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Original Article

Early host adaptation defined by nutritional and inflammatory dynamics predicts disease progression in solid tumours

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Abstract: Background/Objectives: Whether inflammatory and nutritional parameters shift meaningfully in the first months of systemic treatment - and whether these shifts predict outcomes - has not been adequately addressed. We set out to track these changes from treatment initiation to month 3 in adults with newly diagnosed solid tumors, with progression-free and overall survival as primary endpoints. Methods: This was a single-center prospective study running from August 2023 through August 2024. Patients aged ≥ 18 with histologically proven solid tumors who were about to start systemic anticancer therapy were consecutively enrolled; 100 completed both assessment points. At each visit - baseline and month 3 - we recorded anthropometric data, performed BIA for body composition, administered the MNA-SF, measured handgrip strength and gait speed, and collected fasting blood samples for biochemical analysis. Wilcoxon signed-rank test, Spearman correlation, ROC curves, and logistic regression were the main analytical tools. Results: No sarcopenia was detected at baseline. By month 3, only one patient (1%) had developed it. Body fat percentage dropped significantly (P = 0.019); SMI and muscle mass stayed stable. MNA scores were essentially unchanged. Third-month CRP was the only independent predictor of both progression and survival (cut-off 4.74 mg/L; AUC 0.672; P = 0.008). CRP ≥ 4.74 mg/L conferred a 3.63-fold higher progression risk on multivariate analysis (OR 3.63; 95% CI 1.34-9.85; P = 0.011). Albumin correlated with SMI at month 3 (r = 0.214; P = 0.033). Conclusions: Sarcopenia was uncommon this early in the disease course. Inflammation, however, was not. Third-month CRP predicted outcomes independently - even in patients whose nutritional scores and muscle indices remained intact. This dissociation suggests inflammatory activation precedes measurable compositional decline. The positive correlation between albumin and SMI supports albumin's role as both a nutritional and functional marker. Routine CRP and albumin checks during early treatment may prove useful for identifying patients who need closer follow-up.

Keywords: Bioelectrical impedance analysis, C-reactive protein, nutritional status, disease progression

Introduction

Systemic inflammation, malnutrition, and functional decline are among the key determinants of prognosis in cancer patients. Extensive data exist on baseline inflammatory markers - C-reactive protein (CRP) in particular - yet how early post-diagnosis changes in these parameters influence disease course remains poorly defined. These shifts, occurring within the first months of therapy, may carry more prognostic weight than any static baseline value.

Sarcopenia is defined by loss of skeletal muscle mass, reduced strength, and impaired physical performance [1]. Its clinical relevance stems from established links to increased chemotherapy toxicity, reduced quality of life, and shorter survival [2]. Many patients with solid tumors already have muscle deficits at diagno-

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sis, which should inform therapeutic planning and early supportive care [3]. Effective management requires simultaneous assessment of nutrition, body composition, and systemic inflammation.

On a biological level, the way muscle vanishes in malignancy is essentially a metabolic struggle driven by chronic inflammation [4]. The body's internal balance is disrupted by signals originating from the tumor; to be specific, cytokines such as IL-6 and TNF-α force a shift toward protein degradation while simultaneously halting the synthesis of new muscle. Systemic treatment often accelerates this damage. The fatigue and nausea triggered by chemotherapy initiate a detrimental cycle: patients reduce their physical activity, which in turn destroys functional capacity. Within this cascade, CRP reflects the degree of systemic inflammatory burden.

Malnutrition in cancer is rarely a simple issue of reduced intake. It results from tumor-driven metabolic alterations that divert energy away from host tissues, while symptoms such as anorexia further limit caloric consumption [5]. Tools like the Mini Nutritional Assessment (MNA-SF) provide standardized nutritional screening [6], and bioelectrical impedance analysis (BIA) offers objective body composition data [7]. Although baseline CRP correlates with these metrics [8], the prognostic value of early changes in these markers - specifically during the first three months of treatment - remains poorly characterized.

Most existing literature focuses on data collected at a single baseline point or observes patients only during end-stage wasting. However, a vast number of patients initiate therapy with relatively stable nutritional profiles. Whether early inflammatory signals can independently forecast progression in these seemingly stable individuals is not yet established. To address this, we examined how three-month fluctuations in CRP, MNA-SF scores, and BIA-derived data correlate with progression-free and overall survival in newly diagnosed adults with solid tumors. Our secondary objective was to pinpoint a functional three-month CRP cutoff that serves as a reliable clinical indicator for forecasting disease progression in daily oncological practice.

