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Prognostic Significance of Sarcopenic Obesity in Colorectal Cancer Surgery: A Retrospective Cohort Study

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ARTICLE INFO

Article history:
Received 7 December 2025
Received in revised form
21 March 2026
Accepted 2 April 2026
Available online 8 May 2026

Keywords:
Body composition
Computed tomography
Colorectal cancer
Prognosis
Sarcopenic obesity

ABSTRACT

Introduction: Sarcopenic obesity (SO), the coexistence of reduced skeletal muscle mass and excess visceral fat, represents a distinct metabolic phenotype increasingly recognized in surgical oncology. Its prognostic role in colorectal cancer (CRC) surgery remains insufficiently defined, particularly in the Asian populations.

Methods: In this retrospective cohort study, 923 patients undergoing curative CRC resection between 2017 and 2023 were evaluated. Preoperative computed tomography at the L3 level quantified skeletal muscle index and visceral fat area. Sarcopenia and visceral obesity were defined according to the Asian Working Group for Sarcopenia and Japanese criteria, respectively. Patients were classified into four phenotypes: normal, obesity, sarcopenia, and SO. Postoperative complications, overall survival (OS), and recurrence-free survival (RFS) were analyzed using logistic regression, Kaplan–Meier, and Cox proportional hazards models.

Results: The incidence of postoperative complications increased progressively from normal (11.9%) to obesity (17.3%), sarcopenia (20.5%), and SO (27.5%) ($P < 0.001$). SO independently predicted both overall (odds ratio = 2.56, $P = 0.003$) and severe (odds ratio = 3.21, $P = 0.004$) complications. Patients with SO had the poorest 5-y OS and RFS (log rank $P < 0.001$), confirmed by multivariable Cox analysis (OS: hazard ratio = 1.92; RFS: hazard ratio = 1.87).

Conclusions: Sarcopenic obesity independently predicts postoperative morbidity and adverse long-term survival following curative CRC surgery. Incorporating computed tomography–derived body composition assessment into routine preoperative evaluation may improve risk stratification and enable targeted prehabilitation, ultimately enhancing surgical and oncologic outcomes.

Introduction

As one of the leading causes of cancer morbidity and mortality worldwide, colorectal cancer (CRC) poses a serious health challenge.[1,2] Global Cancer Observatory 2022 reported nearly 20 million new cancer cases and 9.7 million deaths globally, of which CRC accounted for roughly 9.6% and 9.3%, respectively, making it the third most common cancer and the second leading cause of cancer-related death.[3] Although substantial progress has been made in minimally invasive techniques,

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0022-4804/$ – see front matter
https://doi.org/10.1016/j.jss.2026.04.007

precision surgery, and perioperative protocols such as enhanced recovery after surgery, postoperative complications continue to occur in 20%-30% of patients, with significant consequences for recovery, quality of life, and long-term survival.[4,5] Thus, identifying preoperative predictors of surgical risk is essential for guiding perioperative management and developing tailored therapeutic strategies.

In recent years, body composition has emerged as an important determinant of postoperative outcomes in CRC. Obesity, particularly central or visceral obesity, has been linked to increased technical difficulty during surgery, greater blood loss, prolonged operative duration, and a higher incidence of postoperative complications.[6-8] However, the conventional reliance on body mass index (BMI) may fail to capture the actual burden of visceral adiposity, especially in the Asian populations. Instead, visceral fat area (VFA) measured by imaging has been regarded as a more accurate parameter.[9,10] In parallel, sarcopenia, defined as a progressive decline in skeletal muscle mass and strength, has been identified as an independent risk factor for poor outcomes such as infectious complications, prolonged hospitalization, and diminished survival.[11-13] Evidence regarding the combined effect of obesity and sarcopenia on surgical outcomes in CRC remains scarce, particularly in the Asian cohorts.

Within this context, sarcopenic obesity (SO), the coexistence of excess adiposity and muscle depletion, has drawn an increasing interest. Preliminary findings suggest that SO may confer a compounded risk for adverse outcomes following CRC surgery, though its prognostic value has not been fully clarified. With the global prevalence of both obesity and sarcopenia steadily rising, early identification of SO patients may enable more precise perioperative optimization and improve both short-term recovery and long-term prognosis. Therefore, the present study aims to assess the prognostic significance of SO in short-term outcomes after CRC resection, providing evidence to support refined risk stratification and individualized perioperative care.

Methods

Patients

We retrospectively reviewed CRC patients who underwent curative resection at the Chongqing University Fuling Hospital between 2017 and 2023. Curative resection was defined as R0 resection with no residual tumor and negative surgical margins confirmed by postoperative pathological examination. Only those who had abdominal computed tomography (CT) scans within 30 d before surgery were eligible. Patients were excluded if they had undergone noncurative surgery, had another primary malignancy, lacked preoperative CT scans within 30 d, or had incomplete follow-up information.

This study was approved by the Ethics Committee of Chongqing University Fuling Hospital (approval no. 2025CDFSFLYYEC-70) and conducted in accordance with the Declaration of Helsinki. Due to the retrospective nature of the study, the requirement for informed consent was waived.

Definition of SO

SO was defined as the combination of sarcopenia and obesity. Preoperative CT images were analyzed using Slice-O-Matic software. The axial image at the mid L3 level was chosen for measurement of skeletal muscle area and VFA.

