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Admission-Based Tumor and Immunonutritional Profiling for Six-Month Mortality after Colorectal Cancer Hospitalization

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ABSTRACT: Hospitalized patients with colorectal cancer (CRC) may deteriorate because tumor burden, systemic inflammation, and nutritional decline interact during and after admission. We evaluated whether the albumin-derived neutrophil-to-lymphocyte ratio (Alb-dNLR), calculated from admission laboratory data, was associated with predefined six-month all-cause mortality and compared it with carcinoembryonic antigen (CEA), serum albumin, dNLR, and recorded nutrient-intake change. Medical records of 569 adults with CRC admitted to Dr. Wahidin Sudirohusodo Hospital, Makassar, Indonesia, were screened; 327 were excluded because laboratory components required for biomarker calculation were incomplete and no imputation was performed. The final cohort included 242 patients. Vital status was determined for six months from the index admission. Discrimination was assessed using receiver operating characteristic curves, and independent associations were examined with a prespecified logistic regression model. During follow-up, 59 patients died (24.4%) and 183 remained alive (75.6%). Alb-dNLR showed modest discrimination (AUC, 0.618; 95% CI, 0.533-0.703; p = 0.007). The ROC-derived cut-off of 1.5 corresponds to an Alb-dNLR score of 2 on the 0-2 ordinal scale, yielding low sensitivity (20.3%) but high specificity (93.4%). After adjustment, Alb-dNLR was the only variable independently associated with six-month mortality (OR, 2.405; 95% CI, 1.124-5.149; p = 0.024). These findings support Alb-dNLR as a highly specific adjunct for recognizing a high-risk inflammatory-nutritional phenotype, not as a stand-alone screening test.

1. INTRODUCTION

Colorectal cancer (CRC) accounts for a substantial share of the global cancer burden [1-4]. In patients who require hospitalization, prognosis is not determined by tumor characteristics alone. Acute infection, bowel obstruction, operative stress, organ dysfunction, cachexia, and inadequate intake can alter clinical reserve during the admission and after discharge. Admission variables that are inexpensive and routinely available may therefore complement conventional oncologic information when early risk needs to be recognized.

A prominent feature of gastrointestinal malignancy is the overlap between systemic inflammation and nutritional deterioration. Inflammatory signaling may reduce appetite and gastrointestinal tolerance while promoting capillary leakage, altered hepatic protein synthesis, and proteolysis. Although serum albumin is often used in nutritional assessment, it is also influenced by hydration, liver synthetic function, vascular permeability, and the acute inflammatory response. Accordingly, a low albumin concentration is better viewed as a marker of impaired host reserve than as a direct measure of malnutrition in isolation [5-7].

The derived neutrophil-to-lymphocyte ratio (dNLR) can be obtained from routine leukocyte and neutrophil counts. By relating neutrophil predominance to the remaining circulating leukocyte pool, it provides a simple index of systemic inflammatory activity. Neutrophil-based indices have been associated with cancer progression, reduced antitumor immune competence, treatment response, and adverse outcomes across solid malignancies [8].

Albumin and dNLR therefore represent different components of the same clinical vulnerability: one reflects reduced physiologic reserve, whereas the other reflects inflammatory activation. Albumin-dNLR (Alb-dNLR) scores and related albumin-neutrophil measures have been studied in rectal cancer receiving neoadjuvant treatment, gastric cancer, older patients with CRC, postoperative cohorts, and selected non-oncologic populations [9-14]. Most of that evidence, however, concerns treatment response, postoperative prognosis, or longer-term survival rather than risk identified at the time of hospital admission.

We used an admission-centered framework to compare tumor-related, inflammatory, albumin-based, immunonutritional, and nutrient-monitoring variables against a fixed six-month mortality endpoint after CRC hospitalization. The analysis was not designed to present Alb-dNLR as a novel biomarker; instead, it tested whether this readily derived score could identify a clinically meaningful high-risk phenotype when measured during routine inpatient care. We specifically evaluated the discrimination and independent association of Alb-dNLR and compared its performance with CEA, albumin, dNLR, and changes in recorded nutrient intake.