Materials and methods

Study setting and recruitment

Conducted between August 2023 and August 2024 at our tertiary oncology center, this prospective observational study enrolled newly diagnosed adults with solid tumors. Every patient was scheduled for systemic therapy. We captured clinical and laboratory data at two intervals: baseline (within 7 days prior to treatment) and at the third month's conclusion. This specific window was utilized to align with the ethics timeline and ensure adequate accrual. We reduced selection bias through consecutive sampling. While the pragmatic goal was 100 participants, power estimates - informed by prior CRP prognostic signals (AUC ≈ 0.65-0.70) - suggested 90 patients would provide ~80% power ($\alpha = 0.05$) to detect a moderate effect size (OR ≈ 3.0). Our monthly accrual of 8-10 eligible cases made this target realistic.

Ethics

Adhering to the 2013 Declaration of Helsinki, the protocol was approved by the Pamukkale University Clinical Research Ethics Committee (ID: E396993; 26 July 2023). Participants provided written informed consent before any procedures. All data were de-identified to maintain privacy.

Eligibility and exclusion criteria

Inclusion required: (i) age ≥ 18 years; (ii) confirmed solid tumor; (iii) planned systemic therapy; (iv) completion of both assessments; and (v) written consent.

Exclusion criteria were: (i) hematologic malignancy; (ii) second primary tumors; (iii) advanced organ failure (e.g., cirrhosis, end-stage renal disease); (iv) mobility-limiting neuromuscular disease; (v) active infection/inflammatory disease; (vi) poor compliance/missing data; and (vii) prior GI surgery or radiotherapy impacting nutrition. No head-neck, esophageal, or gastric primaries were represented in this cohort. All patients were ambulatory with adequate oral intake.

Nutritional and body composition

Nutritional screening: We used the Mini Nutritional Assessment -Short Form (MNA-SF;

0-14). To prevent inter-observer bias, a single trained nurse performed all face-to-face evaluations. Scores were split into malnutrition (0-7), risk (8-11), and normal (12-14). Most patients were nutritionally stable; thus, no protocolized enteral/parenteral interventions were needed beyond standard oncologic care.

Body composition: Measured via Tanita TBF-300 BIA (morning, fasting, post-bladder emptying). Patients avoided exercise (12 h) and alcohol (24 h) prior to testing. Skeletal muscle index (SMI; kg/m^2) was derived from BIA. Per EWGSOP1 cut-offs, low muscle mass was defined as < 8.87 kg/m^2 for men and < 6.42 kg/m^2 for women [9, 10].

Functional and anthropometric tests

Mid-arm and calf circumferences were measured on the dominant side (standing, relaxed). Sarcopenia was defined by calf circumference (< 31 cm women; < 34 cm men) [11, 12]. We used calf circumference as a supportive indicator, not a diagnostic criterion. For mid-arm circumference, we only tracked longitudinal shifts since established reference cut-offs are missing. Strength was gauged via handgrip (Camry EH101), using the best of three trials [12]. Walking speed (m/s) was calculated through a 6-meter walk test following standard protocols [13].

Laboratory and inflammatory indices

Fasting blood samples were analyzed in an accredited laboratory. NLR, PLR, and MLR were manually derived. For biochemical quantification, we utilized immunoturbidimetry (CRP and albumin), electrochemiluminescence (ECLIA for vitamin D), and enzymatic colorimetry (total cholesterol), with results interpreted via laboratory reference ranges [14]. To avoid acute phase interference, patients with clinical infection were excluded at sampling. We calculated the PNI (10 × albumin [g/dL] + 0.005 × lymphocyte count [/mm^3]), Glasgow Prognostic Score (GPS), and modified GPS (mGPS) using CRP (> 5 mg/L) and albumin (< 35 g/L) thresholds.

Statistical analysis

Data were analyzed using IBM SPSS (v25.0). Continuous variables are reported as mean ± SD or median (range) based on the Kolmogorov-Smirnov test. Categorical variables are presented as frequencies and percentages. We used Wilcoxon signed-rank tests for repeated measures and Spearman's correlation. Survival (PFS/OS) was estimated via Kaplan-Meier curves and log-rank tests. Predictors of progression were identified using logistic regression ($P < 0.05$). The change in CRP (ΔCRP) was analyzed as a prognostic variable, dichotomized at ≥ 5 mg/L. To prevent model instability, the number of covariates was intentionally restricted given the limited number of progression events.

Results

One hundred patients receiving systemic anticancer treatment formed the study population. Ages ranged from 30 to 84 years, with a mean of 61.9 ± 11.0 (median 63.0); 59 were aged 65 years or younger. Fifty-nine patients (59%) were male and 41 (41%) were female. Mean height was 164.4 ± 8.3 cm (median 165.0 cm). Mean body weight fell from 70.1 ± 12.5 kg (median 69.1 kg) at baseline to 68.4 ± 14.6 kg (median 68.4 kg) by month 3. BMI followed a similar trajectory, declining from 26.1 ± 4.6 (median 25.5) to 25.3 ± 5.3 (median 25.1).

