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Original Article

Association between the psoas muscle index and disease progression and mortality in testicular germ cell tumours

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Background and objectives: The relationship between sarcopenia and prognosis in patients undergoing chemotherapy for testicular germ cell tumours remains underexplored. We aimed to evaluate the impact of sarcopenia on disease progression and overall survival in these patients.

Methods: This retrospective multicentre study included patients who received chemotherapy for testicular germ cell tumours between January 2010 and December 2023. The psoas muscle index was calculated by measuring the cross-sectional area of the psoas muscle at the third lumbar vertebral level and was divided by the square of the height. Patients were divided into two groups based on changes in PMI (before and after chemotherapy): Group 1 (<10% change) and Group 2 (≥10% change).

Results: A total of 159 patients were analysed. Of these, 113 (71.1%) were in Group 1 and 46 (28.9%) in Group 2. Group 2 showed higher rates of disease progression (26.1% *vs*. 10.6%) and mortality (8.7% *vs.* 1.8%) (P=0.023 and P=0.038, respectively). In multivariable analysis, ≥10 % decrease in psoas muscle index [Hazard Ratio (HR)=6.499, P<0.001], rete testis invasion (HR=3.459, P=0.007), and non-seminomatous/mixed histology (HR=5.777, P=0.020) were identified as independent predictors of disease progression. For mortality, only a ≥10 % decrease in psoas muscle index was found to be a significant predictor (HR=5.994, P=0.049).

Interpretation and conclusions: A reduction in PMI is an independent prognostic factor for both disease progression and mortality in patients undergoing systemic chemotherapy for testicular germ cell tumours.

Keywords Chemotherapy; Psoas major muscle; Sarcopenia; Skeletal muscle index; Testicular cancer,

Testicular germ cell tumours are known for their good response to treatment. Cure rates for metastatic disease exceed 80%. However, failure of first line and salvage treatments results in a poor prognosis.[1,2] The International Germ Cell Consensus Classification Group (IGCCGG) introduced a prognostic classification that includes primary tumour site, metastatic sites, and tumour markers.[3] However, the search for prognostic risk assessment tools to provide a more detailed perspective on the clinical features of these patients continues.[2]

Sarcopenia is characterised by the degenerative and systemic loss of skeletal muscle mass. Many cancer patients undergoing treatment experience alterations such as increased fat mass and decreased skeletal muscle mass.[4] In addition to oncologic surgery and radiotherapy, systemic inflammation, hormonal changes, malnutrition, impaired protein synthesis, and increased sympathetic neuronal activity are significant contributors to sarcopenia.[5,6] Chemotherapy-induced toxicity represents another critical factor.[7] Multidrug regimens are commonly employed to enhance tumour cell eradication; however, chemotherapeutic agents may inhibit the proliferation of muscle stem cells and damage differentiated muscle fibres.[8]

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Sarcopenia has been associated with poor survival outcomes in various malignancies, including lung cancer, gastrointestinal cancers, lymphoma, and melanoma.[9] Studies have also investigated the relationship between skeletal muscle loss and prognosis in urogenital cancers. In patients undergoing nephrectomy for localised renal cell carcinoma (RCC), sarcopenia has been linked to unfavourable oncologic outcomes.[10] It serves as an independent prognostic factor not only in localised disease but also in metastatic RCC.[11] In metastatic urothelial carcinoma, sarcopenia has also been shown to negatively impact survival.[12] Additionally, it is an independent predictor of increased all-cause mortality in patients undergoing radical cystectomy.[13]

Although the association between sarcopenia and prognosis has been well established in other urogenital cancers, studies focusing on sarcopenia in patients receiving chemotherapy for testicular germ cell tumour remain limited.[14-17] Even fewer have specifically examined its relationship with prognostic factors such as disease progression and survival in this population.[14] The present study aimed to investigate the impact of sarcopenia (as measured by psoas muscle index) on progression and survival in patients undergoing systemic chemotherapy for testicular germ cell tumour.

Methods

This retrospective study was undertaken by the department of Urology, Bakirkoy Dr. Sadi Konuk Training and Research Hospital, Istanbul, Turkey. The study was approved by the Institutional Review Board of the University of Health Sciences, Bakirkoy Dr. Sadi Konuk Training and Research Hospital.