2. MATERIALS AND METHODS

2.1 Study Design and Setting

We performed a retrospective cohort analysis at Dr. Wahidin Sudirohusodo Hospital in Makassar, Indonesia. Hospital records of patients admitted with CRC between January 2023 and December 2024 formed the sampling frame. Clinical and laboratory information recorded during the index admission was linked with documented vital status over the following six months. Reporting followed the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) statement [15].

2.2 Participants and Patient Selection

After repeated admissions and duplicate records were removed, 569 unique CRC records were screened. The index hospitalization was defined as the first eligible admission within the study period. Biomarker construction was not possible in 327 records because at least one required laboratory component was absent, such as serum albumin, total white blood cell count, neutrophil data, lymphocyte data and CEA. Missing biomarker components were not imputed because the study relied on routinely documented clinical records and because imputation could distort derived ratios and score classification. The analytic cohort therefore comprised 242 patients with complete data for the primary biomarker analysis.

Inclusion required age 18 years or older, a diagnosis of CRC documented in the medical record, baseline information sufficient to derive the selected biomarkers, and medical-record documentation that allowed six-month vital status to be determined. Records lacking the minimum biomarker components were excluded from biomarker, ROC, and regression analyses. Only one index hospitalization per patient was retained to preserve independent observations. Figure 1 summarizes the cohort-selection process and the handling of incomplete records.

2.3 Outcome Definition

The study endpoint was all-cause death occurring within six months of the index admission. Follow-up was anchored to the first day of that admission. The six-month cut-off was specified before analysis because it captures mortality during hospitalization and the early post-discharge period, when patients with CRC may remain vulnerable to complications, poor intake, treatment intolerance, and progressive host-response deterioration. Hospital records were used to establish whether death occurred during the hospitalization or after discharge. A patient was classified as a nonsurvivor when death was documented before completion of the six-month interval; survivor status required documentation that the patient was alive at six months. Exact post-discharge dates of death were unavailable for some patients, so the outcome was analyzed as a binary six-month status rather than as time-to-event data. Kaplan-Meier and Cox proportional hazards analyses were therefore not used.

2.4 Clinical and Laboratory Variables

Recorded clinical characteristics comprised age, sex, education, occupation, comorbid conditions, metastatic sites, complications, laparotomy, chemotherapy, radiotherapy, and referral to the clinical nutrition team. The comorbidity variables included hypertension, diabetes mellitus, heart disease, hepatitis B or C, acute kidney injury, chronic kidney disease, and tuberculosis. Metastatic sites were determined from radiologic imaging reports and included the liver, lung, ovary/cervix/uterus, bone, pancreas, bladder, and brain. Complications of interest included adhesions, obstructive ileus, pneumonia, sepsis, pleural effusion, encephalopathy, and ascites.

Laboratory variables included total white blood cell count, absolute neutrophil count when available, neutrophil and lymphocyte percentages, serum albumin, neutrophil-to-lymphocyte ratio (NLR), dNLR, total lymphocyte count, and CEA. For baseline analyses, we used the earliest available measurement from the index admission, preferentially from the first 24-72 hours when such data were available.

2.5 Definition of Inflammatory-Nutritional Indices

NLR was calculated from the leukocyte differential percentages as neutrophil percentage divided by lymphocyte percentage. dNLR was calculated using absolute neutrophil count (ANC) and total white blood cell count (WBC) with the same absolute unit: dNLR = ANC / (WBC - ANC). When ANC was derived from WBC and neutrophil percentage, the derived value and WBC were kept in the same unit to avoid unit-related distortion.

Alb-dNLR was scored from two binary components. Serum albumin values of 3.5 g/dL or higher received 0 points and values below 3.5 g/dL received 1 point. A dNLR below 3.0 received 0 points, whereas dNLR values of 3.0 or higher received 1 point. Summing the two components yielded an ordinal score of 0, 1, or 2: score 0 indicated neither hypoalbuminemia nor elevated dNLR, score 1 indicated one abnormal component, and score 2 indicated the coexistence of hypoalbuminemia and elevated systemic inflammation.

2.6 Nutrient Intake Change Assessment

Dietary intake information was taken from routine records generated by the clinical nutrition service. When serial monitoring was available, energy, protein, carbohydrate, and fat intake recorded at the first nutrition assessment was compared with the last assessment before discharge or death.