The cohort's demographic profile, lifestyle habits, and clinical characteristics are detailed in **Table 1**. Regarding smoking, 53% had never smoked; active smokers made up 27% and former smokers 20%. Alcohol use was rare - 93% reported no lifetime consumption, 6% had consumed alcohol in the past, and only 1% were active drinkers at enrollment. At least one comorbidity was present in 42% of patients. Stage distribution at diagnosis skewed toward advanced disease: stage III accounted for the largest share (46%), followed by stage IV (32%), stage II (18%), and stage I (4%). The median skeletal muscle index (SMI) values at baseline were normal for all patients. Sarcopenia developed in only one patient (1%) by month 3. By MNA-SF, the nutritional status was normal (scores 12-14) for the majority of patients at both the time points. Thirty-one percent of the patients were at risk of malnutrition at baseline and 28% at month 3, while 3-4% of the patients had frank malnutrition. The median MNA-SF scores at baseline and at month 3 were 12.5 (range 5-14) and 13.0 (range 6-14) respectively

Table 1. Sociodemographic and clinical characteristics of the study population (n = 100)

Variables	N	%
Age (years)		
Mean ± SD	61.88 ± 11.03	
Median (min-max)	63.0 (30-84)	
≤ 65	59	59.0
> 65	41	41.0
Sex		
Female	41	41.0
Male	59	59.0
Height (cm)		
Mean ± SD	164.43 ± 8.29	
Median (min-max)	165.0 (145.0-180.0)	
Weight (kg) baseline		
Mean ± SD	70.14 ± 12.45	
Median (min-max)	69.1 (38.4-98.0)	
Weight at month 3		
Mean ± SD	68.35 ± 14.56	
Median (min-max)	68.4 (32.0-99.0)	
BMI (kg/m^2) baseline		
Mean ± SD	26.08 ± 4.59	
Median (min-max)	25.5 (17.1-38.0)	
BMI at month 3		
Mean ± SD	25.27 ± 5.32	
Median (min-max)	25.1 (15.9-37.6)	
Smoking status		
Never	53	53.0
Current	27	27.0
Former	20	20.0
Alcohol		
Never	93	93.0
Current	1	1.0
Former	6	6.0
Tumor stage		
Stage I	4	4.0
Stage II	18	18.0
Stage III	46	46.0
Stage IV	32	32.0
Comorbidity		
Absent	58	58.0
Present	42	42.0
SMI at baseline		
Normal	100	100.0
Sarcopenic	0	0.0
SMI at month 3		
Normal	99	99.0
Sarcopenic	1	1.0
MNA at baseline		
Malnutrition	3	3.0
At risk	31	31.0
Normal	66	66.0
MNA at month 3		
Malnutrition	4	4.0
At risk	28	28.0
Normal	68	68.0
Progression		
No	72	72.0
Yes	28	28.0
Survival status		
Alive	88	88.0
Dead	12	12.0
Follow-up (months)		
Mean ± SD	12.0 ± 3.5	
Median (min-max)	12.3 (5.0-24.0)	

and there was no significant difference (P = 0.974). The median follow-up in these patients was 12.3 months (range 5.0-24.0). By the end of the follow-up, 28% of the patients had progressed and 12% had died.

Patients presented with a variety of solid tumors. The two most common were lung cancer, seen in 33 patients (33%), and breast cancer, in 30 (30%). Prostate cancer affected 8 patients (8%). Five had colorectal cancer (5%) and three had pancreatobiliary tumors (3%). Among women, gynecologic cancers were present in 14 patients overall: ovarian in 7, cervical in 4, and endometrial in 3. Sarcoma was diagnosed in 4 patients, primary brain tumors in 2, and mesothelioma in 1. The cohort included no cases of head-neck, esophageal, or gastric cancer.

Treatment was tailored to tumor type according to established standards. Lung cancer patients received platinum-based doublets. Fluoropyrimidine-based regimens were given in colorectal cancer and selected metastatic breast cancer cases, capecitabine included. Pancreatobiliary tumors were managed with gemcitabine-based protocols. In prostate cancer, androgen-deprivation or endocrine therapy was the primary modality; chemotherapy was reserved for select cases. No patient required enteral or parenteral nutrition throughout the

Table 2. Comparison of body composition and anthropometric measurements at baseline and third month

Variables	Baseline	3rd month	*p*-value
	Median (min-max)	Median (min-max)	
Body fat percentage (%)	24.7 (8.10-48.90)	22.6 (1.70-47.80)	0.019
Fat-free mass (kg)	51.2 (33.50-69.20)	49.9 (12.0-70.30)	0.152
Mid-arm circumference (cm)	31.0 (11.0-42.00)	31.0 (12.0-38.00)	0.370
Calf circumference (cm)	39.0 (14.5-48.00)	38.0 (12.0-48.00)	0.238
Skeletal muscle index (SMI, kg/m^2)	18.8 (13.3-24.9)	18.6 (5.1-28.1)	0.142

Wilcoxon signed-rank test; P < 0.05 was considered statistically significant.