This retrospective multicentre study included patients who underwent chemotherapy for testicular germ cell tumour and were followed between January 2010 and December 2023. Patients with other diagnoses, those in whom skeletal muscle measurement could not be performed on axial imaging, and those with incomplete data were excluded.

Measurement of muscle components: Demographic, pathological, and survival data of the patients were collected. Staging was performed using abdominal computed tomography (CT) scans obtained prior to chemotherapy. The psoas muscle was defined as an oval-shaped structure adjacent to the vertebral column in axial view and was measured within a range of −30 to +150 Hounsfield units on CT imaging.[13] The psoas muscle index (PMI) was calculated by measuring the cross-sectional area of the psoas muscles at the third lumbar vertebra (L3) level and dividing this value by the square of the patient's height: PMI (cm^2/m^2) = (sum of the bilateral psoas muscle areas at the L3 level)/(height)^2, using SYNAPSE PACS Software (Fujifilm Medical Systems, Tokyo, Japan).[18] For the skeletal muscle index (SMI), the total muscle area of the psoas, paraspinal, internal oblique, external oblique, rectus abdominis, and transversus abdominis muscles on both sides was measured at the L3 level using the same imaging system, and divided by the square of the height: SMI (cm^2/m^2)=(skeletal muscle cross-sectional area at L3)/(height)^2.[18]

PMI and SMI values were calculated and compared using CT scans obtained before and after the final cycle of first-line chemotherapy. Based on the ROC curve analysis, we determined that −10% was a valid cut-off value. Patients were grouped based on whether reductions in their pre- and post-chemotherapy PMI and SMI values exceeded 10%. The percentage change in index values following chemotherapy was calculated using the following formula: [(post-chemotherapy index − pre-chemotherapy index)/pre-chemotherapy index] × 100.

Correlation between indexes: Studies have emphasised a strong correlation between PMI and SMI values.[14,18] Another advantage of PMI is that imaging only the psoas muscle is sufficient, eliminating the need for expensive image analysis software.[19] The correlation between PMI and SMI was also evaluated in our study. The Pearson correlation coefficient was used to assess the strength of the linear association between PMI and SMI before and after chemotherapy. The agreement between changes in PMI and SMI was evaluated using Bland-Altman plots (**Supplementary Fig. 1**). PMI data were employed as the primary indicator of skeletal muscle loss in this study due to its simpler clinical applicability compared to SMI and the strong correlation between the two indices. Progression after chemotherapy and/or surgery was defined by the presence of one or more of the following: confirmed elevation of serum tumour marker levels, radiological evidence of relapse, and/or histologically confirmed relapse.[20] Treatment and follow-up protocols were conducted in accordance with the established guidelines.[20]

Patients were categorised into two groups based on PMI changes (before and after final cycle of chemotherapy): Group 1 (<10% change) and Group 2 (≥10% change). Demographic and pathological data were compared between these groups. Parameters predictive of disease progression and mortality were subsequently evaluated.

Statistical analysis: Statistical comparisons of patient characteristics were performed using the continuity correction test, Fisher's exact chi-square test, or Pearson chi-square test for categorical variables and the Mann-Whitney *U* test or the independent-samples *t*-test for continuous variables, depending on normality assumptions. The Pearson correlation coefficient was utilised to measure the strength of the linear association between PMI and SMI before and after chemotherapy. Correlations between PMI changes and SMI changes were evaluated using Bland-Altman plots. Kaplan-Meier curves were constructed, and the log-rank test was used to compare progression-free survival and overall survival across groups. Receiver operating characteristic (ROC) curves were plotted to compare PMI and SMI changes in predicting progressive disease. The optimal cutoff value for each method was determined considering the highest sensitivity, reasonably high specificity, and positive and negative predictive values. The area under the curve (AUC) was classified as good (AUC=0.8–1), moderate (0.7–0.8), fair (AUC=0.6–0.7), and poor (0.5–0.6). The AUC analyses of scoring systems were conducted using the MedCalc (trial version 22.030, MedCalc Software Ltd, Ostend, Belgium) programme. The relationship between clinical variables and progressive disease was examined using the Cox proportional hazard model. Multivariable analysis was applied to the parameters that were significant in the univariate analysis. Multivariable logistic regression models were constructed using the stepwise backward Wald method. A significance level of $P< 0.05$ was considered statistically significant.