These measures describe change during hospitalization rather than habitual intake or a single baseline dietary exposure. A nutrient was categorized as improved when its final recorded intake exceeded the initial value; otherwise it was classified as decreased or not improved. Macronutrients were evaluated separately. Because these data were limited to patients who underwent clinical nutrition monitoring and could be influenced by severity of illness, referral practices, timing of documentation, and response to nutritional care, they were treated as exploratory variables. Patients without serial nutrition records were not imputed for these exploratory analyses.

2.7 Statistical Analysis

Statistical processing was carried out with IBM SPSS Statistics for Windows, version 25.0. Continuous variables were described using mean and standard deviation or median and interquartile range, according to their distribution. Categorical data were summarized as frequencies and percentages.

Comparisons were made between patients alive at six months and those who died within that interval. Categorical variables were tested with the Chi-square test or Fisher exact test, whereas continuous variables were examined with an independent-samples t test or Mann-Whitney U test as appropriate. All tests were two-sided, with statistical significance defined at $p < 0.05$.

Discriminative performance for six-month mortality was explored using receiver operating characteristic (ROC) curves for Alb-dNLR, CEA, albumin, dNLR, and nutrient-intake change variables. We reported the area under the curve (AUC), 95% confidence interval (CI), selected threshold, sensitivity, specificity, Youden index, and p value. Thresholds were chosen from the ROC output to maximize the Youden index unless the variable was an ordinal score. For Alb-dNLR, the statistical cut-off of 1.5 was interpreted as score 2 on the 0-2 scale. For variables in which lower values indicate greater biological risk, particularly albumin, an AUC below 0.50 was interpreted in relation to the direction of coding rather than taken automatically to mean absence of clinical relevance.

Independent associations with six-month mortality were examined using multivariable logistic regression, with odds ratios (ORs) and 95% CIs as effect estimates. Logistic regression was selected because the endpoint was a binary six-month vital-status variable. To limit overfitting in relation to the 59 observed deaths, the adjusted model was confined to prespecified clinically relevant variables. Independent observations were supported by retaining only one index hospitalization per patient. Because Alb-dNLR already incorporates albumin and dNLR, the composite score was prioritized over simultaneous entry of its individual components to reduce collinearity. CEA was modeled as a continuous variable, and nutrient-intake change measures were retained as exploratory complete-case covariates. The model was interpreted as associative rather than causal because residual confounding could not be eliminated in the retrospective design.

3. RESULTS AND DISCUSSION

3.1 Patient Selection and Six-Month Outcome

Figure 1 outlines cohort assembly. Of 569 unique CRC records screened after removal of duplicate or repeat admissions, 327 lacked sufficient laboratory information for biomarker derivation and were excluded without imputation. The remaining 242 patients constituted the final analytic sample. This selection process was used to avoid invalid calculation of ratios and composite scores, but it also introduces a potential selection bias that should be considered when interpreting the findings.

By six months from the index admission, 59 patients had died either in hospital or after discharge, while 183 had documented survival to the endpoint. This corresponds to a six-month all-cause mortality proportion of 24.4%.

Nonsurvivor classification included every documented death before completion of the six-month follow-up interval, irrespective of whether it occurred during the index stay or after discharge. Post-discharge vital status was determined from hospital records.

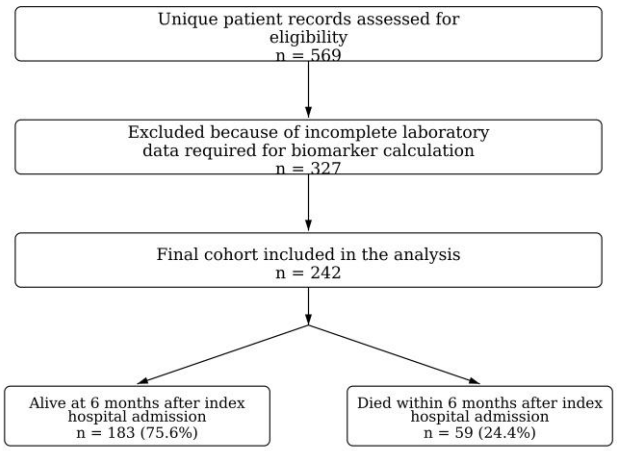

Figure 1. Patient selection and six-month outcome classification.

