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Impact of PG-SGA-assessed malnutrition stratification on clinical outcomes in advanced gastrointestinal malignancies: a retrospective cohort study

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Abstract

Introduction The traditional TNM staging system fails to explain survival heterogeneity in advanced gastrointestinal cancer, while malnutrition remains an underutilized prognostic modulator. We aimed to establish the prognostic value of dynamic nutritional risk stratification using the Patient-Generated Subjective Global Assessment (PG-SGA).

Methods In this retrospective cohort study, 102 patients with AJCC 8th edition stage II–IV gastrointestinal malignancies underwent serial PG-SGA assessments at admission and 3-month follow-up. Nutritional risk was stratified as low-risk (PG-SGA 3–8) or high-risk (PG-SGA ≥ 9). Associations with overall survival (OS) were analyzed via Kaplan-Meier curves and multivariable Cox regression.

Results Among 102 advanced gastrointestinal malignancies patients, 68.63% (70/102) were high-risk nutritional group at admission versus 31.37% low-risk, with lower BMI in high-risk group (19.13 vs. 21.52 kg/m^2, $P < 0.001$) but comparable inflammatory/nutritional markers ($P > 0.05$). By 3 months, high-risk prevalence rose to 77.45% (79/102), where low-risk patients showed superior weight (58.00 vs. 50.00 kg), BMI (20.55 vs. 18.92 kg/m^2), HB (120.13 vs. 100.91 g/L), ALB (41.15 vs. 34.33 g/L), PNI (47.12 vs. 39.72), CRP (1.63 vs. 11.54 mg/L), and dietary intake (all $P < 0.05$). Survival analysis confirmed 5-fold higher 3-year mortality risk in high-risk group (HR = 5.00, 95% CI: 2.00-12.47; $P < 0.001$). Multivariable analysis identified TNM stage IV (HR = 3.78, 95% CI: 1.25–11.42; $P = 0.018$), persistent high nutritional risk (HR = 4.09, 95% CI: 1.38–12.14; $P = 0.011$), and elevated CRP (HR = 2.32, 95% CI: 1.14–4.73; $P = 0.020$) as independent death predictors.

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Conclusion Longitudinal PG-SGA monitoring identifies critical nutritional deterioration threshold of PG-SGA ≥ 9 at 3-month follow-up and enables early intervention. We advocate integrating dynamic nutritional risk assessment into clinical frameworks to shift from empirical to predictive management in gastrointestinal oncology.

Keywords Gastrointestinal malignancies, Malnutrition, PG-SGA, Clinical outcomes

Introduction

Cancer persists as a leading cause of global mortality, making improved long-term survival a key therapeutic goal in oncology [1]. Despite the 8th edition American Joint Committee on Cancer (AJCC) TNM system remaining the gold standard for prognosis and treatment stratification, substantial outcome heterogeneity persists within equivalent staging categories [2]. Clinical evidence reveals that 35%–48% of stage III colorectal cancer patients exhibit survival disparities exceeding median 18 months, underscoring the imperative to identify actionable risk factors beyond conventional anatomical staging paradigms. This prognostic variability highlights an urgent need for novel risk prediction model based on routine clinical examinations that can effectively stratify patients' survival outcomes by quantifying temporal changes in tumor-immune dynamics, thereby enabling timely adjustment of therapeutic strategies [3].

Emerging evidence highlights malnutrition as a critical yet underutilized prognostic modulator in cancer management [4]. Globally, cancer-associated malnutrition affects 25%–70% of oncological patients, in a prospective observational study conducted in 22 medical oncology centers in Italy, 51.1% of cancer patients had malnutrition at their first visit [5]. This metabolic dysregulation establishes a vicious cycle: cachexia-induced treatment intolerance and immune dysfunction interact with poor dietary intake, accelerating functional decline [6].

Crucially, unlike static genetic mutations, nutritional status represents a dynamic therapeutic target. This modifiable nature offers a significant opportunity for clinical intervention. Randomized trials demonstrate that early nutritional intervention in malnourished gastric cancer patients yields a 23% improvement in 2-year survival rates [7].

The PG-SGA is the validated nutritional assessment tool specifically designed for cancer patients, endorsed by the European Society for Clinical Nutrition and Metabolism (ESPEN) [8]. By quantifying critical domains including weight trajectory, dietary intake, symptom burden, activities and physical functions, PG-SGA objectively assesses malnutrition risk [9, 10]. A meta-analysis of 19 studies encompassing diverse cancer types demonstrated that PG-SGA assessment significantly correlates with adverse outcomes [11]. Using established risk stratification criteria (low-risk: PG-SGA ≤ 3; intermediate-risk: 4–8; high-risk: >8), patients with intermediate/high malnutrition risk exhibited significantly worse overall survival (OS) versus low-risk counterparts (adjusted HR = 1.98, 95% CI: 1.77–2.21). Subgroup analysis of gastrointestinal cancers confirmed increased mortality risk (pooled HR = 1.61, 95% CI: 1.39–1.87). However, the limited number of gastrointestinal cancer studies (n = 7) necessitates further investigation to elucidate the prognostic significance of PG-SGA-defined malnutrition for optimizing therapeutic outcomes in this population.

This study assesses nutritional trajectories stratified by PG-SGA in advanced gastrointestinal cancer patients, analyzing associations between dynamic nutritional risk and survival outcomes. Using collected nutritional parameters (body mass index, BMI; serum albumin, ALB; C-reactive protein, CRP; prognostic nutritional index, PNI) at admission and 3-month intervals, we employed multivariate Cox models to identify independent prognostic impacts of 3-month nutritional deterioration (particularly progression to PG-SGA high-risk status) on OS. We developed a prognostic model integrating nutritional risk evolution and routine clinical examinations, offering evidence-based guidance for personalized nutrition strategies in oncology care. This study establishes a paradigm for longitudinal nutritional monitoring and targeted dietary interventions in malignancy management, aiming to delineate optimal timing strategies and evidence-based nutritional support to improve survival outcomes in gastrointestinal cancer.