Posteriori power analysis: A comparison of the areas under the two dependent ROC curves (AUCs) was performed using the method described by Hanley and McNeil,[21] and Obuchowski and McClish.[22] The analysis was conducted with PASS 2021 software, and a posteriori power analysis was carried out. A total of 159 cases were evaluated. The AUC values were 0.985 for the first diagnostic test and 0.891 for the second diagnostic test, with a difference (ΔAUC) of 0.094. This difference was found to be statistically significant. A two-sided z-test was applied with a significance level of α=0.05, and the sample size achieved a statistical power of 98% to detect the observed difference.

Results

Data from 159 patients who underwent chemotherapy for testicular germ cell tumour were analysed. The mean age of the cohort was 30.4±10.2 yr, and the mean body mass index was 25.8±4.00 kg/m². Of these, 113 patients (71.1%) were classified into Group 1, while 46 patients (28.9%) were classified into Group 2. The median follow-up duration for the entire cohort was 47 months (range: 8–142 months) (**Table I**).

Pathological subtypes in the entire patient cohort were distributed as follows: seminoma (40.3%), non-seminoma (18.9%), and mixed germ cell tumours (40.9%). No significant differences were observed between the two groups in terms of pathological type, pathological T stage, clinical N stage, S stage, or overall TNM stage (P>0.05). Although a higher proportion of patients with M1a and M1b stages was observed in Group 2, the difference between groups was borderline insignificant (P=0.055). The number of chemotherapy cycles was significantly greater in Group 2 (P= 0.045). Progression and mortality rates were 10.6% and 1.8% in Group 1, and 26.1% and 8.7% in Group 2, respectively. These differences in progression and mortality rates were statistically significant (P=0.023 and P=0.038, respectively). A comparative analysis of pathological characteristics and staging data is presented in **Table I**.

Kaplan-Meier curves for progression-free survival and overall survival are shown in **Supplementary Figure 2**. Cox regression analysis was conducted to identify predictors of progression and mortality. Multivariable analysis for progression revealed three factors significantly associated with disease progression (**Table II**). A PMI change greater than 10 % [hazard ratio (HR)=6.499, P<0.001], rete testis invasion (HR=3.459, P=0.007), and the presence of non-seminoma or mixed germ cell tumours (HR=5.777, P=0.020) were identified as independent predictors of progression. **Figure** presents the ROC curve analysis of PMI and SMI changes for predicting disease progression. The AUC values (95% confidence interval) were 0.985 (0.951–0.998) for PMI change and 0.891 (0.832–0.935) for SMI change. Exact cutoffs with sensitivity/specificity values are given in **Table III**. Comparison of the AUC values revealed a marginally significant difference between PMI and SMI changes (P=0.038). In Cox regression analysis for mortality prediction, both univariate (HR=5.215, P=0.047) and multivariable (HR=5.994, P=0.049) analyses indicated that only a PMI change greater than 10% was statistically significant (**Table IV**).

Discussion

In this study, reductions in PMI following first-line chemotherapy were identified as independent

Table I. Comparison of categorical variables of patients based on psoas muscle index change (N=318)