3.2 Baseline Clinical, Treatment, and Laboratory Characteristics

Table 1 compares baseline variables by six-month outcome. The survivor and nonsurvivor groups did not differ materially in age or sex. Mean age was 57.8 ± 13.4 years among survivors and 59.8 ± 15.8 years among nonsurvivors.

Table 1. Baseline clinical, treatment, and laboratory characteristics according to six-month vital status.

Variable	Survivors, n = 183	Nonsurvivors, n = 59	p value
Age, years	57.8 ± 13.4	59.8 ± 15.8	0.751
Male sex, n (%)	98 (53.6)	32 (54.2)	0.927
Laparotomy, n (%)	88 (74)	31 (26)	0.959

Variable	Survivors, n = 183	Nonsurvivors, n = 59	p value
Chemotherapy, n (%)	91 (82.7)	19 (17.3)	0.017
Radiotherapy, n (%)	6 (3.3)	2 (3.4)	0.967
Clinical nutrition consultation, n (%)	36 (58)	26 (42)	0.002
WBC, x10^3/uL	9.62 ± 4.32	12.09 ± 6.70	0.144
Neutrophils, %	63.1 ± 17.3	72.7 ± 17.6	0.481
Lymphocytes, %	24.4 ± 12.4	13.1 ± 7.8	0.41
Albumin, g/dL	3.70 ± 0.65	3.37 ± 1.09	0.001
NLR	4.38 ± 4.67	8.11 ± 6.03	0.084
dNLR	2.63 ± 2.45	4.10 ± 2.99	0.134
TLC, x10^3/uL	2.03 ± 0.84	1.35 ± 0.65	0.527

Abbreviations: dNLR, derived neutrophil-to-lymphocyte ratio; NLR, neutrophil-to-lymphocyte ratio; TLC, total lymphocyte count; WBC, white blood cell count. Values are presented as mean ± standard deviation or n (%). Percentages were calculated within each outcome group.

Laparotomy and radiotherapy frequencies were similar between outcome groups. Chemotherapy was documented more often among survivors, whereas consultation by the clinical nutrition team was more frequent among nonsurvivors. Nonsurvivors also had lower serum albumin. White blood cell count, neutrophil percentage, NLR, and dNLR were numerically higher in the nonsurvivor group, although these differences did not reach statistical significance.

3.3 Selected Clinical Factors and Nutrient Intake Improvement

Table 2 presents selected comorbidities, metastatic sites, complications, and changes in recorded intake. The comorbidity profile differed by outcome, with acute and chronic kidney disease accounting for larger proportions among nonsurvivors. No statistically significant association was observed between the distribution of documented metastatic sites and six-month mortality.

Complication patterns differed between groups, with sepsis, pleural effusion, and ascites occurring more prominently among nonsurvivors. Within the subgroup that had serial nutrition records, survivors more frequently showed increases in energy, protein, carbohydrate, and fat intake. These intake comparisons remain exploratory because only patients monitored by the clinical nutrition service contributed such data.

Table 2. Selected clinical factors and nutritional intake improvement according to six-month vital status.

Variable	Survivors	Nonsurvivors	p value
Comorbidity profile, n (%)	n = 39	n = 16	0.033
Hypertension	14 (35.9)	3 (18.8)	
Acute kidney injury	3 (7.7)	4 (25.0)	
Chronic kidney disease	3 (7.7)	3 (18.8)	
Metastatic site, n (%)	n = 24	n = 22	0.145
Liver	17 (70.8)	8 (36.4)	

Variable	Survivors	Nonsurvivors	p value
Lung	2 (8.3)	4 (18.2)	
Brain	0 (0.0)	4 (18.2)	
Clinical complication, n (%)	n = 21	n = 37	0.019
Pneumonia	4 (19.0)	6 (16.2)	
Sepsis	2 (9.5)	9 (24.3)	
Pleural effusion	2 (9.5)	8 (21.6)	
Ascites	0 (0.0)	4 (10.8)	
Nutritional intake improvement, n/N (%)	n = 36	n = 26	
Energy intake improved	33/36 (91.7)	14/26 (53.8)	0.001
Protein intake improved	35/36 (97.2)	18/26 (69.2)	0.002
Carbohydrate intake improved	32/36 (88.9)	12/26 (46.2)	<0.001
Fat intake improved	34/36 (94.4)	13/26 (50.0)	0.001