Table 3. Comparison of nutritional assessment scores at baseline and third month

Variables	Baseline	3rd month	*p*-value
	Median (min-max)	Median (min-max)	
MNA-SF score	12.5 (5-14)	13 (6-14)	0.974

Wilcoxon signed-rank test; P < 0.05 was considered statistically significant.

study. ECOG performance status at baseline was 0-1 in 88 patients (88%) and ≥ 2 in 12 (12%). All participants completed the 6-meter gait-speed and handgrip-strength tests at baseline.

All body composition parameters remained stable over time from baseline to month 3, with the exception of body fat percentage. A significant reduction in body fat percentage was observed, with the median value falling from 24.7% (8.1-48.9) to 22.6% (1.7-47.8) between the two time points (P = 0.019). The remaining parameters - FFM, mid-arm circumference, and calf circumference - were all statistically unchanged (P = 0.152, P = 0.370, and P = 0.238, respectively) (**Table 2**).

MNA-SF screening gave a median of 12.5 (range 5.0-14.0) at enrollment. By the third month, the median had shifted to 13.0 (range 6.0-14.0); Wilcoxon testing confirmed no significant difference between the two assessments (P = 0.974). In terms of category distribution, the normal range (12-14) captured the majority of participants at both time points, while smaller proportions met criteria for nutritional risk (8-11) or malnutrition (0-7) at one or both assessments (**Table 3**).

Haemoglobin was the only biochemical parameter to drop significantly during treatment. Its median at baseline was 12.6 g/dL (8.4-16.8); by month 3, this had fallen to 11.9 g/dL (8.9-16.1) (P = 0.003). Albumin showed no meaningful shift (42.1 vs. 42.2 g/L; P = 0.573), nor did CRP (P = 0.910) or NLR (P = 0.975). Platelet and WBC counts were exceptions - both fell significantly by month 3 (P < 0.001). Full laboratory comparisons are in **Table 4**.

Functional performance assessed by the six-meter walk test showed no significant change between baseline and third month (median 5 s at both time points; P = 0.272) (**Table 5**).

MNA scores and SMI showed consistent positive correlations across time points. At month 3, MNA scores were associated with both concurrent SMI (r = 0.451, P < 0.001) and baseline SMI (r = 0.286, P = 0.004). A weaker association was noted between baseline MNA and month 3 SMI (r = 0.192, P = 0.056). Serum albumin at third month correlated positively with SMI (r = 0.214, P = 0.033). No significant correlation was found between CRP and SMI (**Table 6**).

Regarding nutrition-inflammation interactions, PLR and MLR showed weak but significant negative correlations with MNA scores. At baseline, PLR yielded r = -0.272 (P < 0.01); at month 3, r = -0.217 (P < 0.05). MLR values were r = -0.209 (P < 0.05) at baseline and r = -0.200 (P < 0.05) at month 3. No significant association was observed between the NLR and MNA (**Table 7**).

ROC analyses demonstrated that only CRP and albumin levels at month 3 had significant predictive values for disease progression. CRP

Table 4. Comparison of laboratory parameters between baseline and third month

Variables	Baseline	3rd month	*p*-value
	Median (min-max)	Median (min-max)	
Hemoglobin (g/dL)	12.6 (8.4-16.8)	11.9 (8.9-16.1)	0.003
Albumin (g/L)	42.1 (22.4-46.9)	42.2 (22.5-50.3)	0.573
CRP (mg/L)	3.4 (0.3-141)	2.8 (0.3-276)	0.910
NLR	2.7 (0.7-27.9)	2.6 (0.5-25.6)	0.975
PLR	157.4 (67.4-803.7)	167.2 (34.7-608.1)	0.810
MLR	0.2 (0.1-2.2)	0.3 (0.1-2.1)	0.178
Platelets (/μL)	277000.0 (57000-768000)	240500.0 (65000-574000)	< 0.001
WBC	8120.0 (3450-21890)	6150.0 (2750-15570)	< 0.001

Wilcoxon signed-rank test; P < 0.05 was considered statistically significant.

Table 5. Comparison of physical performance parameters between baseline and third month

Variables	Baseline	3rd month	*p*-value
	Median (min-max)	Median (min-max)	
6-m walk test (seconds)	5.0 (3.0-15.0)	5.0 (2.0-15.0)	0.272

Wilcoxon signed-rank test; P < 0.05 was considered statistically significant.