Methods

Research subjects

A retrospective cohort study was conducted on patients with gastrointestinal malignancies hospitalized in the Department of Medical Oncology at Jiaxing Second Hospital between March 2021 and June 2022. The study protocol strictly adhered to international research ethics standards and was implemented in full compliance with the principles outlined in the Declaration of Helsinki (2013 revision). This study was ratified by the Ethics Committee of Jiaxing Second Hospital (Approval No. 2023JX111-01) and adheres to STROBE guidelines. This retrospective study utilized anonymized data from existing medical records. According to national regulations and the committee's policy, this type of study qualifies for a waiver of informed consent.

Inclusion and exclusion criteria

Inclusion criteria

1. Age ≥ 18 years;
2. Patients with pathologically confirmed gastrointestinal malignancies;
3. Participants presenting with advanced gastrointestinal malignancy according to the 8th edition of the AJCC staging system;
4. The available PG-SGA documentation;
5. Actual observed survival time urvival duration exceeding 3 months.

Exclusion criteria

1. Insufficient clinical data;
2. Severe organ dysfunction;
3. Communication barriers or psychiatric history;
4. Specific religious beliefs or vegetarian/restrictive dietary patterns;
5. Any other medical conditions considered by investigators to potentially compromise study validity or participant safety.

A total of 102 patients met all inclusion criteria and were enrolled in this retrospective cohort study; notably, none had an admission PG-SGA score ≤ 3.

Research method

The study initially enrolled 181 patients, of whom 79 were subsequently excluded based on predefined inclusion and exclusion criteria. The exclusion cohort comprised 3 cases (1.7%) failing to meet enrollment requirements, and 76 cases (41.4%) lost to follow-up resulting from discontinued clinical visits, hospital transfers, or treatment discontinuation. Ultimately, 102 eligible patients were included for statistical analysis. Following the research of Mao et al.'s [12], participants were stratified into two nutritional risk groups using the PG-SGA tool: a low-risk cohort (3 ≤ PG-SGA ≤ 8) and a high-risk cohort (PG-SGA ≥ 9). The complete participant flow diagram is presented in Fig. 1.

Clinical treatment

All treatment protocols adhering to the standardized guidelines outlined in the Clinical Practice Guidelines for Common Digestive System Tumors (2021) by the Chinese Society of Clinical Oncology (CSCO). The management of advanced gastrointestinal malignancies primarily focuses on palliative and systemic therapies, including chemotherapy, targeted therapy, and immunotherapy. The specific treatment regimen is physician-determined based on tumor type and must adhere to evidence-based clinical practice guidelines, particularly those from the National Comprehensive Cancer Network (NCCN) or the CSCO.

Nutritional intervention

Clinical dietitians dynamically adjust patients' nutritional regimens based on disease progression.

All inpatients underwent routine nutritional risk screening at admission using the PG-SGA. A score ≥ 4 typically triggered dietitian referral and intervention. Dietary intake was assessed by trained staff using the 24-hour dietary recall method, with data analyzed using the [WINCOME Nutrition Management System V2.0 (Shanghai WinCome Computer Science And Technology Co., Ltd.). Recommendations encompassed dietary counseling, food fortification, and/or oral nutritional supplements (ONS). If energy intake remained below 60% of the target for 3–5 days, ONS was provided at 120 g/day (≈ 540 kcal). According to the ESPEN practical guidelines, the recommended energy intake for patients was 25–30 kcal/(kg·d), with a protein intake of 1.2–1.5 g/(kg·d) [13]. Protein sources were prioritized from high-quality options such as livestock, poultry, aquatic products, eggs, dairy, and soy products. Dietary planning followed the Chinese Nutrition Society's recommended food exchange portion method (Supplementary Table S1). Outpatients received nutritional advice during their oncology clinic visits.

Data collection and definitions

Patient data were collected across the following categories: demographic characteristics, disease-specific indicators, hematological parameters, dietary intake metrics, nutritional assessment scales, and clinical outcomes.

Demographic characteristics included age, sex, height, weight, and BMI. Disease-specific indicators comprised TNM stage, tumor location, and health insurance type. Hematological parameters included ALB, hemoglobin (HB), CRP, and the PNI. These parameters were selected to provide an objective biochemical profile that complements the patient-reported and functional aspects of the PG-SGA. ALB, HB and PNI reflect visceral protein stores and systemic inflammation, while CRP is a direct marker of the inflammatory response, which is a key driver of cancer-associated malnutrition and cachexia [14–17]. Dietary intake metrics encompassed energy and protein consumption levels. Clinical outcomes were evaluated using 3-year OS, defined as the duration from the date of initial hospital admission to death from any cause or the end of follow-up. The 3-month follow-up was aligned with key treatment milestones, allowing for the detection of clinically meaningful nutritional changes and facilitating timely clinical interventions. Hematological parameters, dietary intake metrics, and nutritional assessment