Abbreviations: AKI, acute kidney injury; CKD, chronic kidney disease. Values are presented as n (%) or n/N (%). Percentages for comorbidity profile, metastatic site, and clinical complication were calculated within the documented subgroup for each domain. Nutritional intake improvement was assessed only in patients with clinical nutrition monitoring records and should be interpreted as an exploratory monitoring variable.

3.4 ROC Analysis for Six-Month All-Cause Mortality

Table 3 summarizes the ROC analyses. Alb-dNLR separated survivors from nonsurvivors with statistically significant but limited discrimination (AUC 0.618). Using 1.5 as the threshold corresponds to classifying only patients with Alb-dNLR score 2 as test-positive. This produced a sensitivity of 20.3% and a specificity of 93.4%, a profile that favors confirmation of a high-risk state rather than broad case detection.

CEA and dNLR also produced statistically significant but weak AUCs. The albumin AUC was below 0.50 because lower, rather than higher, albumin values corresponded to the adverse biological direction used in interpretation. Among the nutrient-monitoring variables, changes in energy, carbohydrate, and fat intake showed significant discrimination, whereas the protein-intake measure did not. These exploratory intake results should be read cautiously because serial intake data were available only in patients who underwent clinical nutrition monitoring.

Table 3. Receiver operating characteristic curve analysis for six-month all-cause mortality after index hospital admission.

Variable	AUC	95% CI	Cut-off	Sensitivity, %	Specificity, %	Youden index	p value
Alb-dNLR score	0.618	0.533-0.703	1.5 (score 2)	20.3	93.4	0.137	0.007
CEA	0.607	0.522-0.693	4.79	79.7	42.9	0.226	0.013
dNLR	0.6	0.515-0.685	2.65	52.5	71.4	0.239	0.021
Albumin	0.319	0.239-0.400	2.95	52.5	19.8	-0.277	<0.001
Energy intake improvement	0.689	0.549-0.829	1.5	46.2	91.7	0.379	0.012

Variable	AUC	95% CI	Cut-off	Sensitivity, %	Specificity, %	Youden index	p value
Protein intake improvement	0.64	0.495-0.785	1.5	30.8	97.2	0.28	0.062
Carbohydrate intake improvement	0.714	0.577-0.850	1.5	53.8	88.9	0.427	0.004
Fat intake improvement	0.722	0.586-0.859	1.5	50	94.4	0.444	0.003

Abbreviations: Alb-dNLR, albumin-derived neutrophil-to-lymphocyte ratio; AUC, area under the curve; CEA, carcinoembryonic antigen; CI, confidence interval; dNLR, derived neutrophil-to-lymphocyte ratio. The albumin AUC should be read in light of its inverse risk direction, because lower concentrations indicate greater risk. The Alb-dNLR cut-off of 1.5 corresponds to score 2 on the 0-2 ordinal scale. Nutrient-intake variables compare the first clinical nutrition assessment with the final assessment before discharge or death.

3.5 Multivariable Logistic Regression Analysis

Results of the adjusted logistic model are reported in Table 4. Alb-dNLR was the only covariate that retained a statistically significant association with six-month mortality. Because the score was analyzed as an ordinal variable, a higher score corresponded to approximately 2.4 times greater odds of death within six months of admission per score increment. Neither CEA nor the exploratory nutrient-intake change variables remained independently associated with the outcome.

Table 4. Multivariable logistic regression analysis for six-month all-cause mortality after index hospital admission.