Table 6. Correlations between SMI at baseline and third month and selected variables

Variables		SMI Baseline	SMI 3rd month
MNA score at baseline	R	0.192	0.286**
	P	0.056	0.004
MNA score at 3rd month	R	0.230*	0.451**
	P	0.021	< 0.001
CRP at baseline	R	0.042	0.000
	P	0.676	0.997
CRP at 3rd month	R	-0.056	-0.158
	P	0.580	0.117
Albumin at baseline	R	-0.059	0.023
	P	0.562	0.817
Albumin at 3rd month	R	0.131	0.214*
	P	0.193	0.033

Spearman's rank correlation test: *P < 0.05 was considered statistically significant and **P < 0.01 was considered highly significant.

showed the highest discriminative performance (AUC = 0.672, P = 0.008), with a cutoff of ≥ 4.74 mg/L (sensitivity 67.9%, specificity 66.7%). Albumin yielded an AUC of 0.631 (P = 0.042), with a cutoff of ≤ 41.89 g/L predicting progression (sensitivity 60.7%, specificity 61.1%). SMI, SMI change, and MNA scores were not significantly different (**Table 8**). ΔCRP values showed wide interindividual variation (approximately -140 to +266 mg/L; median change near zero). ROC analysis of ΔCRP demonstrated modest discrimination for disease progression (AUC = 0.575). A ΔCRP increase ≥ 5 mg/L was associated with a higher risk of progression (OR = 4.08; 95% CI 1.33-12.50; P = 0.014). In a multivariable model including both the third-month CRP and ΔCRP, only the third-month CRP remained independently predictive (per-mg/L OR = 1.029; 95% CI 1.005-1.054; P = 0.020), whereas ΔCRP lost significance (P = 0.63).

In exploratory analyses, composite indices did not outperform single markers. PNI at month 3 had an AUC of 0.538 (optimal cut-off < 43.9; sensitivity 35.7%; specificity 85.9%; permutation P = 0.263), falling short of significance. mGPS reached borderline levels (AUC 0.581; cut-off ≥ 2; sensitivity 25.0%; specificity 92.9%; permutation P = 0.055). CRP, with an AUC of 0.672, remained the strongest individual predictor, in line with the primary analysis.

The one-year overall survival (OS) rate was 91.8%. Median OS was not reached. No significant differences were observed in OS by age (≤ 65 vs. > 65 years, P = 0.942) or sex (P = 0.640). However, OS was significantly different by CRP level. When patients were split by the

Table 7. Correlations between MNA at baseline and month 3 and various variables

Variables		MNA baseline	MNA Month 3
NLR at baseline	R	-0.195	-0.056
	P	0.052	0.583
NLR at 3rd month	R	-0.022	0.027
	P	0.831	0.791
PLR at baseline	R	-.272**	-.217*
	P	0.006	0.030
PLR at 3rd month	R	-0.038	0.027
	P	0.704	0.792
MLR at baseline	R	-0.141	-0.056
	P	0.162	0.583
MLR at 3rd month	R	-0.030	-0.033
	P	0.769	0.747

Spearman's rank correlation test: *P < 0.05 was considered statistically significant and **P < 0.01 was considered highly significant.

4.74 mg/L CRP threshold, those below it had a one-year OS of 98.1%, while those at or above it fared worse at 83.7% (P = 0.008). A parallel pattern emerged for albumin, though it did not reach statistical significance - 98.1% for values above 41.89 g/L versus 84.4% at or below this level (P = 0.090) (**Table 9**).

One-year PFS was 70.3%; median PFS was not reached. Age and sex did not significantly influence PFS (P = 0.234 and P = 0.260, respectively). At the 4.74 mg/L cutoff, CRP separated patients into two distinct outcome groups - one-year PFS was 83.7% below this threshold and 54.1% at or above it (P = 0.003). Patients with albumin exceeding 41.89 g/L trended toward improved PFS, though this did not cross the threshold for statistical significance (P = 0.056) (**Table 10**).

Logistic regression analysis was conducted to identify the predictors of disease progression. In the univariate model, third-month CRP level, albumin level, and MNA score were significantly or borderline associated with the risk of progression. Of these, CRP alone survived multivariate adjustment. At the 4.74 mg/L threshold, third-month CRP carried more than a fourfold odds of progression (OR = 4.22; 95% CI 1.66-10.72; P = 0.002); after multivariate adjustment, this risk estimate remained meaningful (OR = 3.63; 95% CI 1.34-9.85; P = 0.011). CRP thus emerged as an independent predictor of disease progression in this cohort.

