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Skeletal muscle loss and associated clinical outcomes in patients with small-cell lung cancer receiving concurrent chemoradiotherapy

Song Ee Park^{1†} , In Gyu Hwang^{2,3†} and Jin Hwa Choi^{3,4*}

Abstract

Purpose Low muscle mass and skeletal muscle loss have been associated with prognosis in patients with cancer. This study evaluated the effects of changes in the skeletal muscle index (SMI) on overall survival in patients with limited-stage small-cell lung cancer (LD-SCLC) undergoing concurrent chemoradiotherapy (CCRT).

Methods This retrospective study included 55 patients with LD-SCLC who underwent CCRT. Body composition analysis was performed at baseline and post-treatment to determine changes in body mass index (BMI), the SMI at the fourth thoracic vertebral level (T4), and the SMI and areas of fat at the third lumbar vertebral level (L3). The prognostic impact of these parameters was evaluated.

Results At baseline, 44.6% of patients had L3 low muscle mass according to Korean population-specific criteria. Median changes in the L3 SMI, T4 SMI, BMI, and body weight after CCRT were $-5.8 \text{ cm}^2/\text{m}^2$ ($P < 0.001$), $0.1 \text{ cm}^2/\text{m}^2$ ($P = 0.613$), $-0.2 \text{ kg}/\text{m}^2$ ($P = 0.316$), and -0.7 kg ($P = 0.228$), respectively. Univariate analysis associated Eastern Cooperative Oncology Group (ECOG) performance status 2 ($P < 0.001$), baseline T4 low muscle mass, defined as the lowest T4 SMI quartile ($P = 0.016$), and a decrease in the L3 SMI during treatment ($P = 0.011$) with an increased mortality risk. Multivariate analysis identified ECOG 2 (hazard ratio = 5.815; $P = 0.003$) and a decrease in the L3 SMI during treatment (hazard ratio = 2.172; $P = 0.027$) as risk factors for poor prognosis.

Conclusion Skeletal muscle loss during CCRT, particularly a decrease in L3 SMI, was associated with inferior overall survival in LD-SCLC patients. Monitoring and mitigating skeletal muscle loss may improve patient prognosis.

Keywords Low muscle mass, Skeletal muscle, Prognosis, Small-cell lung cancer

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Introduction

Lung cancer is the leading cause of cancer-related mortality worldwide, and small-cell lung cancer (SCLC) accounts for approximately 15% of all lung cancer cases [1]. SCLC is characterized by rapid tumor growth and early dissemination. Despite initial sensitivity to chemotherapy and radiotherapy, the overall prognosis of SCLC is poor, with median survival ranging from 15 to 20 months in patients with limited-stage disease and from 8 to 13 months in those with extensive-stage disease [2].

While disease burden and stage are important determinants of clinical outcomes, host-related factors such as nutritional status, performance status, and muscle mass have been gaining attention. Weight loss, a hallmark of cancer cachexia, is frequently observed in patients with SCLC at the time of diagnosis. However, changes in body weight alone may be insufficient to reflect alterations in body composition, particularly in patients with a large tumor burden or fluid retention such as pleural effusion or edema.

Sarcopenia, which is defined as the progressive and generalized loss of skeletal muscle mass and strength, is a clinically important component of cancer cachexia. Recent advances in the computed tomography (CT)-based quantification of skeletal muscle mass have provided an objective and reproducible method of sarcopenia assessment [3]. Sarcopenia has been associated with reduced physical function, increased treatment-related toxicity, and poor survival outcomes. Its clinical relevance has led to its inclusion in international guidelines as a diagnostic criterion for cancer cachexia [4].

Despite the growing interest in body composition in cancer, data on the prognostic significance of sarcopenia and treatment-related muscle loss in patients with SCLC remain limited. Most previous studies have focused on baseline sarcopenia [5, 6], with few exploring dynamic changes in skeletal muscle during chemoradiotherapy and their potential impact on survival.

