Original Article

Association between Sarcopenia and Anthracycline-Induced Cardiotoxicity in Patients with Cancer

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Highlights

- Postchemotherapy sarcopenia was present in 46% of patients.
- Sarcopenia was associated with adverse echocardiographic findings.
- CT-based muscle assessment and handgrip strength may aid cardio-oncology risk assessment.
- Sarcopenia may help identify patients vulnerable to anthracycline-related cardiac dysfunction.

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ABSTRACT

Background: Anthracycline-based chemotherapy is associated with cancer therapy–related cardiac dysfunction, primarily manifested by a decline in left ventricular ejection fraction (LVEF). Sarcopenia, characterized by reduced skeletal muscle mass and strength, shares mechanistic pathways with anthracycline cardiotoxicity, including mitochondrial dysfunction, oxidative stress, and systemic inflammation. We aimed to investigate the association between postchemotherapy sarcopenia and changes in cardiac function in a heterogeneous cohort of patients with cancer.

Methods: In this single-center prospective observational study, 163 adult patients scheduled to receive anthracycline-containing chemotherapy were enrolled. Transthoracic echocardiography was performed before treatment and 3 months after completion, with assessment of LVEF using the biplane Simpson method and global longitudinal strain. Sarcopenia was assessed postchemotherapy using computed tomography–derived skeletal muscle index at the L3 level combined with handgrip strength. Cardiotoxicity was defined as a reduction in LVEF of 10% or more to below normal and/or new diastolic dysfunction. Multivariable logistic regression was used to evaluate independent associations.

Results: Sarcopenia was identified in 46% of patients following chemotherapy. Significant differences in echocardiographic outcomes were observed between sarcopenic and nonsarcopenic groups (P<.05). Sarcopenia was strongly associated with cardiotoxicity, with logistic regression demonstrating robust predictive performance (Nagelkerke R^2=0.485; P<.001). Patients with low muscle mass and strength exhibited markedly higher odds of cardiac dysfunction.

Conclusion: Postchemotherapy sarcopenia is independently associated with anthracycline-induced cardiac dysfunction. Integrating CT-based muscle assessment and functional testing into cardio-oncology risk stratification may enhance early identification of vulnerable patients and guide cardioprotective strategies.

Keywords: Sarcipenia; Cardiotoxicity; Cancer

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Introduction

According to the Global Cancer Statistics 2022 (GLOBOCAN estimates), there were nearly 20 million new cancer cases and approximately 9.7 million cancer-related deaths worldwide in 2022, illustrating that cancer remains one of the leading causes of death globally.[1] Nonetheless, recent therapeutic advances, including targeted agents and novel systemic therapies, have contributed to significant reductions in cancer mortality and improved patient survival.[2] Despite these advances, treatment-related complications such as chemotherapy-induced cardiotoxicity remain a major clinical concern. Chemotherapy-induced cardiotoxicity is defined as a toxic effect of anticancer drugs on the heart, leading to left ventricular systolic dysfunction, heart failure (HF), and cardiomyopathy.[3,4] The American Society of Echocardiography (ASE) further defines cardiotoxicity as a reduction in left ventricular ejection fraction (LVEF) of 10% or more to a value below 53%.[5]

Anthracyclines are among the most widely used chemotherapeutic agents in the treatment of both hematologic and solid malignancies.[6] Nevertheless, their use is limited by dose-dependent cardiotoxicity, which can result in irreversible myocardial injury, cardiac dysfunction, and HF, potentially compromising oncologic outcomes through treatment dose reduction or discontinuation.[7,8]

Sarcopenia, defined as the loss of skeletal muscle mass and strength, may develop as a consequence of aging, chronic disease, or systemic inflammatory processes.[9] In patients with cancer, chemotherapy-associated sarcopenia arises from a combination of direct and indirect effects of anticancer treatments on skeletal muscle metabolism and physiology, including systemic inflammation, oxidative stress, mitochondrial dysfunction, and treatment-related adverse effects such as anorexia, nausea, fatigue, and reduced physical activity.[10–12]

Previous studies have demonstrated that sarcopenia is associated with an increased risk of treatment-related adverse events, morbidity, and mortality in patients with cancer.[13] Notably, mitochondrial dysfunction driven by excessive generation of reactive oxygen species (ROS), iron-dependent oxidative stress, lipid peroxidation, and disruption of mitochondrial membrane integrity represents a central feature of anthracycline-induced cardiotoxicity and may constitute a shared pathophysiological mechanism linking sarcopenia and cardiac dysfunction.[14]

Several studies have explored the association between sarcopenia and cardiotoxicity in specific cancer populations, including patients with breast and esophageal cancer receiving anticancer therapies.[15,16] However, although a significant relationship between sarcopenia and anthracycline-related cardiotoxicity has been reported, to our knowledge, no study has specifically evaluated the association between computed tomography (CT)-defined sarcopenia and anthracycline-induced cardiotoxicity in a heterogeneous cohort of patients with different cancer types.[17]

Therefore, this study aimed to investigate the association between sarcopenia assessed after completion of chemotherapy and changes in cardiac function evaluated by echocardiography before and after chemotherapy in patients with cancer.