Variable	OR	95% CI	p value
Alb-dNLR score	2.405	1.124-5.149	0.024
CEA	1	1.00001-1.00002	0.871
Energy intake improvement	0.834	0.049-14.263	0.9
Protein intake improvement	2.294	0.114-46.268	0.588
Carbohydrate intake improvement	3.451	0.370-32.204	0.277
Fat intake improvement	6.574	0.920-46.965	0.061

Abbreviations: Alb-dNLR, albumin-derived neutrophil-to-lymphocyte ratio; CEA, carcinoembryonic antigen; CI, confidence interval; OR, odds ratio. Alb-dNLR was modeled as an ordinal score; therefore, its OR represents the effect per 1-point score increase. CEA was modeled continuously; because the estimate represents a 1-unit increment, its OR rounds to approximately 1.000. Nutrient-intake change variables were exploratory covariates and were evaluated in complete cases with available nutrition-monitoring data.

3.6 Discussion

Across the admission variables evaluated in this cohort, Alb-dNLR was the only measure that remained independently associated with six-month mortality. Its AUC was modest, so it should not be interpreted as a high-performance predictive test. The marked specificity at the selected threshold is more consistent with a rule-in role: an Alb-dNLR score of 2 may identify a subgroup with pronounced inflammatory-nutritional vulnerability, but lower scores cannot reliably exclude subsequent death [16].

The clinical timing distinguishes this analysis from much of the earlier literature. Prior work on albumin-neutrophil indices has largely addressed response to therapy, postoperative outcomes, or longer-term prognosis [9-14]. Here, measurement began during hospitalization and the endpoint extended six months beyond the index admission, capturing deaths during the index

stay and deaths later documented in the medical record. This fixed six-month window was selected to reflect early post-hospital vulnerability rather than long-term cancer survival.

A mechanistic link between Alb-dNLR and early mortality is plausible within the framework already described for cancer-related inflammation and nutritional decline [1-4]. Hospitalization may coincide with infection, obstruction, fluid shifts, organ dysfunction, diminished oral intake, and catabolic stress. The score combines two signals that may become abnormal under these conditions: reduced albumin-associated reserve and neutrophil-dominant systemic inflammation.

Serum albumin was significantly lower in nonsurvivors, yet it did not retain an independent association after adjustment. This is not unexpected because albumin integrates several processes beyond nutrient intake, including inflammation, hepatic synthesis, intravascular volume, and capillary permeability [5-7]. Interpreting albumin together with an inflammatory measure may therefore provide more clinical context than relying on albumin alone.

dNLR provides a compact representation of neutrophil-predominant inflammation relative to the non-neutrophil leukocyte pool, and neutrophil-based indices have been linked to inflammatory burden and impaired antitumor immune activity [8]. In the present cohort, dNLR alone showed only weak discrimination, whereas the combined Alb-dNLR score retained the adjusted association. This pattern suggests that simultaneous inflammatory activation and low albumin may be more informative for short-term vulnerability than either component considered separately.

CEA had weak discriminatory ability and lost significance in the multivariable model. The marker remains important in CRC evaluation and surveillance, but it represents a different biological domain from the host-response features captured by Alb-dNLR. In this inpatient cohort, the adjusted results therefore suggest that six-month mortality was more closely associated with the combined inflammatory-reserve phenotype than with CEA alone.

The sub-0.50 AUC observed for albumin is primarily a coding-direction issue. Lower albumin corresponds to greater clinical risk, whereas conventional ROC output assumes that higher test values indicate the positive state unless the direction is reversed. The albumin result should consequently be interpreted according to the expected biological direction rather than as evidence that albumin is clinically irrelevant.

Changes in nutrient intake were associated with outcome in several unadjusted ROC analyses but did not persist as independent predictors. These variables are particularly vulnerable to reverse causation and confounding by clinical severity: intake can deteriorate because of obstruction, infection, procedures, gastrointestinal symptoms, instability, or proximity to death, and it can improve after nutritional intervention. They are therefore best treated as markers of the inpatient clinical course rather than as fixed baseline exposures. The higher frequency of nutrition-team consultation among nonsurvivors may similarly represent confounding by indication, because referral is more likely in patients who are clinically or nutritionally compromised.

The association of complications such as sepsis, pleural effusion, and ascites with mortality further illustrates that early post-admission outcome is multifactorial. Tumor burden, infection, inflammatory stress, organ dysfunction, treatment effects, and nutritional depletion may coexist and interact. Alb-dNLR should thus complement, not replace, tumor staging, complication assessment, treatment planning, and clinical judgment.