Skeletal muscle assessment: Skeletal muscle was segmented using a Hounsfield unit range of −29 to 150 (Fig. 1). The skeletal muscle index (SMI) was calculated as skeletal muscle area at L3 divided by height squared (cm^2/m^2). Sarcopenia was diagnosed when SMI was <40.8 cm^2/m^2 for men or <34.9 cm^2/m^2 for women, according to the previously published thresholds.[14]

Visceral obesity assessment: VFA was identified using a Hounsfield unit range of −150 to −50. Based on the Japanese criteria for metabolic syndrome, visceral obesity was defined as $>$ 130 cm^2 in men and >90 cm^2 in women.[15]

Patients fulfilling criteria for both sarcopenia and visceral obesity were diagnosed with SO.

Data collection

Clinical characteristics, pathological findings, and perioperative details were collected. Postoperative complications were graded according to the Clavien–Dindo classification, with grade III or higher considered severe. Overall survival (OS) and recurrence-free survival (RFS) were documented during follow-up.

Statistical analysis

All analyses were conducted using SPSS version 26.0 (IBM Corp.) and R software (v4.2.1). The normality of continuous variables was assessed using the Shapiro–Wilk test, and homogeneity of variance was evaluated using Levene's test. All continuous variables were found to be approximately normally distributed ($P \geq 0.05$). Continuous variables were expressed as mean ± standard deviation and compared using one-way analysis of variance. Categorical variables were analyzed using the χ^2 test or Fisher's exact test, as appropriate. Logistic regression analysis was performed to identify

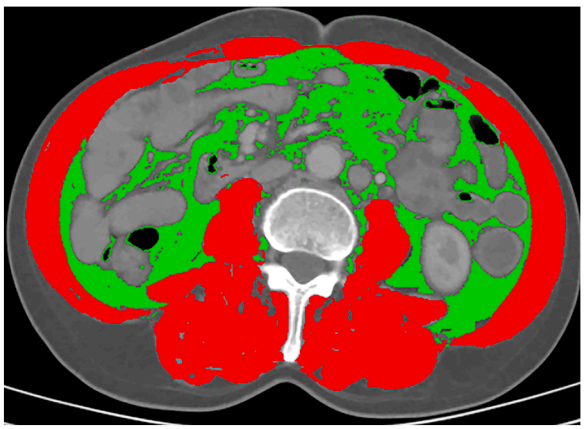

Fig. 1 – CT images illustrating the identification of skeletal muscle and visceral fat areas. Skeletal muscle is marked in red, while visceral fat is marked in green.

Table 1 – Patient baseline features according to body composition classification.

Variables	Normal (369)	Obesity (231)	Sarcopenia (185)	Sarcopenic obesity (138)	P value
Age, y	63.5 ± 7.9	64.3 ± 8.0	71.6 ± 7.6	73.2 ± 6.8	<0.001
BMI, kg/m^2	22.6 ± 2.8	29.2 ± 2.9	21.0 ± 2.5	28.7 ± 3.5	<0.001
SMI, cm^2/m^2	48.2 ± 6.1	50.5 ± 6.5	32.9 ± 3.0	32.5 ± 3.2	<0.001
VFA, cm^2	75.2 ± 10.5	168.2 ± 32.5	72.7 ± 12.1	172.6 ± 35.7	<0.001
Handgrip strength, mean (SD), kg	25.3 ± 9.8	27.5 ± 10.2	18.7 ± 6.7	20.1 ± 7.1	<0.001
Gait speed, mean (SD), m/s	0.9 ± 0.2	1.0 ± 0.2	0.8 ± 0.2	0.8 ± 0.2	<0.001
Gender, male	193 (52.3)	130 (56.3)	102 (55.1)	78 (56.5)	0.736
ASA III-IV	54 (14.6)	36 (15.6)	30 (16.2)	22 (15.9)	0.960
Diabetes	30 (8.1)	58 (25.0)	19 (10.3)	45 (32.6)	<0.001
Hypertension	36 (9.8)	60 (26.0)	21 (11.4)	55 (39.9)	<0.001
Abdominal surgery history	36 (9.8)	27 (11.7)	14 (7.6)	15 (10.9)	0.553
TNM stage					0.908
I	76 (20.6)	44 (19.0)	40 (21.6)	25 (18.1)	
II	108 (29.3)	73 (31.6)	50 (27.0)	46 (33.3)	
III	185 (50.1)	114 (49.4)	95 (51.4)	67 (48.6)	
Open surgery	40 (10.8)	27 (11.7)	28 (15.1)	16 (11.6)	0.526
Tumor size >5 cm	137 (37.1)	84 (36.4)	72 (38.9)	55 (39.9)	0.870
Rectum tumor	129 (35.0)	81 (35.1)	65 (35.1)	48 (34.8)	1.000
Combined resection	34 (9.2)	22 (9.5)	17 (9.2)	18 (13.0)	0.600
Undifferentiated histology	86 (23.3)	53 (22.9)	44 (23.8)	40 (29.0)	0.552
Neoadjuvant chemotherapy	17 (9.2)	15 (11.7)	16 (15.2)	15 (17.6)	0.648
Adjuvant chemotherapy	109 (59.2)	75 (58.6)	56 (53.3)	40 (47.1)	0.371

ASA = American Society of Anesthesiologists; TNM = tumor-node-metastasis classification; BMI = body mass index; SMI = skeletal muscle index; VFA = visceral fat area; SD = standard deviation.

predictors of postoperative complications. OS and RFS were estimated using the Kaplan–Meier method, and differences were compared using the log rank test. Univariable Cox proportional hazards analyses were first performed to identify potential prognostic factors. Variables with a P value of < 0.05 in the univariable analyses were subsequently included in the multivariable Cox proportional hazards models to determine independent prognostic factors. All statistical tests were two-sided, and $P < 0.05$ was considered statistically significant.