Characteristic	All patients (n=159), n (%)	Group 1 (n=113), n (%)	Group 2 (n=46), n (%)	*P* value
Age (yr)	30.37 ± 10.24	29.72 ± 10.26	31.96 ± 10.12	0.081*
BMI (kg/m^2)	25.77±4	26.03±4.07	25.12±3.81	0.172*
Rete testis invasion	79 (49.7)	55 (48.7)	24 (52.2)	0.822*
Lymphovascular invasion	78 (49.1)	51 (45.1)	27 (58.7)	0.169*
Pathologic type				0.249++
Seminoma	64 (40.3)	50 (44.2)	14 (30.4)	
Non-seminoma	30 (18.9)	19 (16.8)	11 (23.9)	
Mixed germ cell	65 (40.9)	44 (38.9)	21 (45.7)	
Progression status				0.023*
Non-progressive	135 (84.9)	101 (89.4)[a]	34 (73.9)[b]	
Progressive	24 (15.1)	12 (10.6)[a]	12 (26.1)[b]	
*Progression time (months)	34.42 ± 24.99	37.58 ± 28.53	31.71 ± 22.26	0.561+
Mortality				0.038+
Absent	153 (96.2)	111 (98.2)[a]	42 (91.3)[b]	
Present	6 (3.8)	2 (1.8)[a]	4 (8.7)[b]	
Postoperative adjuvant therapy				0.682++
Carboplatin	18 (11.3)	15 (13.3)	3 (6.5)	
BEP	133 (83.6)	93 (82.3)	40 (87.0)	
VIP	5 (3.1)	3 (2.7)	2 (4.3)	
Other	3 (1.9)	2 (1.8)	1 (2.2)	
Number of cycles				0.045++
<3	44 (27.7)	31 (27.4)[a]	13 (28.3)[a]	
3 cycles	45 (28.3)	38 (33.6)[a]	8 (17.4)[b]	
>3	70 (44.0)	44 (38.9)[a]	25 (54.3)[b]	
Pathological T stage				0.310++
T1	63 (39.6)	47 (41.6)	16 (34.8)	
T2	72 (45.3)	51 (45.1)	21 (45.7)	
T3	21 (13.2)	12 (10.6)	9 (19.6)	
T4	3 (1.9)	3 (2.7)	0 (0.0)	
Clinical N stage				0.441++
N0	60 (37.7)	44 (38.9)	16 (34.8)	
N1	39 (24.5)	29 (25.7)	10 (21.7)	
N2	32 (20.1)	19 (16.8)	13 (28.3)	
N3	28 (17.6)	21 (18.6)	7 (15.2)	
Clinical M stage				0.055++
M0	131 (82.4)	98 (86.7)[a]	33 (71.7)[b]	
M1a	17 (10.7)	10 (8.8)[a]	7 (15.2)[a]	
M1b	11 (6.9)	5 (4.4)[a]	6 (13.0)[a]	
S stage				0.469++
S0	40 (25.2)	29 (25.7)	11 (23.9)	
S1	65 (40.9)	46 (40.7)	19 (41.3)	
S2	44 (27.7)	33 (29.2)	11 (23.9)	
S3	10 (6.3)	5 (4.4)	5 (10.9)	

(*contd...*)

Characteristic	All patients (n=159), n (%)	Group 1 (n=113), n (%)	Group 2 (n=46), n (%)	*P* value
TNM stage				0.223[++]
I	54 (34.0)	43 (38.1)	11 (23.9)	
II	53 (33.3)	36 (31.9)	17 (37.0)	
III	52 (32.7)	34 (30.1)	18 (39.1)	
IGCCCG classification				0.135[++]
Good	58 (36.5)	43 (38.1)	15 (32.6)	
Intermediate	36 (22.6)	22 (19.5)	14 (30.4)	
Poor	19 (11.9)	11 (9.7)	8 (17.4)	

Group 1 <10% change in the psoas muscle index, Group 2 ≥10% change in the psoas muscle index.
[*]Continuity correction test, [+]Fisher's exact chi-square test, [+]Independent-samples*t*-test, [++]Pearson chi-square test.
[*]Progression time (all patients, n=26; Group 1, n=12; Group 2, n=14)
BMI, body mass index; BEP, bleomycin, etoposide, cisplatin; VIP, etoposide, ifosfamide, cisplatin; IGCCG, International germ cell cancer consensus group

Table II. Univariate and multivariable Cox regression analyses for predicting disease progression