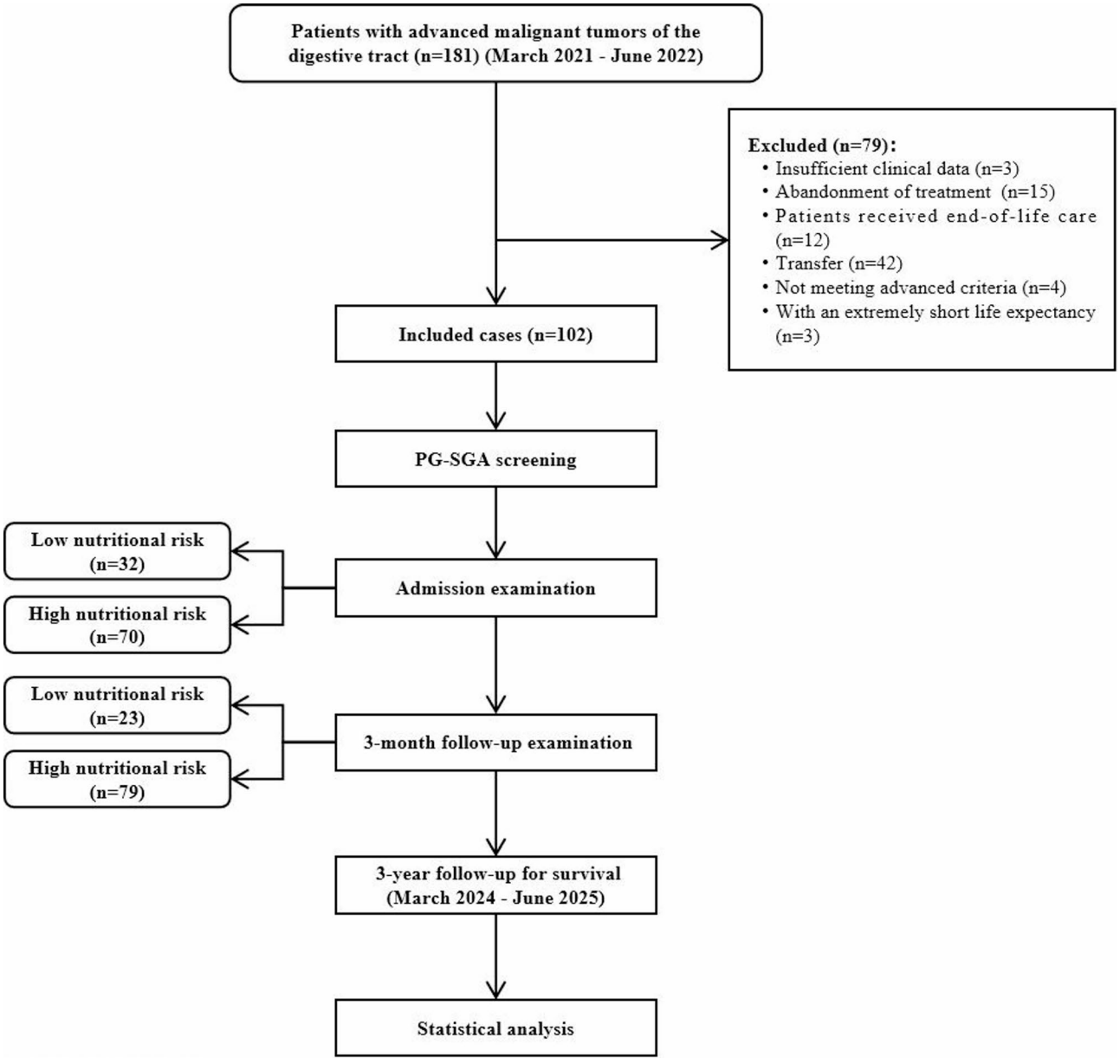

Fig. 1 Flow chart of the study

scales were collected both at admission (enrollment) and at the 3-month follow-up [18].

BMI was calculated as weight (kg) divided by height squared (m^2). PNI was computed using the formula: PNI = 10 × ALB [g/dL] + 0.005 × total lymphocyte count/mm^3 [19]. Energy intake ratio (%) was determined by dividing actual energy intake by guideline-recommended energy intake (25 kcal/kg/day) and multiplying by 100. Similarly, protein intake ratio (%) was calculated as actual protein intake divided by guideline-recommended protein intake (1.2 g/kg/day) multiplied by 100.

Statistical analysis

Statistical analyses were performed using R software (version 4.4.2). A complete-case analysis approach was employed. Categorical data are presented as frequencies (proportions) and compared using the χ^2 test. The Shapiro-Wilk test assessed normality of continuous variables. Data are expressed as mean ± standard deviation (normally distributed) or median [interquartile range] (non-normally distributed), compared with Student's t-test or Mann-Whitney U test, respectively.

Survival analyses included Kaplan-Meier curves with log-rank testing. Univariable and multivariable Cox proportional hazards models were used to identify

prognostic factors. The proportional hazards assumption was verified using Schoenfeld residual tests. A post-hoc power analysis was performed with R, confirming adequate power (> 80%) to detect the observed effect size given the sample size (n = 102) and event rate. A two-tailed p-value < 0.05 defined statistical significance.

Results

Participants characteristics

This study enrolled 102 patients with a median age of 68.50 years (interquartile range, IQR 61.00–73.75), of whom 65.69% (67/102) were male. Median weight at admission was 54.00 kg (IQR 46.25–60.00) with a corresponding BMI of 19.55 kg/m² (IQR 17.78–22.45). At 3-month follow-up, median weight was 52.00 kg (IQR 48.00–60.00) while median BMI decreased to 19.53 kg/m² (IQR 17.73–21.58).

TNM staging at admission was predominantly stage IV (57.84%, 59/102), followed by stage III (28.43%, 29/102) and stage II (13.73%, 14/102). At admission, PG-SGA assessment identified high malnutrition risk in 68.63% (70/102) of patients, a proportion that increased to 77.45% (79/102) at the 3-month follow-up. Furthermore, additional analysis revealed that despite receiving routine nutritional care, the mean PG-SGA score for the entire cohort increased significantly from 10.06 ± 3.38 to 11.21 ± 3.56 (mean difference: 1.15 ± 3.73, P = 0.002). Insurance types were evenly distributed (Employee Basic Medical Insurance: 50.98% vs. Resident Basic Medical Insurance: 49.02%). Upper gastrointestinal tumors accounted for 70.59% (72/102) of cases. Detailed results are presented in Table 1.