Results

Patient characteristics

A total of 923 patients undergoing curative CRC resection were included and categorized into four body composition groups: normal (N, $n = 369$), obesity without sarcopenia (O, $n = 231$), sarcopenia (S, $n = 185$), and SO (SO, $n = 138$). Baseline demographic and clinical features are summarized in Table 1. Compared with the other groups, patients with S or SO were significantly older and exhibited markedly lower SMI (all $P < 0.001$). Conversely, those in the O and SO groups presented higher BMI and VFA (both $P < 0.001$). The prevalence of diabetes and hypertension increased progressively from the N to SO group (both $P < 0.001$).

No significant intergroup differences were observed in sex, American Society of Anesthesiologists (ASA) classification, previous abdominal surgery, tumor-node-metastasis (TNM) stage, tumor size or site, histologic type, operative approach, combined resection, or perioperative chemotherapy (all $P > 0.05$).

Postoperative complications

As presented in Table 2, the overall postoperative complication rates were 11.9%, 17.3%, 20.5%, and 27.5% in the N, O, S, and SO groups, respectively ($P < 0.001$). Patients with SO had the highest incidence of bowel obstruction, infectious, and urinary complications.

Severe complications (Clavien–Dindo grades III-V) occurred in 8.7% of the SO group, compared with 3.0% in the N group ($P < 0.001$). These findings indicate a synergistic detrimental effect of excessive adiposity and reduced muscle mass on postoperative morbidity.

Predictors of postoperative complications

The results of univariable and multivariable logistic regression analyses are summarized in Tables 3 and 4. In multivariable analysis, ASA III-IV status (odds ratio [OR] = 1.58, 95% confidence interval [CI]: 1.01-2.77, $P = 0.047$), diabetes (OR = 2.89, 95% CI: 1.45-5.75, $P = 0.003$), open surgery (OR = 1.83, 95% CI: 1.02-3.29, $P = 0.042$), combined organ resection (OR = 2.68, 95% CI: 1.21-5.93, $P = 0.015$), and body composition category were identified as independent predictors of overall postoperative complications. Relative to the

Table 2 – Postoperative complications across different body composition groups.

Complications	Normal (369)	Obesity (231)	Sarcopenia (185)	Sarcopenic obesity (138)	P value
Total	44 (11.9)	40 (17.3)	38 (20.5)	38 (27.5)	<0.001
Bowel obstruction					
Postoperative ileus/delayed bowel recovery	13 (3.5)	11 (4.8)	11 (5.9)	13 (9.4)	
Anastomotic leakage	3 (0.8)	3 (1.3)	3 (1.6)	4 (2.9)	
Anastomotic stenosis	1 (0.3)	1 (0.4)	1 (0.5)	1 (0.7)	
Anastomotic bleeding	2 (0.5)	2 (0.9)	2 (1.1)	1 (0.7)	
Infectious complications					
Intra-abdominal infection/abscess	4 (1.1)	4 (1.7)	4 (2.2)	4 (2.9)	
Pulmonary infection	8 (2.2)	7 (3.0)	7 (3.8)	6 (4.3)	
Wound infection	4 (1.1)	3 (1.3)	3 (1.6)	3 (2.2)	
Urinary complications					
Urinary tract infection/retention	4 (1.1)	4 (1.7)	3 (1.6)	3 (2.2)	
Other complications					
Lymphorrhagia	1 (0.3)	1 (0.4)	1 (0.5)	1 (0.7)	
Venous thromboembolism	2 (0.5)	2 (0.9%)	2 (1.1)	1 (0.7)	
Cardiovascular events	2 (0.5)	2 (0.9)	1 (0.5)	1 (0.7)	
Clavien-Dindo grade					<0.001
I-II	33 (8.9)	30 (13.0)	28 (15.1)	25 (18.8)	
III-V	11 (3.0)	10 (4.3)	10 (5.4)	13 (8.7)	

ASA = American Society of Anesthesiologists; TNM = tumor-node-metastasis classification.

normal group, obesity (OR = 1.92, $P = 0.049$), sarcopenia (OR = 2.04, $P = 0.025$), and SO (OR = 2.56, $P = 0.003$) were all associated with higher complication risk.

For severe postoperative morbidity (Clavien–Dindo III-V), independent risk factors included ASA III-IV (OR = 1.96, $P = 0.046$), diabetes (OR = 2.47, $P = 0.019$), open surgery (OR = 2.25, $P = 0.027$), and combined resection (OR = 3.02, $P = 0.009$). Using the normal group as the reference, SO exerted the strongest adverse effect (OR = 3.21, 95% CI: 1.45-7.09, $P = 0.004$), followed by sarcopenia (OR = 2.24, $P = 0.042$) and obesity (OR = 2.17, $P = 0.048$), demonstrating a graded dose–response relationship between body composition deterioration and postoperative outcomes.