Several features strengthen the study: it reflects routine inpatient practice, applies an explicit patient-selection pathway, compares multiple readily available markers, and extends outcome ascertainment beyond discharge to a fixed six-month endpoint. Together, these elements make the analysis relevant to pragmatic admission-based risk assessment.

The findings require substantial caution. The study was retrospective and conducted at a single center, limiting external validity and precluding causal inference. Excluding 327 records with incomplete biomarker data creates potential selection bias because patients with complete laboratory documentation may differ systematically from excluded patients. Residual confounding is also likely: tumor stage, molecular subtype, ECOG performance status, treatment intent, chemotherapy regimen, radiotherapy indication, infection severity, organ dysfunction, and timing of interventions were not consistently available. Although post-discharge vital status could be established from the medical record, exact dates of death were incomplete, which prevented time-to-event analysis. Finally, serial intake data were confined to patients seen by the clinical nutrition service and may reflect documentation practices, timing, illness severity, and closeness to death as much as nutritional change itself. For these reasons, Alb-dNLR should be considered an adjunctive risk marker requiring external prospective validation rather than a tool ready for stand-alone routine implementation.

4. CONCLUSION

Among hospitalized patients with CRC, a higher admission Alb-dNLR score was independently associated with all-cause mortality within six months. The score's high specificity at the selected cut-off favors use as an adjunct for identifying patients with a pronounced inflammatory-nutritional risk phenotype rather than as a screening instrument. External prospective validation is needed to determine whether Alb-dNLR adds useful prognostic information when combined with tumor stage, performance status, treatment intent, comorbidity burden, complication severity, and validated nutritional assessment methods.

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AUTHOR CONTRIBUTIONS

Pina: Study conception and design, data collection, statistical analysis, interpretation of results, manuscript drafting, critical revision, and approval of the final manuscript.

Agussalim Bukhari: Supervision, conceptualization, methodology, and critical revision.

Suryani As'ad: Supervision, methodology, and manuscript review.

Nurpudji Astuti Daud: Validation, formal analysis, and manuscript review.

Marniar: Validation, methodology, and manuscript review.

Andi Faradilah: Validation, investigation, and manuscript review.

All authors read and approved the final manuscript.

CONFLICT OF INTEREST

No author reports a conflict of interest related to this work.

ETHICS STATEMENT

Ethical approval was granted by the Ethics Committee of the Faculty of Medicine, Hasanuddin University, Makassar, Indonesia (No. 785/UN4.6.4.5.31/PP36/2025; valid October 14th 2025 to October 14th 2026). The study conformed to the Declaration of Helsinki. Because the analysis used anonymized retrospective medical-record data, the committee waived the requirement for written informed consent.

DATA AVAILABILITY STATEMENT

The dataset is not publicly posted because it originates from hospital medical records and includes sensitive clinical information. A de-identified dataset may be considered for sharing by the corresponding author upon reasonable request, subject to institutional authorization.

REFERENCES

[1] Bray, F., Laversanne, M., Sung, H., Ferlay, J., Siegel, R.L., Soerjomataram, I., et al., 2024. Global cancer statistics 2022: GLOBOCAN estimates of incidence and mortality worldwide for 36 cancers in 185 countries. *CA Cancer Journal for Clinicians* 74(3), 229–263. https://doi.org/10.3322/caac.21834.

[2] Siegel, R.L., Miller, K.D., Wagle, N.S., Jemal, A., 2023. Cancer statistics, 2023. *CA Cancer Journal for Clinicians* 73(1), 17–48. https://doi.org/10.3322/caac.21763.

[3] Dekker, E., Tanis, P.J., Vleugels, J.L.A., Kasi, P.M., Wallace, M.B., 2019. Colorectal cancer. *The Lancet* 394(10207), 1467–1480. https://doi.org/10.1016/S0140-6736(19)32319-0.

[4] Hanahan, D., 2022. Hallmarks of cancer: new dimensions. *Cancer Discovery* 12(1), 31–46. https://doi.org/10.1158/2159-8290.CD-21-1059.