Therefore, the aim of this study was to investigate changes in body composition parameters, including the skeletal muscle index (SMI), in patients with limited-stage (LD)-SCLC undergoing concurrent chemoradiotherapy (CCRT) and to evaluate the prognostic implications of muscle loss. These results could provide evidence supporting the incorporation of body composition monitoring into routine clinical management.

Methods

Study design and patient population

This retrospective cohort study reviewed the medical records of patients diagnosed with LD-SCLC who underwent CCRT between September 2010 and January 2022 at Chung-Ang University Hospital. The inclusion criteria were as follows: pathologically confirmed

SCLC, administration of standard etoposide/cisplatin (EP) based CCRT, mediastinum included in radiation field, and availability of enhanced abdominopelvic CT images obtained less than two weeks before CCRT initiation and less than four weeks after treatment completion. Although post-treatment CT scans were intended to be performed within four weeks after CCRT completion, the actual timing varied in clinical practice. In our cohort, the mean interval between treatment completion and post-treatment CT was 2.5 months, with a median of 2.0 months (range, 0–9.0 months). All patients underwent thoraco-abdominal-pelvic CT, brain magnetic resonance imaging, and positron emission tomography-CT at baseline, and staging was reassessed according to the American Joint Committee on Cancer (AJCC) 8th edition TNM classification to confirm limited-stage disease. Bone marrow biopsy was not routinely performed. The exclusion criteria were as follows: lack of baseline or post-treatment imaging, administration of non-standard treatment regimens, or lost to follow-up within three months of treatment initiation. This study was approved by the Institutional Review Board of the Chung-Ang University Hospital (No. 2207-009-19426), which waived the requirement for informed consent due to the retrospective nature of the study.

Assessment of body composition

All abdominopelvic and thoracic CT scans used for body composition analysis were obtained with a standardized tube voltage of 120 kV, which is the institutional protocol at Chung-Ang University Hospital for both diagnostic and treatment-planning CT examinations. Body composition was assessed using CT at baseline, defined as within two weeks prior to CCRT initiation, and at post-treatment, defined as within four weeks after CCRT completion. Body composition analysis was performed using TeraRecon Aquarius iNtuition software (TeraRecon, Durham, NC, USA) to measure the skeletal muscle area (SMA), visceral fat area (VFA), and subcutaneous fat area (SCFA). These areas were automatically calculated from single axial CT slices at the third lumbar vertebra (L3) and fourth thoracic vertebra (T4), with both transverse processes visible. Areas were quantified based on predefined Hounsfield unit (HU) thresholds: SMA, −29 to +150 HU; VFA, −150 to −50 HU; and SCFA, −190 to −30 HU [7]. In addition to absolute cross-sectional areas, the visceral fat area index (VFI) and subcutaneous fat area index (SFI) were calculated by normalizing VFA and SCFA to patient height squared (m^2). The L3 SMI, which has been shown to correlate well with total body skeletal muscle mass, was calculated as the L3 SMA (cm^2) divided by the square of the patient's height (m^2). L3 low muscle mass was defined as an L3 SMI $< 49 cm^2/m^2$ for men and $< 31 cm^2/m^2$ for women, according to criteria specific for

Korean populations [8]. Patients were classified as having a high or low VFI and SFI using the mean baseline values as thresholds.

The cross-sectional areas of the thoracic skeletal muscles, including the pectoralis, intercostalis, paraspinous, serratus, and latissimus muscles were measured at the T4 level as described previously [9]. The T4 SMI was calculated by dividing the thoracic SMA (cm²) by the square of the patient's height (m²). There is no established T4 SMI cutoff point for defining T4 low muscle mass; therefore, we divided the patients into quartiles according to the T4 SMI, and the lowest quartile was defined as the low muscle mass group, as described previously [9].

Body weight and height information were extracted from patient medical records. Body mass index (BMI) was calculated as weight divided by height squared (kg/m²), and patients were classified as underweight (< 20.0 kg/m²), normal (20.0–25.0 kg/m²), or overweight (>25.0 kg/m²). Body weight was measured within two weeks of the corresponding CT scan. Although the WHO classification defines underweight as a BMI < 18.5 kg/m², we applied a cutoff of 20 kg/m² based on prior studies in Asian and cancer populations, where this threshold has been associated with malnutrition, reduced treatment tolerance, and poor prognosis [10, 11].