Overall survival

Survival by body composition

The median follow-up time was 37.3 mo (interquartile range 21.9-52.7 mo), with no significant difference among the body composition groups. Kaplan–Meier analysis revealed significant survival disparities across the four body composition groups (Fig. 2A). The SO cohort exhibited the poorest 5-y OS, followed by the sarcopenia group, whereas patients with normal composition achieved the most favorable outcomes (log rank $P < 0.001$).

Survival by TNM stage, combined resection, and histologic differentiation

As shown in Figure 2B-D, TNM stage, combined organ resection, and histologic differentiation were all significantly associated with OS (log rank $P < 0.001$). Patients with stage III disease, combined resection, or undifferentiated carcinoma experienced markedly worse survival. In the multivariable Cox regression analysis (Table 5), these variables, together with body composition, remained independent prognostic determinants of OS.

Disease-free survival

Survival by body composition

Trends for RFS paralleled those observed for OS (Fig. 3A). Both the SO and S groups had significantly lower RFS compared with the normal group (log rank $P < 0.001$).

Survival by TNM stage, combined resection, and histologic differentiation

Kaplan–Meier curves for TNM stage, combined resection, and histologic differentiation (Fig. 3B-D) also demonstrated significant differences in RFS (log rank $P < 0.001$). Patients with stage III disease, combined resection, or undifferentiated carcinoma exhibited the shortest RFS intervals. Consistent with the Cox proportional hazards model (Table 6), TNM III stage, combined resection, and undifferentiated histology independently predicted inferior RFS.

Discussion

This retrospective cohort study provides robust evidence that SO, the coexistence of diminished skeletal muscle mass and

Table 3 – Univariable and multivariable logistic regression analyses of risk factors for postoperative complications.

Variables	Univariable analysis			Multivariate analysis		
	OR	95% CI	P value	OR	95% CI	P value
Age, y	1.02	0.99-1.05	0.186			
Gender						
Male	1					
Female	0.93	0.62-1.42	0.759			
ASA						
I-II	1			1		
III-IV	1.72	1.05-2.83	0.031	1.58	1.01-2.77	0.047
Diabetes						
No	1			1		
Yes	2.53	1.54-4.12	<0.001	2.89	1.45-5.75	0.003
Hypertension						
No	1					
Yes	1.14	0.70-1.89	0.606			
Abdominal surgery history						
No	1					
Yes	1.26	0.67-2.34	0.478			
TNM stage						
I	1					
II	1.11	0.59-2.06	0.752			
III	1.19	0.67-2.09	0.542			
Operation method						
Laparoscopy	1			1		
Open	1.96	1.07-3.58	0.028	1.83	1.02-3.29	0.042
Tumor size						
≤5 cm	1					
>5 cm	1.10	0.62-1.95	0.743			
Tumor location						
Colon	1					
Rectum	1.23	0.72-2.10	0.447			
Combined resection						
No	1			1		
Yes	3.02	1.59-5.72	<0.001	2.68	1.21-5.93	0.015
Histologic type						
Differentiated	1					
Undifferentiated	1.24	0.66-2.33	0.502			
Neoadjuvant chemotherapy						
No	1			1		
Yes	2.11	1.14-3.93	0.019	1.52	0.67-3.44	0.316
Adjuvant chemotherapy						
No	1					
Yes	1.02	0.65-1.61	0.921			
Sarcopenic obesity						
Normal	1			1		
Obesity	2.37	1.28-4.38	0.006	1.92	1.00-3.70	0.049
Sarcopenia	2.21	1.27-3.83	0.005	2.04	1.09-3.84	0.025
Sarcopenic obesity	4.52	2.47-8.25	<0.001	2.56	1.36-4.81	0.003

ASA = American Society of Anesthesiologists; TNM = tumor-node-metastasis classification; OR = odds ratio; CI = confidence interval.

Table 4 – Univariable and multivariable logistic regression analyses of risk factors for severe postoperative complications.

Variables	Univariable analysis			Multivariate analysis		
	OR	95% CI	*P* value	OR	95% CI	*P* value
Age, y	1.03	1.00-1.07	0.048	1.02	0.99-1.06	0.136
Gender						
Male	1					
Female	0.91	0.52-1.58	0.737			
ASA						
I-II	1			1		
III-IV	2.31	1.23-4.36	0.009	1.96	1.01-3.81	0.046
Diabetes						
No	1			1		
Yes	2.82	1.48-5.36	0.002	2.47	1.16-5.23	0.019
Hypertension						
No	1					
Yes	1.27	0.67-2.42	0.460			
Abdominal surgery history						
No	1					
Yes	1.34	0.62-2.90	0.458			
TNM stage						
I	1					
II	1.15	0.53-2.48	0.724			
III	1.36	0.70-2.65	0.362			
Operation method						
Laparoscopy	1			1		
Open	2.61	1.31-5.20	0.006	2.25	1.10-4.61	0.027
Tumor size						
≤5 cm	1					
>5 cm	1.28	0.66-2.46	0.467			
Tumor location						
Colon	1					
Rectum	1.39	0.71-2.74	0.336			
Combined resection						
No	1			1		
Yes	3.95	1.84-8.49	<0.001	3.02	1.32-6.90	0.009
Histologic type						
Differentiated	1					
Undifferentiated	1.22	0.58-2.56	0.593			
Neoadjuvant chemotherapy						
No	1			1		
Yes	2.44	1.13-5.24	0.023	1.88	0.77-4.56	0.164
Adjuvant chemotherapy						
No	1					
Yes	0.95	0.53-1.69	0.855			
Sarcopenic obesity						
Normal	1			1		
Obesity	2.85	1.35-6.01	0.006	2.17	1.01-4.65	0.048
Sarcopenia	2.62	1.23-5.58	0.013	2.24	1.03-4.86	0.042
Sarcopenic obesity	5.84	2.71-12.58	<0.001	3.21	1.45-7.09	0.004