Compared to patients excluded due to loss of follow-up (n = 76), the analyzed cohort (n = 102) had a higher proportion of upper gastrointestinal tract cancers (70.59% vs. 3.95%, P = 0.023). No other significant differences in baseline characteristics were observed (Supplementary Table S2).

Table 1 Characteristics of participants in the study

Characteristics	
Age (years), (IQR)	68.50 (61.00, 73.75)
Gender, n (%)	
Male	67 (65.69)
Female	35 (34.31)
Height (m), (IQR)	1.65 (1.56, 1.70)
TNM, n (%)	
II	14 (13.73)
III	29 (28.43)
IV	59 (57.84)
Type of medical insurance, n (%)	
General residents' health insurance	50 (49.02)
Employee medical insurance	52 (50.98)
Digestive Tract Tumors, n (%)	
Upper	72 (70.59)
Lower	30 (29.41)
Weight at admission (kg), (IQR)	54.00 (46.25, 60.00)
Weight at 3-month follow-up (kg), (IQR)	52.00 (48.00, 60.00)
BMI at admission (kg/m^2), (IQR)	19.55 (17.78, 22.45)
BMI at 3-month follow-up (kg/m^2), (IQR)	19.53 (17.73, 21.58)
PG-SGA score at admission	10.06 ± 3.38
PG-SGA subgroup at admission, n (%)	
Nutritional low-risk group	32 (31.37)
Nutritional high-risk group	70 (68.63)
PG-SGA score at 3-month	11.21 ± 3.56
PG-SGA subgroup 3-month follow-up, n (%)	
Nutritional low-risk group	23 (22.55)
Nutritional high-risk group	79 (77.45)
Comorbidity burden, n (%)	
None	18 (17.65)
Other Malignancies only	10 (9.80)
Other chronic diseases only	72 (70.59)
Both	2 (1.96)

Categorical data are represented by n (%), non-normally distributed data were reported as IQR (interquartile range) [M (P25, P75)]

BMI Body mass index, *PG-SGA* Patient-generated subjective global assessment, *Low-risk* 3 ≤ PG-SGA ≤8, *High-risk* PG-SGA ≥9

Serial biomarker divergence by nutritional risk between admission and 3-month follow-up

Comparison of laboratory parameters between baseline and the 3-month follow-up demonstrated a significant associated with improvement in nutritional markers and a reduction in inflammatory indicators (n = 102). At admission, the low-risk group demonstrated significantly higher weight (median 58.15 vs. 50.50 kg, Z=−2.86, P = 0.004) and BMI (median 21.52 vs. 19.13 kg/m², Z=−3.34, P < 0.001) compared to the high-risk group. No significant intergroup differences were observed in HB, ALB, PNI, or CRP levels (all P > 0.05).

Patients were re-stratified based on PG-SGA scores at the 3-month follow-up, revealing statistically significant differences between the two groups. The low-risk group maintained superior weight (58.00 vs. 50.00 kg, Z=−2.90, P = 0.004) and BMI (20.55 vs. 18.92 kg/m², Z=−3.13, P = 0.002). Significant associated with improvements were observed in HB (120.13 vs. 100.91 g/L; t = 4.86, P < 0.001), ALB (41.15 vs. 34.33 g/L; t = 7.44, P < 0.001), and PNI (47.12 vs. 39.72; t = 6.29, P < 0.001) in the low-risk group, accompanied by substantially lower CRP levels (median 1.63 vs. 11.54 mg/L, Z=−3.24, P = 0.001), indicating reduced systemic inflammation. See Table 2 for comprehensive statistical details.

These results indicate that admission differences were limited to anthropometric measures, while the high-risk group exhibited progressive deterioration across multiple nutritional and inflammatory markers during follow-up. The data underscore the clinical importance of targeted

Table 2 Analysis of laboratory parameters in patient subgroups at different time points

A. Comparison of relevant blood nutritional indicators between low-risk and high-risk patient groups at baseline

Variables	Low-risk group (*n* = 32)	High-risk group(*n* = 70)	t/Z-value	Effect Size	*P*-value
Weight (kg), IQR	58.15 (52.50–63.50)	50.50 (43.62–58.83)	−2.86	0.28	0.004
BMI (kg/m^2), IQR	21.52 (19.04–23.39)	19.13 (17.59–20.76)	−3.34	**0.33**	< 0.001
HB (g/L)	105.43 ± 27.52	107.09 ± 23.25	−0.32	−0.07	0.753
ALB (g/L)	37.49 ± 3.93	36.83 ± 5.50	0.61	0.14	0.542
PNI	44.24 ± 8.36	42.77 ± 7.00	0.92	0.19	0.359
CRP (mg/L), IQR	5.63 (2.54–16.66)	16.22 (1.35–44.97)	−1.52	0.15	0.129

B. Comparison of relevant blood nutritional indicators between low-risk and high-risk patient groups at the 3-month follow-up

Variables	Low-risk group (n=23)	High-risk group（n=79）	t / Z-value	Effect Size	*P*-value
Weight (kg), IQR	58.00 (52.50-63.0)	50.00 (45.1-59.25)	-2.90	0.29	0.004
BMI (kg/m^2), IQR	20.55 (19.42-23.73)	18.92 (17.38-20.81)	-3.13	0.31	0.002
HB (g/L)	120.13 ± 17.34	100.91 ± 16.49	4.86	**1.14**	<0.001
ALB (g/L)	41.15 ± 3.27	34.33 ± 5.44	7.44	**1.52**	<0.001
PNI	47.12 ± 4.42	39.72 ± 6.52	6.29	**1.33**	<0.001
CRP (mg/L), IQR	1.63 (0.80-4.85)	11.54 (1.37-63.81)	-3.24	0.32	0.001

Normally quantitative data are presented as mean ± standard deviation (SD), non-normally distributed data were reported as IQR (interquartile range) [M (P25, P75)], categorical data are represented by n (%), effect sizes are reported as rank-biserial correlation (r) or Cohen's d. Bold values indicate statistically significant results with a *P*-value < 0.05.