Chemotherapy regimen

All patients received a standard EP regimen concurrently with thoracic radiotherapy. Cisplatin was administered at a dose of 25 mg/m²/day on days 1–3, and etoposide at 100 mg/m²/day on days 1–3, every 21 days. A total of four cycles were planned, and patients who tolerated treatment generally completed all four cycles. The treatment completion rate for concurrent chemoradiotherapy was 92.7% (51/55 patients); four patients discontinued chemotherapy early due to hematologic or non-hematologic toxicities but still completed radiotherapy.

Radiotherapy

All patients received definitive thoracic radiotherapy using either three-dimensional conformal radiotherapy or intensity-modulated radiotherapy. The total prescribed dose was 60 Gy, delivered in 30 fractions (2.0 Gy per fraction), once daily, five days per week. The mediastinum was included in the radiation field in all cases. All patients have completed scheduled thoracic radiotherapy. Prophylactic cranial irradiation was administered at the discretion of the treating physician after completion of CCRT.

Endpoints and clinical variables

The primary endpoints were the changes in body composition indices, including the T4 SMI and L3 SMI, VFI, and SFI from baseline to post-treatment, and the incidence of

significant muscle loss. Secondary endpoints included changes in BMI and body weight, overall survival (OS), treatment response rate, and the association between skeletal muscle loss and prognosis. Tumor response was evaluated by applying the RECIST guidelines version 1.1 to CT scans acquired every six to eight weeks. Additional clinical and laboratory data included age, sex, Eastern Cooperative Oncology Group (ECOG) performance status scales, AJCC 8th edition stage group, chemotherapy regimen, serum albumin level, and survival status.

Statistical analysis

Data are presented as median (range), mean $\pm$ standard deviation, or number (percentage). Continuous variables were tested for normality using the Kolmogorov–Smirnov test. Paired t-tests or Wilcoxon signed-rank tests were used to compare baseline and post-treatment body composition parameters. Between-group differences were assessed using the Student's t-test or Mann–Whitney U test for continuous variables and the chi-squared test or Fisher's exact test for categorical variables. OS was defined as the time from the start of chemotherapy to the date of death from any cause and was estimated using the Kaplan–Meier method. Group comparisons of OS were performed using log-rank tests. Hazard ratios (HRs) and 95% confidence intervals (CIs) were calculated using the Cox proportional hazards model. Variables with $P < 0.10$ in univariate analysis were entered into multivariate analysis. All statistical analyses were performed using SPSS software version 25.0 (IBM Corp., Armonk, NY, USA); a two-tailed P -value < 0.05 was considered statistically significant.

Results

Patients

A total of 55 patients with LD-SCLC who underwent CCRT between September 2010 and January 2022 were included in the study. The baseline characteristics of the study population are summarized in Table 1. The median age of the patients was 67 (range, 43–80) years and 63.6% of the patients were aged 65 years or older. Most patients (92.7%) were male and 5 patients (9.1%) had an ECOG performance status of 2. The mean BMI at baseline was 23.0 ± 3.3 kg/m²; 14.3% of patients were classified as underweight and 39.3% as overweight. The mean T4 and L3 SMI were 44.1 ± 8.1 and 49.1 ± 7.9 cm²/m², respectively. At baseline, 25 patients (44.6%) had L3 low muscle mass and 13 patients with T4 low muscle mass defined as the lowest T4 SMI quartile; 9 patients with T4 low muscle mass (69.2%) also had L3 low muscle mass. The L3 SFI and VFI were 36.0 ± 20.3 and 46.9 ± 27.8 cm², respectively.