ASA = American Society of Anesthesiologists; TNM = Tumor-Node-Metastasis classification; OR = odds ratio; CI = confidence interval.

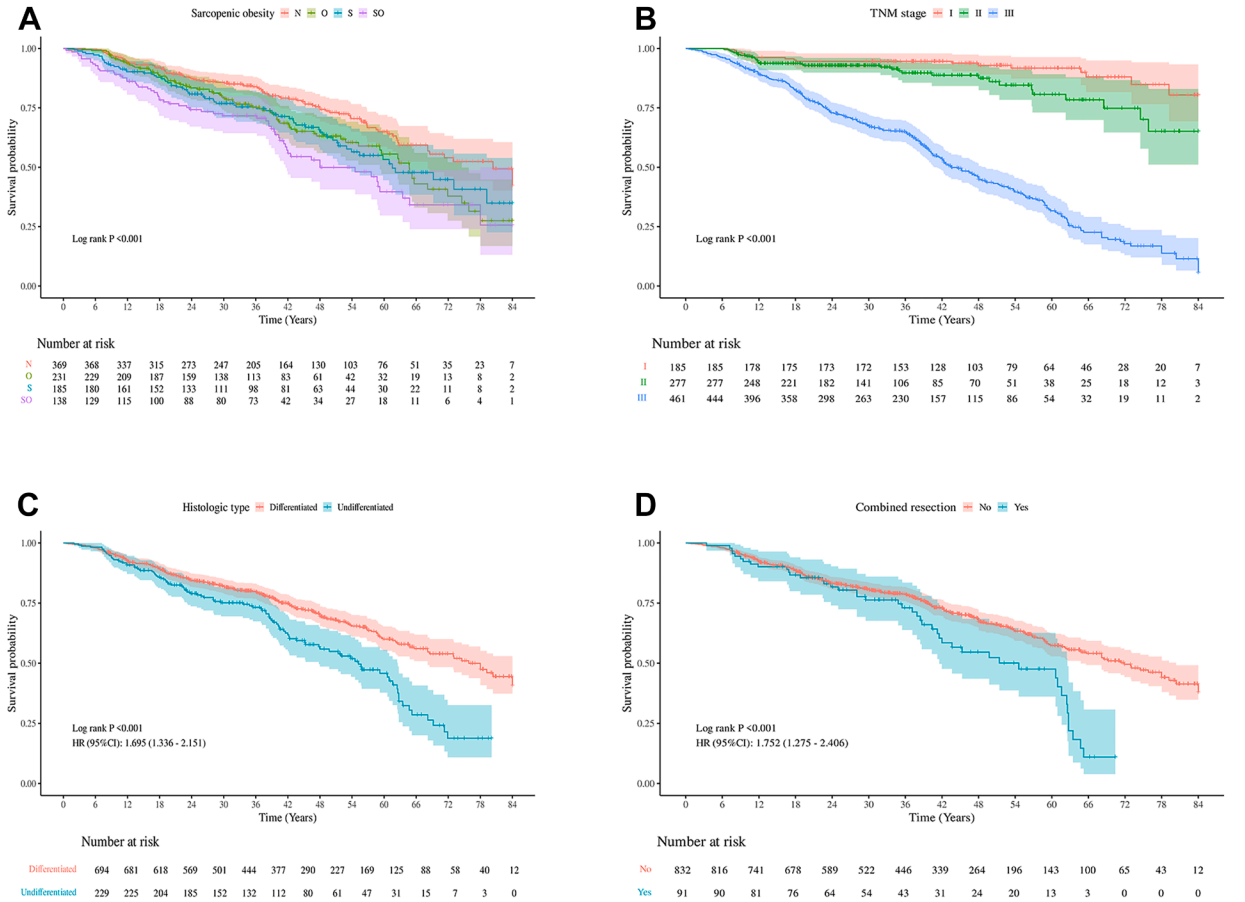

Fig. 2 – Kaplan–Meier survival curves for OS across various clinical subgroups. (A) OS stratified by body composition categories (normal, obesity, sarcopenia, and sarcopenic obesity); (B) OS stratified by TNM stage; (C) OS stratified by histological type; (D) OS comparison between patients undergoing combined resection and standard surgery. OS = overall survival.

excessive adiposity, represents a distinct and clinically significant metabolic phenotype in patients undergoing curative resection for CRC. Among the four body composition phenotypes analyzed, SO was associated with the highest incidence of postoperative complications, particularly intestinal, infectious, and urinary events, and with the poorest OS and RFS. These findings suggest that aberrant body composition is not merely a manifestation of malnutrition but a biologically active state influencing systemic metabolism, immune competence, and tumor behavior. Integrating quantitative CT-based body composition profiling with functional muscle assessment may therefore refine perioperative risk stratification and guide individualized management.