BMI Body mass index, *HB* Serum hemoglobin, *ALB* Serum albumin, *PNI* Prognostic nutritional index, *CRP* C-reactive protein, *PG-SGA* Patient-generated subjective global assessment, *low-risk* 3 ≤ PG-SGA ≤8, *High-risk* PG-SGA ≥9

Reference Range: HB, 130-175 (g/L); ALB 40-55 (g/L); CRP, 0-8 (mg/L)

nutritional interventions and systematic monitoring for high-risk patients.

Comparison of dietary intake by nutritional risk between admission and 3-month follow-up

Table 3 presents a comparison of dietary intake between patients in different nutritional risk groups at initial hospitalization and 3-month follow-up. Admission data revealed no significant differences ($P > 0.05$) between low-risk and high-risk groups in total energy intake, energy intake ratio, protein intake, or protein intake ratio, indicating comparable initial nutritional status between groups. At 3-month follow-up, the re-stratified based on PG-SGA of low-risk group demonstrated significantly higher total energy intake (1495.83 ± 301.21 vs. 1345.11 ± 309.95 kcal/d; t = 2.06, $P = 0.042$), protein intake (57.52 ± 10.18 vs. 46.64 ± 12.51 g/d, t = 3.82, $P < 0.001$), and protein intake ratio (84.10 ± 14.92 vs. 73.71 ± 20.77% DRI; t = 2.23, $P = 0.028$) compared to the high-risk group. No significant difference was observed in energy intake ratio ($P = 0.979$), suggesting persistent nutritional inadequacy in high-risk patients. These findings underscore the necessity for enhanced nutritional interventions in high-risk populations and emphasize the importance of monitoring longitudinal dietary intake patterns.

Table 3 Comparison of dietary intake across patient groups at different time points

A. Comparison of energy and protein intake metrics between patients in low-risk and high-risk groups at baseline

Variables	Low-risk group (*n* = 32)	High-risk group(*n* = 70)	t-value	Effect Size	*P*-value
Total energy (kcal/d)	1199.72 ± 292.86	1151.60 ± 387.92	0.62	−0.15	0.534
Energy intake ratio (%DRI)	71.89 ± 16.42	75.09 ± 25.87	−0.75	0.14	0.452
Protein (g/d)	41.72 ± 15.69	40.66 ± 20.23	0.26	0.06	0.794
Protein intake ratio (%DRI)	62.79 ± 24.12	62.27 ± 31.99	0.08	0.02	0.935

B. Comparison of energy and protein intake levels between low-risk and high-risk patient groups at the 3-month follow-up

Variables	Low-risk group (n=23)	High-risk group（n=79	t -value	Effect Size	*P*-value
Total energy (kcal/d)	1495.83 ± 301.21	1345.11 ± 309.95	2.06	**0.49**	0.042
Energy intake ratio (%DRI)	86.90 ± 13.79	86.79 ± 19.72	0.03	0.01	0.979
Protein (g/d)	57.52 ± 10.18	46.64 ± 12.51	3.82	**0.57**	<0.001
Protein intake ratio (%DRI)	84.10 ± 14.92	73.71 ± 20.77	2.23	**0.95**	0.028

Normally quantitative data are presented as mean ± standard deviation (SD), effect sizes are reported as rank-biserial correlation (r) or Cohen's d. Bold values indicate statistically significant results with a *P*-value < 0.05.

PG-SGA Patient-generated subjective global assessment, *Low-risk* 3 ≤ PG-SGA ≤8, *High-risk* PG-SGA ≥9

Survival analysis at distinct time points

Among 102 patients followed for 3 years from admission, median OS was significantly shorter in the high-risk nutritional group (11.00 months, 95% CI: 8.00–19.00)

versus the low-risk group (21.00 months) (Fig. 2A). Despite this clinically meaningful disparity, initial survival curves at admission showed no significant intergroup difference (HR = 1.40, 95% CI: 0.79–2.46; P = 0.234; Fig. 2A). Nevertheless, it likely signifies greater vulnerability to treatment toxicity, complications, and poorer quality of life. Subsequent landmark analysis initiated at 3-month follow-up demonstrated a significant survival divergence: the high-risk cohort had a 3-year mortality hazard 5.00 times greater than the low-risk group (95% CI: 2.00–12.47; $P < 0.001$; Fig. 2B), indicating dynamic risk evolution.

Analysis of prognostic factors at different time points

Multivariable regression analysis of 102 patients (Table 4) revealed several significant predictors of overall survival. Most notably, high-risk PG-SGA status at 3-month follow-up was strongly associated with reduced survival (adjusted HR = 4.09, 95% CI: 1.38–12.14; P = 0.011), indicating a greater than 4-fold increase in mortality risk that underscores the critical importance of nutritional status assessment during treatment. Stage IV disease (adjusted HR = 3.98, P = 0.009) and elevated CRP at admission (adjusted HR = 3.01, P = 0.002) also significantly increased mortality risk. Comorbidity burden, particularly the presence of multiple conditions, further predicted poor outcomes (admission HR = 8.36, P = 0.037; 3-month HR = 9.29, P = 0.035). Other nutritional indices including PNI, ALB, and PA were not significantly associated with survival in the adjusted models. A post-hoc power analysis indicated that the study had approximately 91.33% power to detect the observed hazard ratio (HR = 4.09) between the nutritional risk groups, suggesting sufficient power to identify this large effect size at the 3-month follow-up. However, the power to detect smaller effects, such as the non-significant association observed at admission, was limited. Furthermore, the wide confidence intervals for some hazard ratios reflect the constraints of the sample size and the resultant imprecision in these estimates, which should be taken into account when interpreting the findings.