Third-month albumin ≤ 41.89 g/L was associated with elevated progression risk in univariate analysis (OR = 2.42; P = 0.049), yet this effect did not persist after multivariate adjustment (P = 0.253). MNA score, SMI, age, sex, and inflammatory indices - NLR, PLR, and MLR - likewise failed to show independent associations with disease progression. The model performance metrics indicated a -2 log likelihood of 107.263 and an R^2 of 0.154. The detailed regression results are presented in **Table 11**.

Discussion

Few clinical efforts have mapped how sarcopenia, nutritional status, and systemic inflammation interact in newly diagnosed solid tumor patients. This prospective work addresses that gap directly. Much of the existing literature is based on cross-sectional data from a single point in time [1, 15]. These snapshots usually miss the rapid metabolic shifts that occur early in cancer. We looked at these dynamics by tracking patients at two specific landmarks: right before starting systemic therapy and again at the three-month point. This longitudinal approach was key to monitoring biological changes as they happened. By doing this, the study caught early shifts that anyone relying on a single baseline snapshot would likely miss.

Our data points to one particularly clear result: having a serum CRP level ≥ 4.74 mg/L at the three-month mark acts as an independent prognostic marker for both disease progression and survival. Many studies focus on pretreatment CRP or advanced cachexia, but our results show this early on-treatment window is critical. This prognostic weight persists even when a patient's nutritional and functional picture looks reassuring at the outset. This probably mirrors underlying tumor-driven catabolism and a growing resistance to treatment. These findings match Shrotriya et al., who found CRP ≥ 5 mg/L predicted poor survival across nearly 5,000 patients with solid tumors (HR 1.65;

Table 8. Predictive value of various parameters in discriminating disease progression

Variables	AUC	95% CI	Cut-off	Sensitivity (%)	Specificity (%)	*p*-value
SMI at 3rd month	0.510	0.395-0.625	≤ 18.67	50.0	50.0	0.881
SMI-change	0.579	0.452-0.706	≥ 0.35	60.7	59.7	0.221
MNA at 3rd month	0.581	0.452-0.710	≤ 12.50	57.1	61.1	0.208
CRP at 3rd month	0.672	0.550-0.794	≥ 4.74	67.9	66.7	0.008
Albumin at 3rd month	0.631	0.495-0.768	≤ 41.89	60.7	61.1	0.042
NLR at 3rd month	0.514	0.373-0.656	≤ 2.61	53.6	51.4	0.824
PLR at 3rd month	0.573	0.450-0.695	≤ 164.39	50.0	51.4	0.261
MLR at 3rd month	0.525	0.396-0.653	≤ 0.31	53.6	54.2	0.704
PNI at 3rd month	0.538	0.410-0.660	< 43.9	35.7	85.9	0.263
mGPS at 3rd month	0.581	0.447-0.716	≥ 2	25.0	92.9	0.055

AUC: Area under the curve; 95% CI: 95% confidence interval.

Table 9. Comparison of overall survivals (1-year OS %) (n = 100)

Variables	1-year OS (%)	Median OS (months)	*p*-value
Overall	91.8	Not reached	
Age			
≤ 65	91.2	Not reached	0.942
> 65	92.7	Not reached	
Sex			
Male	92.3	Not reached	0.640
Female	91.5	Not reached	
CRP at 3rd month			
< 4.74	98.1	Not reached	0.008
≥ 4.74	83.7	Not reached	
Albumin at 3rd month			
> 41.89	98.1	Not reached	0.090
≤ 41.89	84.4	Not reached	

Kaplan-Meier method; log-rank test; P < 0.05 was considered statistically significant.

Table 10. Comparisons of progression-free survivals (n = 100)

Variables	1-year PFS (%)	Median PFS (95% CI)	*p*-value
Overall	70.3	Not reached	
Age			
≤ 65	65.7	Not reached	0.234
> 65	76.6	Not reached	
Sex			
Male	74.8	Not reached	0.260
Female	67.1	Not reached	
CRP at 3rd month			
< 4.74	83.7	Not reached	0.003
≥ 4.74	54.1	Not reached	
Albumin at 3rd month			
> 41.89	79.3	Not reached	0.056
≤ 41.89	59.6	Not reached	

Kaplan-Meier method; Log-rank test; P < 0.05 was considered statistically significant.

95% CI 1.49-1.82; P < 0.001) [16]. Composite markers like the CRP-to-albumin ratio are also relevant - a meta-analysis of 10,556 patients linked elevated CAR to worse overall survival (HR 1.95; 95% CI 1.71-2.22; P < 0.001) [17] - yet our models pointed to the absolute CRP value at month three as the most dependable independent predictor in this group.