Table 1 Baseline patient characteristics

Characteristics	All (n=55)
Age (years)	
Median (range)	67 (43–80)
≥ 65	35 (63.6%)
Sex, n (%)	
Male	51 (92.7%)
Female	4 (7.3%)
ECOG performance status	
ECOG 0–1	50 (90.9%)
ECOG 2	5 (9.1%)
Stage	
T stage	
T1	8 (14.5%)
T2	23 (41.8%)
T2a	5 (9.1%)
T2b	18 (32.7%)
T3	8 (14.5%)
T4	16 (29.2%)
N stage	
N0	8 (14.5%)
N1	8 (14.5%)
N2	25 (45.5%)
N3	14 (25.5%)
Stage group (AJCC 8th)	
Stage I	4 (7.3%)
Stage IIA	2 (3.6%)
Stage IIB	5 (9.1%)
Stage IIIA	20 (36.4%)
Stage IIIB	16 (29.1%)
Stage IIIC	8 (14.5%)
Body mass index (kg/m) ²	
Underweight (< 20)	8 (14.3%)
Normal weight (20–25)	25 (44.6%)
Overweight (> 25)	22 (39.3%)
T4 skeletal muscle index (cm ² /m ²)	44.1 ± 8.1
L3 skeletal muscle index (cm ² /m ²)	49.1 ± 7.9
L3 low muscle mass, n (%)	25 (44.6%)
T4 low muscle mass, n (%)	14 (25.0%)
L3 subcutaneous fat area index (cm ² /m ²)	36.0 ± 20.3
L3 visceral fat area index (cm ² /m ²)	46.9 ± 27.8
Serum albumin (g/dL)	
Median (range)	4.0 (2.8–5.0)
< 3.5	7 (12.7%)

Data are presented as median (range), mean ± standard deviation, or number (percentage)

T4 Fourth thoracic vertebra, L3 Third lumbar vertebra

Changes in body composition during treatment

Changes in body composition parameters from baseline to post-treatment are shown in Table 2. The mean L3 SMI declined significantly from 49.1 ± 7.9 to 43.2 ± 7.7 cm²/m², representing a reduction of 11.8% ($P < 0.001$). In contrast, no significant change was observed in the T4 SMI, which showed a mean difference of 0.1 cm²/m² ($P = 0.613$). Mean body weight and BMI also decreased

during treatment, but the mean differences of −0.7 kg and −0.2 kg/m², respectively, did not reach statistical significance. The SFI showed a slight increase that did not reach statistical significance, whereas the VFI increased significantly following treatment ($P = 0.029$). The mean change in the L3 SMI was used as a cutoff value; patients with a decrease in the L3 SMI of ≥ 5.8 cm²/m² were included in the decreased L3 SMI group. Similarly, an increase in the L3 VFI of ≥ 5.1 cm²/m², the mean change in this parameter, was used to define the increased VFI group.

Survival according to baseline low muscle mass and changes in body composition

The median OS was 35.5 months, with a median follow-up duration of 81.9 months. Univariate Cox regression analysis identified several body composition parameters as potential predictors of poor OS (Table 3), with median OS determined using Kaplan–Meier analysis (Fig. 1). Median OS was shorter in patients with baseline L3 low muscle mass than in those without L3 low muscle mass (26.1 vs. 58.3 months), although this difference did not reach statistical significance ($P = 0.092$) (Fig. 1A). Patients with baseline T4 low muscle mass had a significantly shorter median OS than that of patients without T4 low muscle mass (15.5 vs. 58.0 months; $P = 0.016$) (Fig. 1B). The patients were divided into high and low groups based on the mean value of each fat index. Median OS was shorter in the high VFI group than in the low VFI group (26.5 vs. 68.2 months) but this difference did not reach statistical significance (HR = 1.887; 95% CI: 0.926–3.846; $P = 0.076$) (Fig. 1C). Median OS was similar in the high and low SFI groups, at 31.5 and 26.1 months, respectively (HR = 0.802; 95% CI: 0.402–1.598; $P = 0.531$) (Fig. 1D).