The adverse prognostic implications of abnormal body composition in CRC have been consistently documented. Sarcopenia alone has been linked to increased infectious morbidity, prolonged hospitalization, and reduced long-term survival. Lieffers *et al.* demonstrated that preoperative sarcopenia independently predicted postoperative infection and delayed recovery following CRC surgery,[16] while Trejo-Avila *et al.* confirmed through meta-analysis that sarcopenia significantly decreases both OS and disease-free survival.[17] Visceral obesity, in turn, has been correlated with greater technical difficulty, prolonged operative time, and higher anastomotic leakage risk.[18] However, the combined state of sarcopenia and visceral adiposity, SO, has been comparatively underexplored in CRC. Hopkins *et al.*[19] reported that SO independently predicted worse long-term survival, and Cespedes Feliciano *et al.*[20] demonstrated in the large C-SCANS cohort that SO was associated with approximately 45% higher CRC-specific mortality. These findings, corroborated by Kroenke and Caan's CT-based analysis[21] and the multicenter prospective work of Dolan *et al.*,[18] collectively confirm that progressive deterioration from normal composition to obesity, sarcopenia, and ultimately SO confers a graded increase in perioperative and oncologic risk. Our results indicate that SO is an independent determinant of both short- and long-term outcomes in CRC. Beyond short-term surgical morbidity, accumulating evidence highlights the enduring oncologic relevance of SO. Multiple large-scale and multicenter investigations consistently identify SO as an independent predictor of reduced OS and RFS after CRC surgery. This convergence of evidence, coupled with our findings, underscores SO as a robust prognostic marker and warrants

Table 5 — Univariable and multivariable cox regression analyses of prognostic factors for overall survival.

Variables	Univariable analysis			Multivariate analysis		
	HR	95% CI	*P* value	HR	95% CI	*P* value
Age, y	1.01	0.98-1.04	0.423			
Gender						
Male	1					
Female	0.94	0.62-1.43	0.776			
ASA						
I-II	1			1		
III-IV	1.32	0.84-2.09	0.221			
Diabetes						
No	1			1		
Yes	1.45	0.90-2.33	0.123			
Hypertension						
No	1					
Yes	1.16	0.70-1.93	0.569			
Abdominal surgery history						
No	1					
Yes	1.21	0.66-2.22	0.532			
TNM stage						
I	1			1		
II	2.12	1.23-3.98	0.008	1.84	1.05-3.24	0.032
III	10.83	6.61-17.73	<0.001	8.65	5.20-14.39	<0.001
Operation method						
Laparoscopy	1					
Open	1.42	0.83-2.45	0.198			
Tumor size						
≤5 cm	1					
>5 cm	1.13	0.65-1.97	0.661			
Tumor location						
Colon	1					
Rectum	1.25	0.73-2.12	0.416			
Combined resection						
No	1			1		
Yes	1.75	1.25-2.44	0.001	2.21	1.18-4.12	0.013
Histologic type						
Differentiated	1			1		
Undifferentiated	1.59	1.24-2.03	<0.001	1.47	1.09-1.98	0.011
Neoadjuvant chemotherapy						
No	1					
Yes	1.36	0.85-2.18	0.194			
Adjuvant chemotherapy						
No	1					
Yes	0.98	0.63-1.52	0.941			
Sarcopenic obesity						
Normal	1			1		
Obesity	1.50	1.12-2.01	0.007	1.81	1.01-4.65	0.042
Sarcopenia	1.49	1.10-2.03	0.011	1.92	1.05-3.52	0.034
Sarcopenic obesity	2.06	1.50-2.83	0.005	2.74	1.39-5.42	0.004

ASA = American Society of Anesthesiologists; TNM = Tumor-Node-Metastasis classification; CI = confidence interval.