	Univariate		Multivariable	
Variables	HR (95%CI)	*P* value	HR (95%CI)	*P* value
Age (yr)	1.027 (0.976–1.081)	0.306	---	---
BMI (kg/m^2)	0.873 (0.761–1.001)	0.052	---	---
PMI change				
<10% (n=113)	Reference		---	---
≥10% (n=46)	3.632 (1.556–8.479)	0.003	6.499 (2.428–17.399)	<0.001
Rete testis invasion				
Absent (n=80)	Reference		---	---
Present (n=79)	2.513 (1.074–5.878)	0.034	3.459 (1.401-8.540)	0.007
Lymphovascular invasion				
Absent (n=81)	Reference		---	---
Present (n=78)	1.636 (0.698–3.830)	0.257	---	---
Pathology				
Seminoma (n=64)	Reference		---	---
Non-seminoma and mixed germ cell (n=95)	4.819 (1.123–20.684)	0.034	5.777 (1.322–25.242)	0.020
Number of cycles				
≤3 (n=89)	Reference		---	---
>3 (n=70)	4.315 (1.266–14.704)	0.019	1.495 (0.303-7.362)	0.621
Stage				
2 (n=53)	Reference		---	---
3 (n=52)	2.207 (0.794–6.139)	0.129	---	---
First-line pre-chemotherapy prognosis stage				
Good or intermediate (n=94)	Reference		---	---
Poor (n=19)	2.639 (1.060–6.568)	0.037	2.002 (0.717-5.595)	0.185

HR, hazard ratio; CI, confidence interval; PMI, psoasmuscle index

risk factors for both progression and mortality. PMI change was also found to be a more precise predictive tool than SMI. Mitsui *et al*[14] analysed data from 50 patients undergoing first-line chemotherapy for testicular germ cell tumour and investigated the relationship between progression and changes in skeletal muscle indices after chemotherapy. In their study, the SMI index—also utilised in our study—was

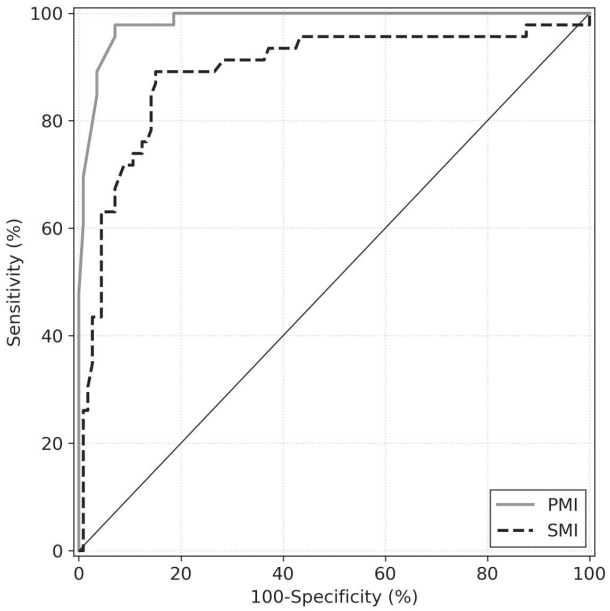


Figure. ROC curve analysis comparing the predictive ability of PMI and SMI changes for progressive disease. ROC, receiver operating characteristic.

can also serve as a reliable surrogate for total skeletal muscle mass.[24] In our study, despite the substantial correlation between SMI and PMI, PMI was selected as the primary evaluation metric owing to its practical measurement, simpler clinical applicability, and statistically superior predictive performance.

Many studies define sarcopenic patients by setting a threshold value for sarcopenia and evaluating prognosis accordingly.[10-12] However, studies similar to ours examined the kinetics of indices representing sarcopenia in relation to prognosis.[13-17,25] Fukushima *et al*[25] analysed SMI kinetics in patients with metastatic RCC undergoing cytoreductive nephrectomy and demonstrated that increasing SMI positively affected overall survival. In another study, Miyake *et al*[13] examined the effects of abdominal skeletal muscle and adipose tissue on prognosis in patients undergoing radical cystectomy for urothelial carcinoma. Similar to our study, they adopted a 10 % cut-off for muscle volume loss. While sarcopenia (HR 2.2, *P*=0.03) and psoas volume loss (HR 2.4, *P*=0.02) were independent

Table III. Cut-off points, AUC, sensitivity, specificity, PPV, NPV and accuracy of PMI and SMI