Stratification by the degree of L3 SMI change during CCRT revealed that the median OS of the decreased L3 SMI group was significantly shorter than that of the preserved L3 SMI group (6.3 vs. 28.7 months; HR = 2.402; 95% CI: 1.223–4.717; $P = 0.011$) (Fig. 2). However, changes in the VFI during CCRT were not related to prognosis, as median OS did not differ significantly between the maintained and increased VFI groups (45.7 vs. 35.5 months; $P = 0.811$). Because T4 SMI and SFI did not change significantly during CCRT, they were not further analyzed for prognostic value. In multivariate analysis, ECOG performance status ≥ 2 (HR = 5.815; 95% CI: 1.856–18.215; $P = 0.003$) and decreased L3 SMI (HR = 2.172; 95% CI: 1.092–4.319; $P = 0.027$) remained independent predictors of poor OS.

Causes of death

Most deaths (48/55, 87.3%) were due to disease progression and 7 (12.7%) due to pneumonia. In the T4 low muscle mass group, 11 patients died from progression and 3 from pneumonia. Pneumonia-related mortality

Table 2 Body composition parameters

Parameter	Baseline		Post CCRT		Change		P-value
	Mean	SD	Mean	SD	Mean	95% CI	
Body weight (kg)	62.9	10.3	62.1	10.3	−0.7	−2.0 to 0.4	0.228
Body mass index (kg/m ²)	23.0	3.3	22.8	3.5	−0.2	−0.6 to 0.2	0.316
L3 SMI (cm ² /m ²)	49.1	7.9	43.2	7.7	−5.8	−7.2 to −4.4	< 0.001
T4 SMI (cm ² /m ²)	44.1	8.1	44.3	8.4	0.1	−0.3 to 0.5	0.613
Subcutaneous fat area index (cm ² /m ²)	36.0	20.3	37.6	20.4	1.6	−1.6 to 4.9	0.318
Visceral fat area index (cm ² /m ²)	46.9	27.8	52.0	29.0	5.1	0.5 to 9.7	0.029

CCRT Concurrent chemoradiotherapy, SD Standard deviation, CI Confidence interval, T4 Fourth thoracic vertebra, L3 Third lumbar vertebra, SMI Skeletal muscle index

Table 3 Univariate and multivariate analyses of factors associated with overall survival

Risk factor	Univariate analysis			Multivariate analysis		
	HR	95% CI	P-value	HR	95% CI	P-value
Age ≥ 65	1.630	0.807–3.292	0.173			
Male	0.899	0.273–2.963	0.861			
ECOG 2	5.940	2.153–16.391	< 0.001	5.815	1.856–18.215	0.003
Stage (AJCC 8th)						
Stage I	Reference					
Stage II	0.233	0.021–2.579	0.235			
Stage III	2.124	0.507–8.903	0.303			
L3 low muscle mass	1.817	0.899–3.675	0.096			
T4 low muscle mass	2.427	1.177–5.005	0.016	1.551	0.701–3.435	0.279
Decreased L3 SMI	2.402	1.223–4.717	0.011	2.607	1.299–5.232	0.007
High SFI	0.802	0.402–1.598	0.531			
High VFI	1.887	0.926–3.846	0.076			
Increased VFI	0.752	0.384–1.474	0.407			
Body mass index (kg/m ²)						
Underweight (< 20)	Reference					
Normal weight (20–25)	0.487	0.205–1.154	0.102			
Overweight (> 25)	0.442	0.180–1.088	0.076			

HR Hazard ratio, CI Confidence interval, T4 Fourth thoracic vertebra, L3 Third lumbar vertebra, SMI Skeletal muscle index, SFI Subcutaneous fat area index, VFI Visceral fat area index

was relatively more common in the T4 low muscle mass group, although the overall distribution was not statistically different. Similarly, among the 7 pneumonia-related deaths, 3 occurred in the decreased L3 SMI group. These findings suggest that severe infections tended to be more frequent in patients with sarcopenia, despite the absence of significant differences in overall treatment-related adverse events.

Discussion

In this retrospective cohort study of patients with LD-SCLC undergoing CCRT, significant reductions in the SMI at the L3 vertebral level were common and strongly associated with worse OS. These findings underscore the clinical importance of treatment-related skeletal muscle loss, beyond traditional body composition parameters of body weight or BMI, as a prognostic factor in SCLC.

