient is not the rigid attainment of a numerical target but the restoration of an adequate and improving intake trajectory that supports recovery. Setting a pragmatic short-term goal of at least 80% of requirements, as was done here, is realistic, avoids the risks of overfeeding in a previously depleted patient, and is compatible with excellent outcomes. The steady day-by-day increase from 60% to 86% to 93% likely mattered more than any single threshold, because it reflected resolving symptoms, returning appetite, and a tolerable, well-accepted feeding plan.

It is important to interpret the rapidity of the hematologic recovery with appropriate caution. Granulocyte colony-stimulating factor and blood-product transfusion are the established and most powerful interventions for febrile neutropenia and pancytopenia, and they undoubtedly accounted for much of the count recovery. The contribution of nutrition therapy is most plausibly understood as permissive and supportive: adequate energy, high-biological-value protein, and repleted micronutrients provide the conditions under which marrow recovery, immune reconstitution, and tissue repair can proceed efficiently. Framed this way, nutrition is neither a competing explanation nor a marginal add-on but an integral element of comprehensive supportive care during the nadir.

3.7. Survivorship, physical activity, and recurrence prevention

The discharge plan extended the nutritional intervention into survivorship. The patient was advised to maintain a high-calorie, high-protein intake, to continue the oral nutritional supplement, and to attend the outpatient nutrition clinic, alongside a structured recommendation for moderate-intensity physical activity. This emphasis is supported by trial evidence: a randomized study in women with breast cancer demonstrated that a combined exercise-and-nutrition program improved chemotherapy completion, and broader evidence indicates that maintaining a healthy body weight and an active lifestyle, together with a diet rich in vegetables, fruit, whole grains, and low in saturated fat and red meat, is associated with improved survival and a lower risk of recurrence after a breast cancer diagnosis.[9] Rebuilding skeletal muscle through combined resistance and aerobic activity directly counters the sarcopenia that is common during breast cancer treatment and that predicts worse outcomes.[7,8] Nutritional care, in other words, does not end at discharge; it transitions from acute repletion to long-term maintenance of nutritional status, muscle mass, and metabolic health.

These recommendations also align with the broader principle that nutritional status is a modifiable prognostic factor across the cancer continuum. Because GLIM-defined malnutrition predicts worse treatment outcomes and survival, routine nutritional screening of all patients with advanced cancer, followed by prompt assessment and intervention when risk is detected, is justified on both clinical and prognostic grounds.[2,4] Early detection of inadequate intake, weight loss, and low BMI allows metabolic and nutritional problems arising from the disease or its treatment to be addressed before they entrench, which is the central practical implication of this case.

3.8. Practical implications and a stepwise bedside framework

Translated into routine practice, this case supports a simple, reproducible sequence that can be applied at the bedside even where advanced body-composition technology is unavailable. First, screen every patient with cancer for nutritional risk at admission and at key treatment milestones using a validated tool. Second, in those who screen positive, establish and grade the diagnosis with the GLIM criteria, using carefully measured anthropometry such as mid-upper-arm circumference as a pragmatic surrogate for muscle mass when impedance or imaging is not at hand.[17] Third, set energy and protein targets referenced to a desirable body weight, placing actively treated, underweight patients at the higher, repletion-

oriented end of the ESPEN ranges. Fourth, deliver nutrition by the least invasive effective route, beginning with food and counseling, adding an oral nutritional supplement matched to gastrointestinal tolerance, and escalating to enteral or parenteral routes only if oral intake remains inadequate.[3,20]

Fifth, treat the symptoms that limit intake as aggressively as the nutritional deficit itself, because controlling nausea, vomiting, and diarrhea is often the rate-limiting step in restoring intake. Sixth, monitor objectively and frequently, tracking the daily proportion of the energy target achieved alongside weight, electrolytes, and albumin, and remaining alert to refeeding risk in the most depleted patients. Finally, extend the plan into survivorship with continued supplementation as needed, dietary counseling, and a structured physical-activity prescription to rebuild muscle and support long-term outcomes.[9] None of these steps requires resources beyond those of a typical hospital nutrition service, which is precisely why the approach is generalizable. This case demonstrates that the disciplined application of an inexpensive, predominantly oral strategy, delivered in concert with standard hematologic and infectious support, can accompany rapid and complete recovery even in a patient who began care during a profound neutropenic nadir.

3.9. Limitations

This report has the inherent limitations of a single-patient observation. The temporal association between the integrated supportive strategy and the patient's recovery cannot establish causation, and the relative contributions of nutrition therapy, granulocyte colony-stimulating factor, transfusion, and antimicrobial-supportive measures cannot be separated. Body composition was assessed by MUAC alone because bioelectrical impedance analysis and cross-sectional imaging were not available, so the quantification of muscle mass was necessarily approximate. Follow-up was limited to the acute admission and early discharge plan, and longer-term nutritional, functional, and oncologic outcomes were not captured. Finally, biochemical markers of inflammation such as C-reactive protein were not measured, which would have allowed a more precise characterization of the inflammatory component of the malnutrition syndrome. Despite these constraints, the case offers a concrete, reproducible illustration of how early individualized nutrition therapy can be embedded within acute oncologic care.

4. Conclusion

Breast cancer is the most common malignancy in women and a leading cause of cancer death, and patients with cancer are at high risk of malnutrition arising not only from the disease itself but also from the effects of anticancer therapy. This case demonstrates that early, individualized medical nutrition therapy, initiated before severe malnutrition developed and built on a protein-focused soft oral diet, a well-tolerated peptide-based oral nutritional supplement, and appropriate micronutrient repletion, was associated with concurrent improvement in nutritional status, symptoms, physical function, and hematologic recovery in a patient with recurrent luminal B breast cancer complicated by post-chemotherapy febrile neutropenia and pancytopenia. The patient improved meaningfully even though her daily intake never reached 100% of the prescribed target, underscoring that a steadily improving intake trajectory, rather than rigid attainment of a numerical goal, is what matters most during acute illness.

Because malnutrition is among the most prevalent and consequential comorbidities in oncology, routine nutritional screening of all patients with advanced cancer is recommended, with prompt assessment and intervention when inadequate intake, weight loss, or a low BMI is detected. Adequate nutritional support plays a crucial role in improving nutritional status, supporting immune function, reducing treatment-related complications, and enhancing both therapeutic response and survival. Patients should be encouraged to maintain regular moderate physical activity and an ideal body weight with a diet rich in vegetables, fruit, and whole grains and low in saturated fat, lifestyle factors that can reduce recurrence and improve long-term survival. Integrating nutrition as a core component of comprehensive cancer care, rather than as an

afterthought during acute complications, is both feasible and clinically valuable.

Declarations

Ethics approval and consent to participate

This case report was conducted in accordance with the principles of the Declaration of Helsinki. Written informed consent was obtained from the patient for the use and publication of her anonymized clinical information.

Consent for publication

Written informed consent for publication of this case report and any accompanying data was obtained from the patient. No identifying images or personal details are included in this report.

Availability of data and materials

The clinical data supporting the findings of this case report are included within the article. Further details are available from the corresponding author upon reasonable request, subject to protection of patient confidentiality.

Competing interests

The authors declare that they have no competing interests. Mention of a specific commercial nutritional product reflects the care actually delivered and does not constitute endorsement.

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Authors' contributions

Both authors contributed to the clinical management of the patient, the conception of the report, the drafting and critical revision of the manuscript, and approved the final version for submission.

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