Index/Score	Cut-off	AUC (95%CI)	Sensitivity (%)	Specificity (%)	PPV (%)	NPV (%)
PMI	≤-0.55	0.985 (0.97-0.99)	97.8	92.9	84.9	99.1
SMI	≤-3.5	0.891 (0.83-0.95)	89.1	84.9	70.7	95.0

AUC, area under curve; PPV, positive predictive value; NPV, negative predictive value; SMI, skeletal muscle index

assessed alongside psoas muscle volume (PMV), measured using automated volume analysis software on CT images. Their univariate analysis revealed that poor risk classification according to the IGCCCG, metastases excluding the lungs, and PMV change were significant predictors of progression. However, in multivariate analysis, only PMV change remained significant (HR=4.681, *P*=0.033). PMV change was identified as a more accurate predictive factor than SMI change (AUC=0.832 *vs*. 0.695), consistent with our findings. Similarly, Baky *et al*[15] evaluated patients undergoing post-chemotherapy retroperitoneal lymph node dissection and concluded that reductions in PMI were predictive of postoperative morbidity.

SMI is widely accepted in sarcopenia-related studies due to its strong correlation with total body muscle mass.[9] However, many studies have assessed SMI alongside PMI, utilising both as indicators of sarcopenia.[13,14,18,23] Research demonstrating a strong correlation between PMI and SMI suggests that PMI predictors of overall survival, neither was significant for disease-specific survival. Phuong *et al*[16] evaluated changes in skeletal muscle and adipose tissue in 141 patients undergoing cisplatin-based chemotherapy for testicular germ cell tumours and investigated the association with chemotherapy-associated adverse events. While pre-chemotherapy skeletal muscle indicators and post-chemotherapy SMI were not associated with adverse events, a decrease in SMI (odds ratio: 0.89, *P*=0.02) was independently associated with a higher incidence of adverse events. Similarly, a multicentre study by Buxton *et al*[17] demonstrated that reductions in SMI following cisplatin-based chemotherapy were associated with an increased risk of grade 3 adverse events in patients with testicular germ cell tumour.

Even with successful primary cancer treatment, obesity and sarcopenia can adversely affect quality of life, reduce survival, and contribute to metabolic syndrome and secondary malignancies.[23,26] One study

Table IV. Univariate and multivariate Cox regression analyses for predicting mortality

	Univariate		Multivariable	
Variables	HR (95%CI)	*P* value	HR (95%CI)	*P* value
Age (yr)	1.016 (0.936–1.103)	0.704	---	---
BMI (kg/m^2)	0.881 (0.674–1.153)	0.356	---	---
PMI change				
<10% (n=113)	Reference		---	---
≥10% (n=46)	5.215 (0.953–28.529)	0.047	5.994 (1.007–35.677)	0.049
Rete testis invasion	2.699 (0.485–15.014)	0.257	---	---
Absent (n=80)	Reference		---	---
Present (n=79)	2.699 (0.485–15.014)	0.257	---	---
Lymphovascular invasion				
Absent (n=81)	Reference		---	---
Present (n=78)	0.932 (0.187–4.641)	0.931	---	---
Pathology				
Seminoma (n=64)	Reference		---	---
Non-seminoma and mixed germ cell (n=95)	2.698 (0.314–23.212)	0.366	---	---
Number of cycles				
≤3 (n=89)	Reference		---	---
>3 (n=70)	4.622 (0.536–39.885)	0.164	---	---
Stage				
2 (n=53)	Reference		---	---
3 (n=52)	4.181 (0.486–35.960)	0.193	---	---
First-line pre-chemotherapy prognosis stage				
Good or intermediate (n=94)	Reference		---	---
Poor (n=19)	2.805 (0.465–16.936)	0.261	---	---

demonstrated that chemotherapy for testicular cancer altered body composition by reducing lean mass and impairing taste and smell perception.[14] Takai *et al*[23] retrospectively analysed body composition changes in 44 patients with testicular germ cell tumours undergoing systemic chemotherapy. While fat mass progressively increased during treatment, skeletal muscle mass initially declined but returned to baseline levels by the 12th month.[23] Their study also identified an association between the number of chemotherapy cycles and skeletal muscle loss. Another study showed that three cycles of bleomycin-etoposide-cisplatin therapy reduced muscle fibre size and strength, indicating a direct detrimental effect of chemotherapeutic agents on skeletal muscle mass.[27] Furthermore, significantly lower levels of free and total testosterone have been reported in patients with testicular germ cell tumour,[28] and the post-chemotherapy disruption in body composition may be linked to these decreased testosterone levels.[4]