Although sarcopenia is increasingly recognized as a component of cancer cachexia and a predictor of poor outcomes in various malignancies [4, 12, 13], its relevance in SCLC, particularly during curative-intent

therapy, remains underexplored. Our results demonstrated that nearly half of the patients exhibited low muscle mass at baseline and that muscle depletion progressed significantly during CCRT. Importantly, patients in the decreased L3 SMI group had a median OS of just 6.3 months, compared to 28.7 months in those with preserved muscle mass, despite receiving the same standard of care. These results suggest that while baseline low muscle mass predicts poor outcomes, dynamic loss of skeletal muscle during treatment exerts an even stronger prognostic impact. Longitudinal monitoring of changes in muscle mass may therefore provide prognostic information beyond initial body composition assessments, reflecting treatment tolerance, systemic inflammation, and metabolic deterioration.

These findings are consistent with those of previous studies on gastrointestinal and breast cancers [5, 6, 14], further supporting the hypothesis that skeletal muscle loss reflects systemic inflammation, metabolic dysregulation, and reduced treatment tolerance. Prior studies have reported that CT-derived skeletal muscle mass

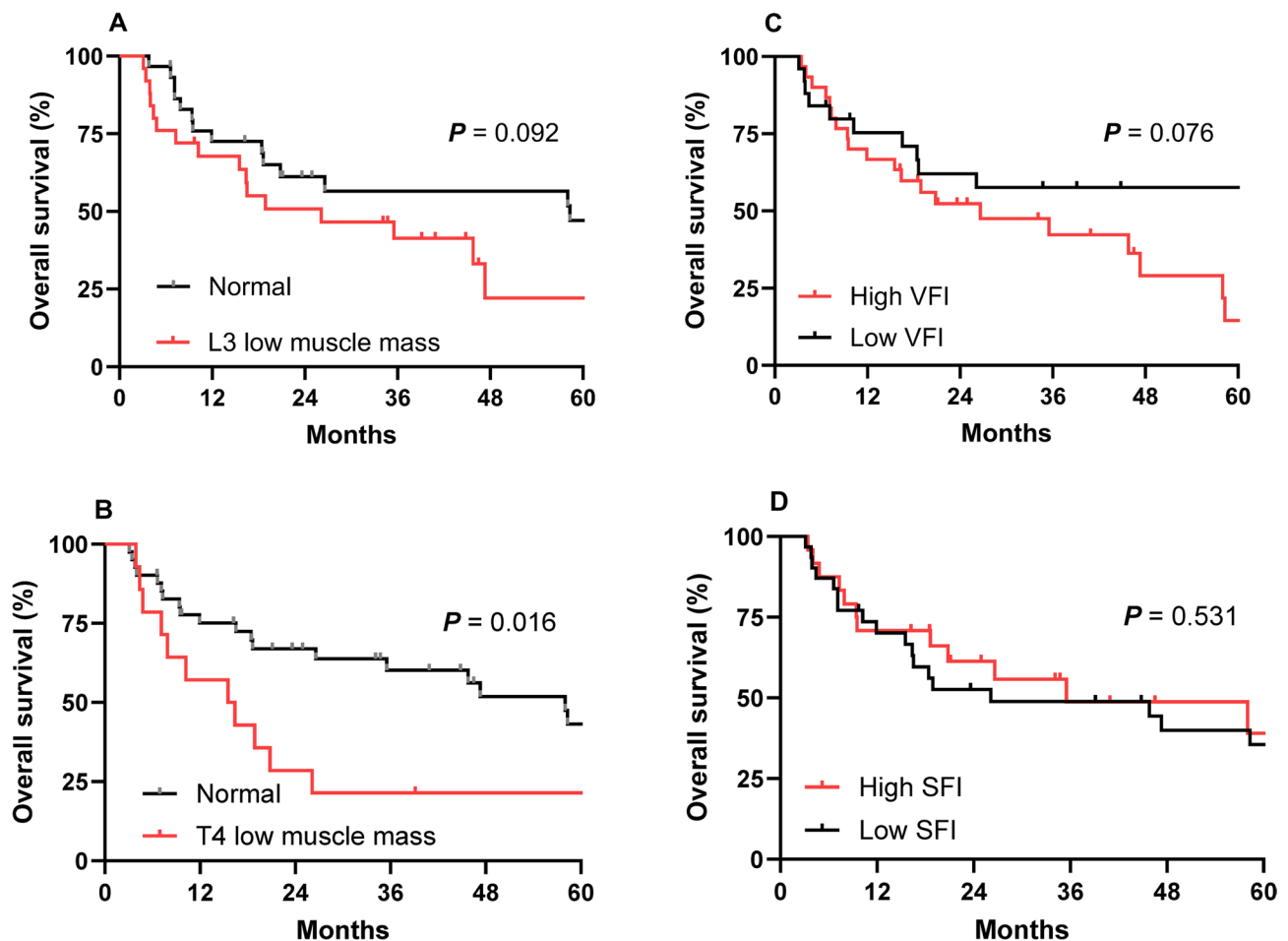


Fig. 1 Overall survival of patients with limited-stage small-cell lung cancer according to baseline body composition parameters. **A** Third lumbar vertebra (L3) skeletal muscle index (SMI), **B** Fourth thoracic vertebra (T4) SMI, **C** Visceral fat area index (VFI), and **D** subcutaneous fat area index (SFI)

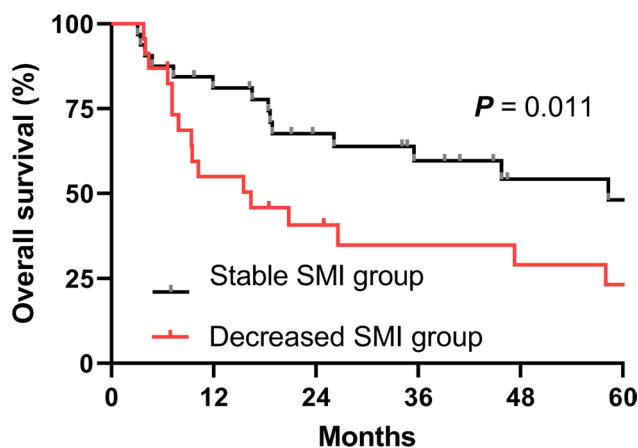


Fig. 2 Overall survival of patients with limited-stage small-cell lung cancer according to the change in the third lumbar vertebra skeletal muscle index (SMI) during concurrent chemoradiotherapy