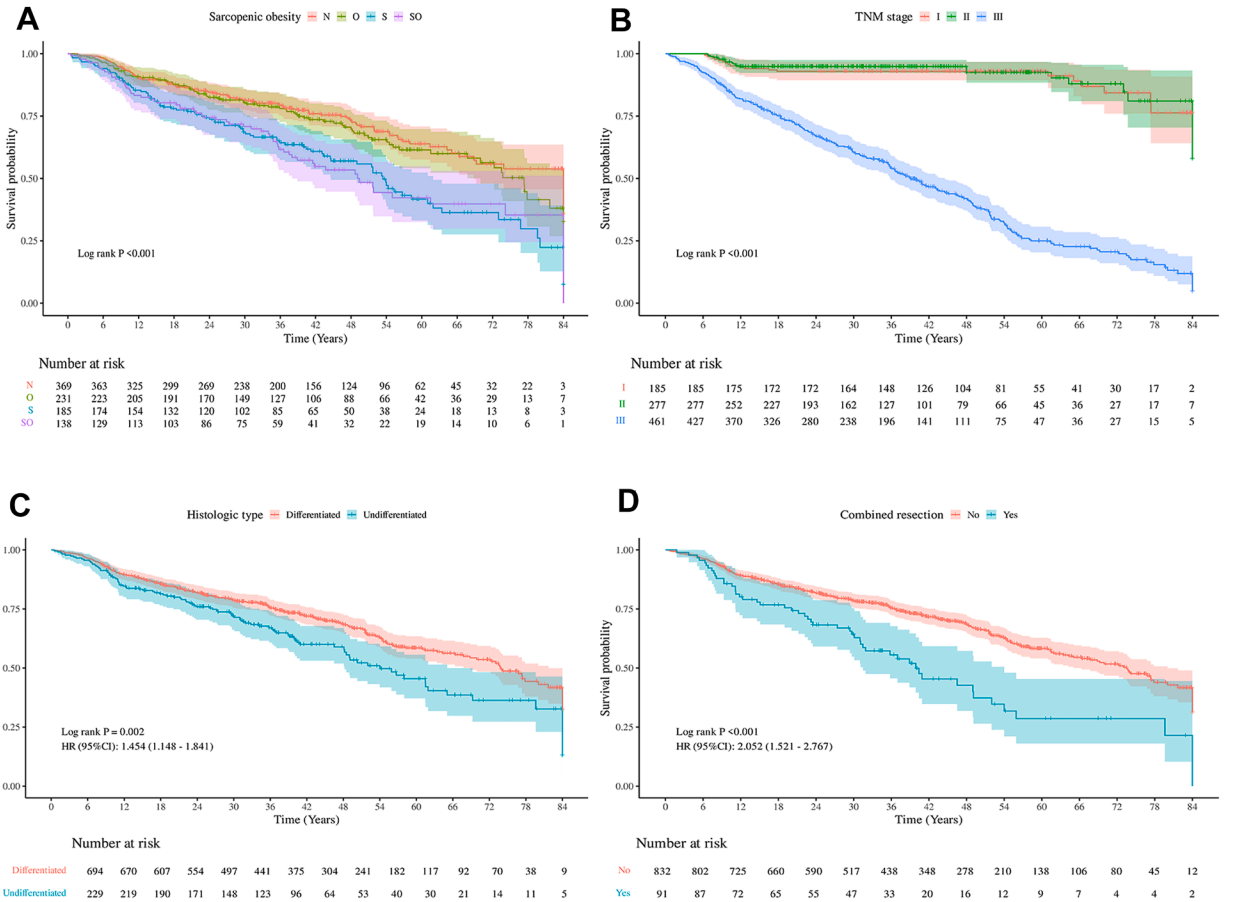

Fig. 3 – Kaplan–Meier survival curves for RFS across various clinical subgroups. (A) RFS stratified by body composition categories (normal, obesity, sarcopenia, and sarcopenic obesity); (B) RFS stratified by TNM stage; (C) RFS stratified by histological type; (D) RFS comparison between patients undergoing combined resection and standard surgery. RFS = recurrence-free survival.

deeper mechanistic investigation into its metabolic–immune pathways.

Mechanistically, the adverse impact of SO appears to result from the convergence of metabolic, inflammatory, and immune disturbances. Visceral adipose tissue functions as a highly active endocrine organ that secretes excessive pro-inflammatory cytokines such as interleukin-6 (IL-6), tumor necrosis factor-α, and C-reactive protein, together with adipokines including leptin and resistin [22,23]. This chronic inflammatory milieu promotes oxidative stress, endothelial dysfunction, and systemic insulin resistance, leading to impaired microcirculation and delayed wound healing after surgery.[24,25] Simultaneously, loss of skeletal muscle mass diminishes the release of beneficial myokines, such as irisin, myonectin, and IL-15, that normally exert anti-inflammatory, insulin-sensitizing, and antitumor effects.[26-28] Reduced myokine signaling contributes to immune exhaustion, metabolic inflexibility, and an increased production of reactive oxygen species.[29] Collectively, this bidirectional disruption of the muscle–fat axis amplifies systemic inflammation, attenuates host immune surveillance, and fosters a tumor-permissive microenvironment, providing a plausible mechanistic basis for the higher complication rates and poorer survival observed in SO patients.[30,31] Moreover, recent studies suggest that redox- and metabolism-sensitive signaling pathways, such as nuclear factor erythroid 2–related factor 2, AMP-activated protein kinase–mechanistic target of rapamycin, and interleukin-6/Janus kinase/signal transducer and activator of transcription 3, may participate in regulating these metabolic-immune interactions, providing a biological basis for the potential link between SO, tumor progression, and immune suppression.[32-35]

In clinical practice, BMI is an imprecise indicator of body composition, as it fails to differentiate adipose tissue distribution or reflect muscle quality and functional reserve.[36] In contrast, quantitative CT analysis provides an objective and reproducible assessment of skeletal muscle and visceral adiposity, offering a more accurate reflection of patients' nutritional and metabolic status.[37] Complementary functional tests, such as handgrip strength and gait speed, further capture muscle performance and physical resilience beyond structural measurements. Integrating morphological and functional parameters enables a more comprehensive and precise evaluation of perioperative risk. Recent evidence has

Table 6 – Univariable and multivariable cox regression analyses of prognostic factors for disease-free survival.