Regular CRP and CAR monitoring during treatment supports early identification of high-risk patients. Serial assessment may help guide the timing of personalized supportive care. Rising CRP from baseline to month three was associated with progression in univariate analysis. The absolute month-three value, however, proved more informative in multivariable models. Tracking the trend has value, but the later measurement carries more prognostic weight. Given the modest sample size, these thresholds should be interpreted with caution. They do reflect early progression dynamics within a one-year follow-up.

ROC analysis showed acceptable discrimination for CRP,

Table 11. Univariate and multivariate logistic regression results for various variables on progression risk

Variable	Univariate OR (95% CI)	Univariate *p*-value	Multivariate OR (95% CI)	Multivariate *p*-value
Age > 65 vs. ≤ 65	0.59 (0.23-1.48)	0.263	-	-
Female vs. Male	1.68 (0.67-4.23)	0.264	-	-
SMI at 3rd month (continuous)	0.97 (0.83-1.14)	0.789	-	-
MNA at 3rd month (continuous)	0.85 (0.70-1.05)	0.138	0.99 (0.78-1.25)	0.936
CRP ≥ 4.74 vs. CRP < 4.74	4.22 (1.66-10.72)	0.002	3.63 (1.34-9.85)	0.011
Albumin ≤ 41.89 vs. > 41.89	2.42 (0.99-5.93)	0.049	1.78 (0.66-4.78)	0.253
NLR at 3rd month (continuous)	1.05 (0.96-1.15)	0.278	-	-
PLR at 3rd month (continuous)	0.99 (0.99-1.01)	0.216	-	-
MLR at 3rd month (continuous)	1.41 (0.43-4.58)	0.566	-	-

Model fit: -2 Log likelihood = 107.263, Nagelkerke R^2 = 0.154.

with an AUC of 0.672 (P = 0.008). This falls within the 0.65-0.75 range generally accepted as indicative of acceptable discrimination [18]. In multivariable analysis, CRP ≥ 4.74 mg/L was associated with a 3.63-fold increased risk of progression (OR 3.63; 95% CI 1.34-9.85; P = 0.011). CRP reflects systemic inflammation, muscle catabolism, and nutritional status simultaneously. This makes it a practical marker for both risk stratification and timing of supportive interventions.

At baseline, all patients had SMI values above sex- and height-adjusted cutoff thresholds, and no sarcopenia was detected. At the end of the third month, only one patient developed new-onset sarcopenia - cancer-related catabolic processes, it seems, were only partially activated this early. Sarcopenia prevalence at diagnosis has been reported anywhere from 21% to 57% in the literature, a range that reflects the substantial influence of diagnostic criteria, measurement method, and disease stage on these estimates [1, 2].

That said, this picture needs some qualification. Short follow-up and a cohort with largely preserved baseline nutritional status may have limited our ability to catch early compositional changes - what looks like biological stability may partly be a detection gap. The low sarcopenia rate at baseline most likely reflects the dominance of early-to-intermediate stage disease and the absence of major metabolic disruption before treatment began. If anything, this early window may be exactly when nutritional support and structured exercise can do the most good.

Most patients entered the study at relatively early disease stages and had not yet crossed into a phase of full catabolic activation. By month three, sarcopenia appeared in just one case. Whether the nutritional counseling and physical activity guidance offered from the start played a role in this outcome is hard to say with certainty - but given the adherence levels observed, it seems plausible. Prado et al. found that low pre-chemotherapy SMI in gastrointestinal cancer patients was tied to worse overall survival (HR 1.63; 95% CI 1.17-2.27; P = 0.004) [19]. Seen against that backdrop, the near-absence of sarcopenia in our first three months looks like a clinically meaningful signal - one that may reflect both the value of early intervention and a functional reserve that was still largely intact.

Body fat percentage dropped significantly over the first three months (P = 0.019). This fits with what we would expect from the energy imbalance and catabolic pressure that come with active treatment. Some patients lost weight alongside this, while others managed to hold onto their muscle mass. In gastrointestinal and advanced solid tumor patients, adipose tissue loss has been shown to carry prognostic weight comparable to sarcopenia [19, 20] - a reminder that fat is not simply stored fuel but an organ with real endocrine and immune functions. Martin et al., working with 1,473 gastrointestinal and lung cancer patients, found that low CT-measured SMI independently predicted worse overall survival (HR: 2.62; 95% CI: 1.76-3.89; P < 0.001), with SMI improving model discrimination substantially (c-statistic: 0.73-0.92) [21]. Both muscle and fat composition,

then, appear to matter for prognosis. The fat loss we saw may be an early warning of metabolic trouble ahead - though with baseline BMI sitting in the overweight range, part of this decline could simply reflect a return toward a lower but still non-critical adiposity level rather than true pathological wasting.