is inversely correlated with serum C-reactive protein (CRP) in small cell lung cancer, whereas sarcopenia and myosteatosis are associated with elevated neutrophil-to-lymphocyte ratios (NLR) in colorectal cancer. Specifically, elevated levels of proinflammatory cytokines such as interleukin-6 (IL-6) and tumor necrosis factor- α (TNF- α) have been implicated in promoting muscle proteolysis and inhibiting protein synthesis, thereby accelerating sarcopenia in cancer patients [15]. Moreover, treatment-related toxicity from chemotherapy and radiotherapy may exacerbate systemic inflammation and metabolic stress, further contributing to muscle wasting [16]. These mechanisms likely explain the strong prognostic impact of dynamic muscle loss observed in our study.

We also examined the T4 SMI, which can be readily obtained from routine chest CT. Although the L3 SMI remains the gold standard for assessing skeletal muscle mass, abdominal CT is not always available in lung cancer patients. Previous studies in idiopathic pulmonary fibrosis have reported that low T4 muscle mass predicts poor outcomes [9]. In our study, baseline T4 low muscle

mass was associated with shorter OS in univariate analysis, but treatment-related changes in T4 SMI were minimal and not prognostic. Notably, measurements at T4 have important limitations, including potential confounding by the diaphragm and the absence of a universally established cutoff for defining low muscle mass at this level. These factors may partly explain why T4 SMI was less predictive than L3 SMI in our study. Nevertheless, thoracic-level indices remain useful alternatives when abdominal imaging is unavailable, although further validation in larger cohorts is warranted [17–19]. Overall, these findings highlight the superior sensitivity of lumbar measurements for detecting clinically meaningful muscle loss while acknowledging the practical utility of thoracic-level assessments in specific clinical settings.