Variables	Univariable analysis			Multivariate analysis		
	HR	95% CI	*P* value	HR	95% CI	*P* value
Age, y	1.01	0.98-1.04	0.412			
Gender						
Male	1					
Female	0.93	0.62-1.39	0.726			
ASA						
I-II	1					
III-IV	1.28	0.83-1.97	0.259			
Diabetes						
No	1					
Yes	1.33	0.87-2.05	0.186			
Hypertension						
No	1					
Yes	1.09	0.70-1.70	0.694			
Abdominal surgery history						
No	1					
Yes	1.22	0.69-2.15	0.496			
TNM stage						
I	1			1		
II	1.09	0.59-2.02	0.782			
III	10.51	6.60-16.75	<0.001	8.74	5.29-14.45	<0.001
Operation method						
Laparoscopy	1					
Open	1.32	0.83-2.12	0.241			
Tumor size						
≤5 cm	1					
>5 cm	1.17	0.72-1.89	0.523			
Tumor location						
Colon	1					
Rectum	1.21	0.73-2.01	0.456			
Combined resection						
No	1			1		
Yes	2.05	1.52-2.77	<0.001	1.88	1.12-3.17	0.017
Histologic type						
Differentiated	1			1		
Undifferentiated	1.45	1.15-1.84	0.002	1.37	1.05-1.79	0.021
Neoadjuvant chemotherapy						
No	1					
Yes	1.27	0.88-1.83	0.199			
Adjuvant chemotherapy						
No	1					
Yes	0.95	0.63-1.43	0.811			
Sarcopenic obesity						
Normal	1			1		
Obesity	1.14	0.85-1.53	0.379			
S	1.93	1.46-2.56	<0.001	1.78	1.23-2.58	0.003
Sarcopenic obesity	1.91	1.40-2.62	<0.001	1.83	1.26-2.67	0.002

ASA = American Society of Anesthesiologists; TNM = Tumor-Node-Metastasis classification; CI = confidence interval.

highlighted the growing clinical importance of multimodal prehabilitation, which combines nutritional optimization and physical conditioning. Interventions such as high-protein and leucine-enriched supplementation, omega-3 fatty acid intake, resistance training, and anti-inflammatory strategies have been shown to enhance skeletal muscle mass, improve metabolic reserve, and reduce postoperative complications in high-risk surgical patients.[38-41] These findings underscore the potential of targeted metabolic and functional optimization to improve short-term recovery and long-term outcomes in CRC surgery.

Importantly, SO represents a modifiable and potentially reversible metabolic phenotype rather than an irreversible condition. Recent interventional studies have demonstrated that multimodal prehabilitation programs, combining high-protein and leucine-enriched supplementation, omega-3 fatty acid intake, resistance exercise, and anti-inflammatory strategies, can significantly improve skeletal muscle quality, enhance metabolic reserve, and reduce postoperative morbidity in CRC patients.[41,42] Incorporating structured metabolic optimization into routine perioperative care may therefore shift CRC management from passive risk recognition to proactive physiological correction, particularly during the preoperative window when metabolic plasticity remains greatest.

From a translational standpoint, our findings highlight the importance of routine CT-based body composition analysis as part of perioperative risk assessment, integrated with established nutritional risk tools such as Nutritional Risk Screening 2002 and Global Leadership Initiative on Malnutrition criteria. International guidelines from the European Society for Clinical Nutrition and Metabolism, the European Association for the Study of Obesity, and the American Society of Clinical Oncology already recommend preoperative nutritional screening and individualized support.[43-46] Extending these recommendations to include objective quantification of skeletal muscle and visceral adiposity could refine patient stratification, guide personalized prehabilitation, and improve both short- and long-term outcomes in CRC surgery.

This study possesses several strengths including a retrospective design with standardized CT and muscle mass measurements, a large Asian cohort addressing geographic gaps in the literature, and comprehensive evaluation of both short- and long-term outcomes. Limitations include its single-center nature, reliance on preoperative body composition measurements without longitudinal follow-up, and the absence of detailed inflammatory or lifestyle covariates. In addition, due to the retrospective nature of the study, objective measures of muscle strength and physical performance (such as handgrip strength and gait speed) were not available, precluding functional evaluation of sarcopenia. Future multicenter longitudinal studies integrating dynamic body composition tracking and molecular profiling are needed to elucidate causal pathways between SO and oncologic prognosis.

Conclusions

In conclusion, SO independently predicts postoperative complications and adverse survival after curative CRC surgery. The coexistence of skeletal muscle loss and visceral adiposity amplifies surgical stress and oncologic progression risk. Incorporating CT-derived body composition metrics into routine preoperative assessment may markedly enhance risk prediction and enable targeted nutritional and rehabilitative interventions. Early identification and correction of SO hold promise for improving postoperative recovery, therapeutic tolerance, and long-term survival, steering colorectal surgery toward a truly metabolic–nutritional–immunologic paradigm of precision care.

Disclosure

The authors declare that they have no competing interests.

Funding

This research received no external funding.

Availability of Data

The data of this study are available from the corresponding authors on reasonable request.

CRediT authorship contribution statement

Cong Huang: Data curation, Conceptualization. **Bo Yang:** Software, Project administration, Methodology, Formal analysis. **Lianwei Wang:** Validation, Methodology. **Ouyue Wang:** Writing – review & editing, Writing – original draft, Visualization, Validation, Supervision, Formal analysis, Data curation.

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