Discussion

Gastrointestinal malignancies are a major cause of cancer mortality. In addition to conventional treatments, nutritional status is a key independent prognostic factor [20]. Despite this recognition, notable disparities persist in clinical nutrition management quality compared to developed countries, primarily manifested in three critical domains: (1) inadequate integration of standardized nutritional risk screening into routine oncology care pathways [21]; (2) delayed implementation of key period personalized nutritional intervention [22]; and (3) absence of institutionalized multidisciplinary nutrition support models [23]. These systemic limitations frequently result in missed opportunities for evidence-based nutritional optimization during crucial phases of antitumor therapy, potentially exacerbating malnutrition-related complications and survival outcome disparities.

This study employed the PG-SGA for its specificity to cancer patients and its comprehensive inclusion of patient-generated symptoms. Although other validated

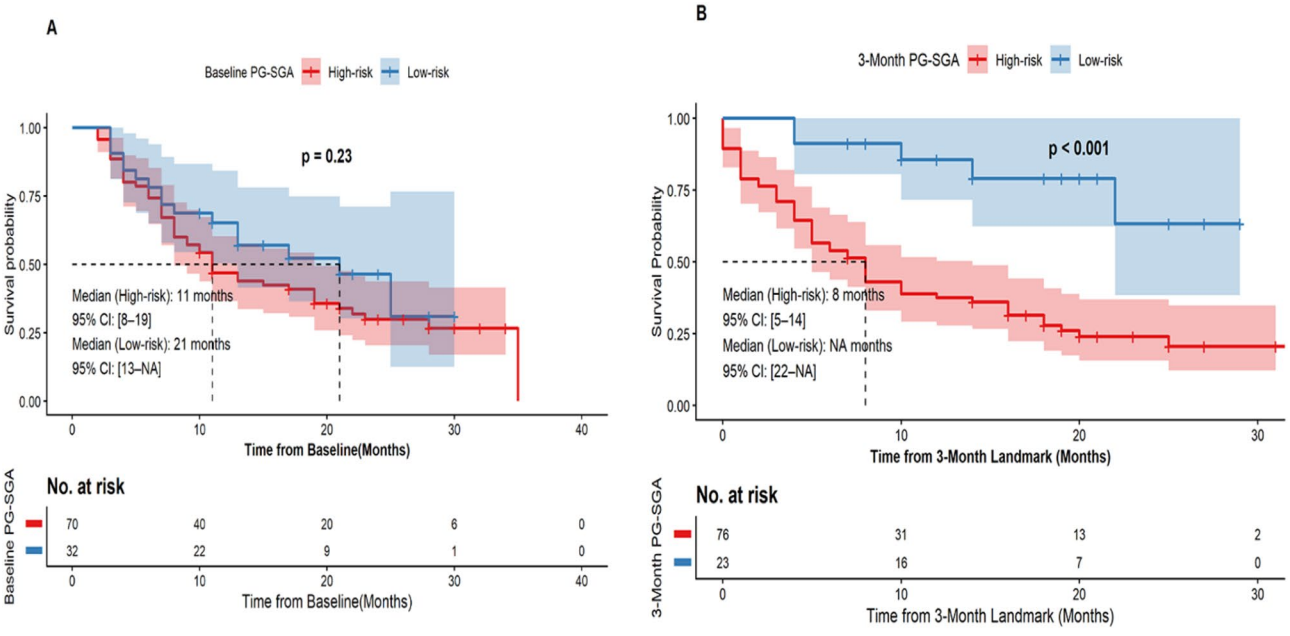

Fig. 2 Kaplan-meier survival analysis stratified by nutritional risk at different time points. **A** At admission survival curves; (**B**) At 3-month follow-up survival curves. Note: PG-SGA, patient-generated subjective global assessment; low-risk, 3 ≤ PG-SGA ≤ 8; high-risk, PG-SGA ≥ 9

Table 4 Multivariable cox regression results for overall survival

Variables	Admission			3-month follow-up		
	Univariate analysis	Multivariate analysis		Univariate analysis	Multivariate analysis	
	P-value	HR (95% CI)	*P*-value	*P*-value	HR (95% CI)	*P*-value
Age (year)	0.021	1.00 (0.97, 1.03)	0.287	0.021	0.98 (0.95, 1.01)	0.271
Gender (Male vs. Female)	0.477			0.477		
TNM (Ref: Ⅱ)						
Ⅲ	0.246			0.246		
Ⅳ	0.003	3.98 (1.41, 11.28)	**0.009**	0.003	3.78 (1.25, 11.42)	**0.018**
Weight	0.198			0.467		
BMI	0.212			0.492		
PG-SGA (Ref: Low-Risk) High-Risk	0.238			0.000	4.09 (1.38, 12.14)	**0.011**
Tumor Location (Ref: Lower) Upper	0.522			0.522		
PNI (< 45 vs.≥45)	0.005	0.69 (0.33, 1.42)	0.307	0.000	0.55 (0.24, 1.26)	0.157
ALB (< 40 vs.≥40)	0.015	0.82 (0.36, 1.89)	0.645	0.000	0.55 (0.18, 1.65)	0.285
CRP (< 8 vs.≥8)	< 0.001	3.01 (1.51, 6.02)	**0.002**	< 0.001	2.32 (1.14, 4.73)	**0.020**
HB (< 120 vs.≥120)	0.778			0.036	1.71 (0.77, 3.83)	0.191
PA (< 150 vs.≥150)	0.000	0.64 (0.31, 1.31)	0.221	< 0.001	0.67 (0.32, 1.38)	0.278
Comorbidity burden (Ref: None)						
Other Malignancies only	0.108			0.108		
Other chronic diseases only	0.003	2.95 (1.04, 8.36)	**0.041**	0.003	3.09 (1.12, 8.52)	**0.029**
Both	0.038	8.36 (1.14, 61.40)	**0.037**	0.038	9.29 (1.17, 73.55)	**0.035**
Pseudo R-squared		0.416			0.510	
AIC		502.6			484.6	