For cut-off values, we applied sex-specific thresholds validated in the Korean population [8]. Alternative disease-specific cutoffs in lung cancer cohorts have also shown prognostic value [20, 21]. For T4 SMI, no established thresholds exist; thus, we used the lowest quartile, as in prior studies [9]. Further studies are needed to define standardized disease-specific values.

Regarding adiposity, a higher baseline VFI showed a trend toward worse OS, though not statistically significant. Prior studies have reported conflicting results for visceral fat, with both detrimental and protective associations depending on tumor type and treatment modality [22–25]. In our cohort, VFI increased significantly during CCRT but was not prognostic. Similarly, BMI and body weight changes were not associated with survival, underscoring the limited utility of weight-based assessments in SCLC patients with factors such as tumor burden, effusion, or edema. CT-based body composition analysis therefore provides a more objective and clinically relevant tool for risk stratification.

From a clinical perspective, our findings emphasize the importance of early recognition of skeletal muscle loss during curative-intent therapy for SCLC. Routine assessment at baseline and after treatment may help identify patients at high risk who could benefit from nutritional support, exercise interventions, or pharmacologic strategies aimed at preserving muscle mass. Emerging evidence suggests that such multimodal interventions may improve treatment tolerance and quality of life, though definitive survival benefits remain to be established [26].

This study has several limitations, including its retrospective single-center design and relatively small sample size. The cutoff used to define decreased L3 SMI was derived from the intra-cohort mean change and has not been externally validated. Low muscle mass was defined solely by CT-derived skeletal muscle area, without functional performance assessments, which may have led to misclassification. Inflammatory markers such as CRP and NLR were not consistently available, limiting exploration

of mechanistic pathways. Future multicenter prospective studies incorporating both imaging-based and functional measures, as well as inflammatory biomarkers, are needed to validate our findings and establish standardized thresholds for clinically meaningful muscle loss.

Conclusions

Skeletal muscle loss is common in patients with LD-SCLC undergoing CCRT and is significantly associated with poor survival. ECOG performance status and a decline in the L3 SMI during treatment were independent predictors of mortality, emphasizing the prognostic relevance of host-related factors alongside traditional clinical parameters. Routine monitoring of skeletal muscle mass and timely supportive interventions may improve treatment tolerance and clinical outcomes in this patient population.

Abbreviations

BMI	Body mass index
CCRT	Concurrent chemoradiotherapy
CI	Confidence interval
CRP	C-reactive protein
CT	Computed tomography
ECOG	Eastern Cooperative Oncology Group
EP	Etoposide/cisplatin
HR	Hazard ratio
IL-6	Interleukin-6
L3	The third lumbar vertebral level
LD-SCLC	Limited-stage small-cell lung cancer
NLR	Neutrophil-to-lymphocyte ratios
OS	Overall survival
PFS	Progression-free survival
SCFA	Subcutaneous fat area
SCLC	Small-cell lung cancer
SFI	Subcutaneous fat area index
SMA	Skeletal muscle area
SMI	Skeletal muscle index
T4	Fourth thoracic vertebra
TNF- α	Tumor necrosis factor-alpha
VFA	Visceral fat area
VFI	Visceral fat area index

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Authors' contributions

All authors contributed to the study conception and design. Material preparation, data collection and analysis were performed by Song Ee Park, In Gyu Hwang and Jin Hwa Choi. The first draft of the manuscript was written by Song Ee Park and all authors commented on previous versions of the manuscript. All authors read and approved the final manuscript.

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Data availability

The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

Declarations

Ethics approval and consent to participate

This study was approved by the Institutional Review Board of the Chung-Ang University Hospital (No. 2207-009-19426) and was performed in accordance

with the principles outlined in the Declaration of Helsinki. The requirement for informed consent was waived due to the retrospective nature of the study.

Consent for publication

Not applicable.

Competing interests

The authors declare no competing interests.

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