[5] Muscaritoli, M., Arends, J., Bachmann, P., Baracos, V., Barthelemy, N., Bertz, H., et al., 2021. ESPEN practical guideline: Clinical Nutrition in cancer. *Clinical Nutrition* 40(5), 2898–2913. https://doi.org/10.1016/j.clnu.2021.02.005.

[6] Cederholm, T., Jensen, G.L., Correia, M.I.T.D., Gonzalez, M.C., Fukushima, R., Higashiguchi, T., et al., 2019. GLIM criteria for the diagnosis of malnutrition: a consensus report from the global clinical nutrition community. *Clinical Nutrition* 38(1), 1–9. https://doi.org/10.1016/j.clnu.2018.08.002.

[7] Schuetz, P., Fehr, R., Baechli, V., Geiser, M., Deiss, M., Gomes, F., et al., 2019. Individualised nutritional support in medical inpatients at nutritional risk: a randomised clinical trial. *The Lancet* 393(10188), 2312–2321. https://doi.org/10.1016/S0140-6736(18)32776-4.

[8] Templeton, A.J., McNamara, M.G., Seruga, B., Vera-Badillo, F.E., Aneja, P., Ocana, A., et al., 2014. Prognostic role of neutrophil-to-lymphocyte ratio in solid tumors: a systematic review and meta-analysis. *Journal of the National Cancer Institute* 106(6), dju124. https://doi.org/10.1093/jnci/dju124.

[9] Pan, Z., Wang, Y., Li, S., Cai, H., Guan, G., 2024. The prognostic role of the change in albumin-derived neutrophil-to-lymphocyte ratio during neoadjuvant chemoradiotherapy in patients with locally advanced rectal cancer. *Biomolecules and Biomedicine* 24(3), 612–624. https://doi.org/10.17305/bb.2023.9787.

[10] Nakazawa, N., Sohda, M., Tateno, K., Endo, Y., Sano, A., Sakai, M., et al., 2023. Albumin-derived neutrophil-to-lymphocyte ratio score as a marker of nivolumab treatment sensitivity in gastric cancer: a multicenter study. *In Vivo* 37(2), 818–824. https://doi.org/10.21873/invivo.13147.

[11] Wang, J., Chang, C., Lai, C., Hung, Y., Chou, W., 2025. The predictive role of albumin and neutrophil-to-lymphocyte ratio score on postoperative outcomes and survival in older patients with colorectal cancer. *Cancer Control* 32, 10732748251374410. https://doi.org/10.1177/10732748251374410.

[12] Xiu, W.J., Yang, H.T., Zheng, Y.Y., Ma, Y.T., Xie, X., 2022. ALB-dNLR score predicts mortality in coronary artery disease patients after percutaneous coronary intervention. *Frontiers in Cardiovascular Medicine* 9, 709868. https://doi.org/10.3389/fcvm.2022.709868.

[13] Onuma, S., Hashimoto, I., Suematsu, H., Takeno, S., Yamashita, R., Hara, K., et al., 2023. Clinical effects of the neutrophil-to-lymphocyte ratio/serum albumin ratio in patients with gastric cancer after gastrectomy. *Journal of Personalized Medicine* 13(3), 432. https://doi.org/10.3390/jpm13030432.

[14] Miyata, T., Hayama, T., Ozawa, T., Nozawa, K., Misawa, T., Fukagawa, T., 2024. Predicting prognosis in colorectal cancer patients with curative resection using albumin, lymphocyte count and RAS mutations. *Scientific Reports* 14(1), 14428. https://doi.org/10.1038/s41598-024-65457-8.

[15] von Elm, E., Altman, D.G., Egger, M., Pocock, S.J., Gotzsche, P.C., Vandenbroucke, J.P., 2007. The Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) statement: guidelines for reporting observational studies. *The Lancet* 370(9596), 1453–1457. https://doi.org/10.1016/S0140-6736(07)61602-X.

[16] Parikh, R., Mathai, A., Parikh, S., Sekhar, G.C., Thomas, R., 2008. Understanding and using sensitivity, specificity and predictive values. *Indian Journal of Ophthalmology* 56(1), 45–50. https://doi.org/10.4103/0301-4738.37595.