BMI Body mass index, *PG-SGA* Patient-generated subjective global assessment, *Low-risk* 3 ≤ PG-SGA ≤ 8, *High-risk* PG-SGA ≥ 9, *PNI* Prognostic nutritional index, *ALB* Serum albumin, *CRP* C-reactive protein, *HB* Serum hemoglobin, *PA* Prealbumin. Bold values indicate statistically significant results with a *P*-value < 0.05.

Reference Range: HB, 130–175 (g/L); ALB 40–55 (g/L); CRP, 0–8 (mg/L); PA, 250–400 (mg/L)

tools such as the NRS-2002 and GLIM criteria are also commonly used in oncology, the PG-SGA is particularly suited for longitudinal nutritional monitoring. Currently, there is no universally accepted gold standard for nutritional assessment in cancer care. As a cancer-specific tool, the PG-SGA integrates both subjective patient-reported outcomes and objective clinical measures, and is recommended by major professional societies including the American Dietetic Association, CSPEN, CSCO, and CACA [24]. Importantly, in addition to conventional nutritional indicators such as BMI and ALB levels, CRP has emerged as a clinically significant biomarker that bridges inflammatory status and nutritional deterioration [25]. Chronic inflammation within the tumor microenvironment contributes to malnutrition through dual mechanisms of enhanced catabolic processes and suppressed protein synthesis [26]. Elevated CRP levels not only indicate systemic inflammatory activation but also exhibit strong correlations with nutritional decline manifestations, particularly muscle wasting and hypoalbuminemia [27]. Our findings of severe malnutrition prevalence (PG-SGA score ≥ 9 in 68.63% of cases) aligns with previous reports. International studies report variable malnutrition rates ranging from 25% to 85% among cancer populations, while Chinese epidemiological data consistently demonstrate malnutrition prevalence exceeding 50% in oncology patients.

This study investigated the association between PG-SGA-assessed nutritional status and clinical outcomes in patients with advanced gastrointestinal malignancies. Longitudinal analysis from admission to the 3-month follow-up demonstrated significantly lower admission body weight (Z=−2.86, $P = 0.004$) and BMI (Z=−3.34, $P < 0.001$) in the high-risk nutritional group compared to the other. These intergroup differences in weight and BMI remained statistically significant at the 3-month follow-up ($P < 0.05$). Evaluation at 3 months further revealed a marked decline in ALB levels ($t = 7.44$, $P < 0.001$) coupled with elevated CRP concentrations in the high-risk group (Z=−3.24, $P = 0.001$), suggesting a potential temporal interaction between deteriorating nutritional status and enhanced systemic inflammatory responses during anticancer therapy.

Additionally, longitudinal evaluation of gastrointestinal cancer patients demonstrated a significant association between PG-SGA-defined nutritional status and survival outcomes. Patients maintaining high nutritional risk (PG-SGA ≥ 9) at 3-month follow-up exhibited markedly reduced median OS (10 months vs. counterparts, $P <$ 0.001), corroborating Bauer et al.'s proposed mechanism

of nutrition-mediated metabolic dysregulation [28]. The markedly higher mortality risk (HR = 5.00) associated with nutritional risk (PG-SGA ≥ 9) at 3 months in our study compared to prior meta-analyses (HR ≈ 1.98) may stem from our focus on advanced gastrointestinal cancer, the use of dynamic rather than baseline assessment, and differences in PG-SGA cut-offs or statistical adjustment [29]. Multivariate analysis revealed progressive elevation in mortality risk for admission TNM stage IV patients during follow-up (admission HR = 2.66, 95% CI:0.99–7.15; P = 0.053; 3-month follow-up HR = 3.78, 95% CI:1.25–11.42; P = 0.018), indicating cumulative interactions between disease advancement and nutritional depletion. admission CRP elevation independently predicted inferior survival (HR = 3.01, 95% CI:1.51–6.02; P = 0.002), supporting Oh et al.'s inflammation-nutrition axis hypothesis [30]. Serial PG-SGA assessments demonstrated superior prognostic discrimination at 3-month follow-up (HR = 4.09, 95% CI:1.38–12.14; P = 0.011), with survival differences predominantly manifesting within 12 months post-assessment (P < 0.001). These observations imply that treatment-induced metabolic stressors (mucosal toxicity, anorexia) may potentiate nutritional decline, while PG-SGA's temporal resolution enhances risk stratification during therapeutic phases. Clinically, this underscores the necessity for multidimensional nutritional management entails proactive screening at diagnosis, serial monitoring during treatment, and targeted protein supplementation [31].