MNA scores nudged upward slightly from baseline to month three, but not significantly so. Most patients stayed in the normal range throughout; at-risk and malnourished categories remained small minorities. The exclusion of patients with prior GI surgery or radiotherapy, combined with routine dietary counseling, likely helped keep nutritional status stable during the first three months of treatment. No formal medical nutrition therapy was in place at our center - patients got standard oncology care and general dietary advice, nothing more. The MNA-SF breakdown changed little: 66% normal, 31% at risk, 3% malnourished at baseline versus 68%, 28%, and 4% at month three. Formal interventions probably didn't drive the associations we saw. Still, we can't rule out individual diet changes or unsupervised supplements. These things might have diluted our effect estimates. Future work needs to specifically enroll at-risk and malnourished patients on formal nutrition therapy. That is the only way to find out whether such interventions actually shift the prognostic value of inflammatory or body-composition markers. Cancer-related malnutrition is a tough, complex problem. It involves anorexia, GI toxicity, tumor inflammation, and psychosocial stress. This combination often resists quick fixes, even with aggressive short-term support [22]. Overcoming it requires an intensive, individualized, and multidisciplinary approach right from the start.

The MNA functions as both a screening instrument and a prognostic signal. A 2015 prospective study showed that gastrointestinal tumor patients with low MNA scores had shorter survival and more treatment toxicity [23]. In geriatric oncology, even a one-point MNA gain has been linked to meaningful survival improvement [24].

Most patients in this cohort held onto normal nutritional status from diagnosis through month three; those flagged as at-risk or malnourished stayed a clear minority. The clinical implication is straightforward: catching these vulnerabilities early - before deterioration sets in - matters more than reacting after the fact. When elevated CRP is also present, the margin for MNA improvement appears to narrow considerably. This is why standard nutritional approaches alone may not be enough; anti-inflammatory and metabolic support strategies have been recommended as adjuncts in this setting [25, 26].

Third-month albumin discriminated disease progression with an AUC of 0.631 ($P = 0.042$). Univariate analysis linked low albumin to higher progression risk ($P = 0.049$), though this did not survive multivariate adjustment ($P = 0.253$). Albumin captures both nutritional depletion and inflammatory load - a dual role well-documented in the prognostic literature. Gupta and Lis showed pretreatment hypoalbuminemia worsened survival outcomes [27]; McMillan pointed to systemic inflammation as a key driver of albumin's clinical relevance [28].

Albumin and SMI correlated positively at month three ($r = 0.214$; $P = 0.033$), suggesting albumin may reflect muscle integrity as much as nutritional state. NLR, PLR, and MLR showed no meaningful change over the study period [29-31]. Among these, PLR was the only ratio with a detectable relationship - an inverse correlation with baseline MNA scores ($r = -0.272$; $P = 0.006$) [29]. Looking across the full dataset, CRP and albumin carried more discriminatory weight than any of the hematologic ratios. What happens to these trajectories beyond the current observation window remains an open question - one that will need larger, multicenter studies with longer follow-up to answer properly.

Several methodological strengths merit mention. Patients were enrolled from diagnosis and followed with repeated assessments covering anthropometric, biochemical, functional, and inflammatory parameters. Parallel tracking allowed observation of how sarcopenia, nutritional decline, and inflammation evolve together. Single-center enrollment with a consistent team and uniform protocols kept measurement variability low. Handgrip strength and gait speed were recorded but excluded from multivariable models given the limited event count; their associations were explored descriptively.

This study has several limitations. Enrolling patients across multiple solid tumor types introduced biological and clinical heterogeneity. The sample size is sufficient for exploratory analysis but limits generalizability. BIA is practical but vulnerable to hydration-related error and less precise than CT for muscle quantification. Three months of follow-up leaves the longer-term trajectory of sarcopenia and nutritional indices uncharted. Advanced-stage patients were underrepresented, limiting subgroup power; multicenter, longer-term studies are needed. Head and neck, esophageal, and gastric cancers were absent from this series, so extrapolation to those populations should be cautious. PNI and mGPS showed prognostic value in other cohorts, yet neither outperformed CRP here. CRP remains the most informative single marker at three months; PNI < 45 showed only modest stratification in dichotomized analyses.

Conclusion

Sarcopenia was uncommon at initial diagnosis in this prospective cohort; malnutrition and systemic inflammation, however, were encountered more frequently and carried real clinical weight. CRP measured at month three emerged as one of the strongest predictors of both disease progression and overall survival. The albumin-SMI correlation raises the possibility that albumin reflects not just nutritional adequacy but also functional reserve. Together, these findings argue for assessing inflammatory and nutritional parameters in tandem - not in isolation - at the start of treatment. Routine tracking of CRP and albumin could sharpen early risk stratification and allow supportive care to be deployed before clinical deterioration becomes established.

Disclosure of conflict of interest

None.

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