Tumour progression, tumour-induced systemic inflammation, and metabolic dysregulation constitute tumour-associated factors, while deterioration of overall condition and functional status reflect host-related factors. Together, these elements position sarcopenia as a distinctive biomarker indicative of tumour aggressiveness.[11] Nutritional support and exercise have been shown to mitigate sarcopenia, with both interventions demonstrating potential oncological benefits. Early initiation of an exercise programme during neoadjuvant therapy has been reported to reduce skeletal muscle loss, as assessed by CT imaging.[29]

In our study, the prognostic value of pathological characteristics was also assessed. Various pathological features have been reported as prognostic indicators for testicular tumours in the literature. For seminoma, prognostic factors typically include the size of the primary tumour and rete testis invasion, whereas lymphovascular invasion is considered the most

शोध-संदेश

इस अध्ययन में अंडकोष में होने वाले जर्म सेल ट्यूमर (testicular germ cell tumour) के उन रोगियों में, जो कीमोथेरेपी (औषधियों द्वारा उपचार) ले रहे थे, मांसपेशियों की कमी (sarcopenia) और रोग की प्रगति व मृत्यु दर के बीच संबंध का मूल्यांकन किया गया। Sarcopenia को नापने के लिए psoas muscle index (PMI) नामक संकेतक का विश्लेषण किया गया। अध्ययन के परिणामों से स्पष्ट हुआ कि PMI में कमी रोग की प्रगति और मृत्यु दोनों के पूर्वानुमान में सहायक सिद्ध हो सकती है। अर्थात, जिन रोगियों में मांसपेशियों का स्तर कम पाया गया, उनमें बीमारी बढ़ने और मृत्यु का जोखिम अधिक था। इन निष्कर्षों की पुष्टि के लिए अधिक रोगियों पर आधारित एवं भविष्य-उन्मुख (प्रॉस्पेक्टिव) अध्ययनों की आवश्यकता है।

important prognostic factor for non-seminomatous germ cell tumours.[30] Embryonal carcinoma histology is also recognised as a poor prognostic factor.[30,31] Although our study did not analyse embryonal carcinoma as a distinct subgroup, non-seminomatous and mixed histological types, which encompass embryonal carcinoma, were identified as risk factors for progression in multivariable analysis. Similarly, *rete testis* invasion emerged as an independent predictor of progression. While lymphovascular invasion was significant in univariate analysis, it did not retain significance in the multivariable model, possibly due to the limited sample size affecting statistical power.

The number of studies investigating the impact of skeletal muscle loss on progression and survival in patients with testicular germ cell tumour remains limited.[14] Our study represents the first multicentre investigation in this field and is notable for its relatively larger patient cohort. Nevertheless, the sample size still constitutes a potential limitation. Additional limitations include the retrospective study design and heterogeneity in pathological subtypes. Patients received varying numbers of chemotherapy cycles and regimens, but due to insufficient patient numbers, a statistically significant subgroup analysis cannot be performed. Additionally, aging, comorbid chronic diseases, prolonged inactivity, and malnutrition can impair skeletal muscle health.[5] These data are not consistently available for all patients, and their effects on sarcopenia could not be examined. Mortality occurred in only 6 (3.8%) patients, which reduces the statistical power of multivariable analysis, reduces the reliability of hazard ratios, and may lead to overfitting.

In conclusion, for patients with testicular germ cell tumour undergoing systemic chemotherapy, a reduction in skeletal muscle mass serves as a prognostic factor for both disease progression and mortality. PMI change, which can be measured using CT imaging, represents a simple and reliable method for assessing sarcopenia.

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