Malnutrition adversely impacts clinical outcomes in cancer patients through distinct yet interrelated pathophysiological mechanisms, as supported by the findings of this study. Protein deficiency-induced sarcopenia substantially compromises tolerance to anticancer therapies, frequently necessitating dose modifications due to heightened treatment toxicity and subsequent interruptions, thereby attenuating cumulative treatment efficacy [32]. Our results directly demonstrate the central role of systemic inflammation, as elevated CRP was a strong independent predictor of mortality (HR = 2.83, 95% CI: 1.42–5.63; P = 0.003). Furthermore, protein-energy malnutrition compromises biosynthetic capacity, evidenced by hypoalbuminemia and a reduced PNI in our high-risk group, which collectively impair immunity and therapeutic responsiveness [33]. These pathophysiological mechanisms, nutritional depletion, sarcopenia, and inflammation, were all observed in our cohort and corroborate Cederholm's inflammation-malnutrition-cachexia axis theory, illustrating the self-perpetuating cycle between chronic inflammation and metabolic dysregulation [34]. Therefore, our findings support the strategy of longitudinal nutritional status assessment combined with personalized nutritional interventions to mitigate this pathological cascade and potentiate antitumor therapy effectiveness [35].

The principal innovation of this study resides in the pioneering integration of serial longitudinal nutritional assessments (admission and 3-month follow-up) with prognostic evaluation in advanced gastrointestinal malignancies. Our analysis revealed a significant correlation between evolving nutritional parameters and survival outcomes during antitumor therapy progression, establishing the critical importance of sustained nutritional surveillance. These findings substantiate the clinical imperative for implementing early-stage, comprehensive nutritional interventions with adequate caloric-protein provision in oncological management. Based on our findings, we recommend that patients with a PG-SGA score ≥ 9 at the 3-month follow-up should be referred for intensive, protocol-driven nutritional support, which may include personalized dietary counseling, high-energy high-protein oral nutritional supplements, and close monitoring for the need of enteral nutrition. The demonstrated prognostic value of serial nutritional assessments strongly advocates for the standardized incorporation of PG-SGA scoring into routine clinical practice for nutritional status monitoring.

While the statistical significance of the survival difference is clear, its clinical relevance merits emphasis. The observed 10-month disparity in median overall survival is substantial. A difference of 10 months indicating that dynamic PG-SGA-based risk stratification identifies groups with profoundly divergent prognoses. This magnitude of benefit is comparable to that achieved by many effective chemotherapy regimens, highlighting the critical role of nutritional status as a key prognostic modulator.

However, this study also has several limitations. The generalizability of this study's findings may be limited by its retrospective single-center design, relatively small cohort (n = 102), and overrepresentation of patients with upper gastrointestinal tumors in the final analysis cohort. Second, this study's sample size, although sufficient for the primary analysis, precluded meaningful subgroup analyses by specific cancer types, treatment modalities, or even more detailed AJCC stages. Gastrointestinal cancers are a heterogeneous group, and the impact of malnutrition may vary across these subtypes. Our findings should therefore be interpreted as representing an overall effect across advanced GI malignancies. Variations in the delivery of and adherence to standard nutritional care may have diluted the observed prognostic impact of malnutrition, potentially leading to an underestimation of its true association with survival. Additionally, the two-time-point design could not fully capture short-term nutritional fluctuations. Future studies with larger,

disease-specific cohorts and more frequent assessments are needed to confirm these results.

Based on the above findings, nutritional status evaluated by the PG-SGA demonstrated significant prognostic relevance in patients undergoing antitumor therapy. In advanced gastrointestinal malignancies, compromised PG-SGA scores at 3-month follow-up emerged as an independent prognostic indicator, emphasizing the critical role of longitudinal nutritional monitoring in clinical management. These findings substantiate the necessity for implementing early-stage, multidimensional, and sufficient nutritional support in oncological care. Further investigations should prioritize the development of precision nutritional interventions while elucidating the mechanistic relationships between nutritional status and tumor microenvironment dynamics as well as therapeutic responsiveness, to advance precision nutrition therapeutics in oncology. Additionally, implementing serial PG-SGA assessments may represent a cost-effective strategy by reducing later complications and hospitalizations, a hypothesis that merits further investigation.

Abbreviations

PG-SGA	Patient-Generated Subjective Global Assessment
OS	Overall survival
AJCC	American Joint Committee on Cancer
ESPEN	European Society for Clinical Nutrition and Metabolism
BMI	Body mass index
ALB	Serum albumin
CRP	C-reactive protein
HB	Hemoglobin
PNI	Prognostic nutritional index
CSCO	Chinese Society of Clinical Oncology
NCCN	National Comprehensive Cancer Network
ONS	Oral nutritional supplements;
IQR	Interquartile range

Supplementary Information

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Supplementary Material 1.

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Authors' contributions

Conceptualization, T.Z., S.W. and Q.L.; methodology, Q.L.; software, J.C., B.X. and P.Z.; validation, X.C., L.L. and X.C.; formal analysis, B.X.; investigation, P.Z.; resources, P.Z. and Q.L.; data curation, P.Z.; writing original draft preparation, P.Z., J.C. and B.X.; writing (review and editing), T.Z. and S.W.; visualization, Q.L. and B.X.; supervision, T.Z. and S.W.; project administration, S.W.; funding acquisition, P.Z., X.C. and L.L. All authors have read and agreed to the published version of the manuscript.

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Data availability

The datasets analysed during the current study are not publicly available because they contain personally identifiable information in medical records, but are available from the corresponding author on reasonable request.

Declarations

Ethics approval and consent to participate

This study was performed in line with the principles of the Declaration of Helsinki. The study protocol was approved by the Ethics Committee of Jiaxing Second Hospital (Approval No. 2023JX111-01). The requirement for informed consent was waived by the ethics committee because this study involved the analysis of existing anonymous data.

Consent for publication

Not applicable.

Competing interests

The authors declare no competing interests.

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