Methods

Study Design and Population

This single-center, prospective observational study was conducted from September 2024 through July 2025 at Rajaie Cardiovascular Institute, Iran University of Medical Sciences, Tehran, Iran (Ethics Committee approval: IR.RHC.REC.1404.088). Adults aged 18 years or older with a confirmed diagnosis of malignancy who were scheduled to receive anthracycline-based chemotherapy were consecutively enrolled. All participants underwent baseline transthoracic echocardiography before initiation of chemotherapy and follow-up echocardiographic assessment after completion of treatment. Sarcopenia was assessed after completion of chemotherapy using CT imaging performed as part of routine oncologic evaluation. The study was approved by the Ethics Committee of Rajaie Cardiovascular Institute, and written informed consent was obtained from all participants before enrollment. Because CT is an invasive procedure

and was not clinically indicated for baseline sarcopenia assessment in this population, baseline imaging was not performed for ethical reasons. To address potential selection bias related to baseline nutritional status, the study enrolled urban residents with higher socioeconomic status.

Eligibility Criteria

Inclusion criteria were age of 18 years or older, histologically confirmed malignancy, planned anthracycline-containing chemotherapy, available baseline echocardiography, and postchemotherapy CT imaging suitable for sarcopenia assessment. Patients were excluded if they had preexisting severe LV dysfunction (eg, LVEF <50%), prior exposure to anthracyclines, known cardiomyopathy, significant valvular disease, poor-quality CT or echocardiographic images, or concurrent cardiotoxic therapies.

Echocardiographic Assessment

All participants underwent baseline transthoracic echocardiographic assessment before initiation of chemotherapy, and follow-up echocardiography was performed 3 months after completion of chemotherapy. Echocardiographic examinations were conducted using a commercially available ultrasound system (Philips EPIC 7). LVEF was measured using the biplane Simpson method from apical 4- and 2-chamber views, in accordance with current guidelines. Global longitudinal strain (GLS) was assessed using 2D speckle-tracking echocardiography, with strain analysis performed from standard apical views. All echocardiographic measurements were performed by experienced cardiologists who were blinded to the participants' clinical data and sarcopenia status.

In this study, cardiotoxicity was defined as systolic dysfunction based on a reduction in LVEF of 10% or more from baseline to a value below the lower limit of normal, consistent with contemporary cancer therapy–related cardiac dysfunction definitions, and as diastolic dysfunction according to ASE criteria using standard echocardiographic Doppler and tissue velocity parameters.[18,19] Studies with inadequate image quality for strain analysis were excluded.

Sarcopenia Assessment

Sarcopenia was assessed using CT imaging obtained as part of routine oncologic evaluation. CT examinations were performed after completion of chemotherapy using a 32-slice CT scanner (SOMATOM go.Now, Siemens Healthineers, Germany), with a gantry aperture of 70 cm. Images were acquired using standardized acquisition parameters, including a tube voltage ranging from 70 to 130 kV and tube current up to 345 mA, with a rotation time of up to 0.8 seconds. Axial images were reconstructed using standard soft-tissue reconstruction algorithms, with a field of view of up to 500 mm.

Cross-sectional skeletal muscle area was measured on a single axial CT slice at the level of the third lumbar vertebra (L3), a validated anatomical landmark that correlates strongly with whole-body skeletal muscle mass. The L3 slice was selected at the midvertebral level, defined as the image in which both transverse processes were clearly visible. Skeletal muscle was segmented bilaterally, including the psoas, paraspinal muscles (erector spinae and quadratus lumborum), and abdominal wall muscles (transversus abdominis, internal and external obliques, and rectus abdominis). Skeletal muscle was identified using predefined Hounsfield unit thresholds ranging from −29 to 150 HU. The total skeletal muscle area was calculated in square centimeters (cm^2) and normalized to patient height squared (m^2) to derive the skeletal muscle index (SMI, cm^2/m^2). Sarcopenia was defined according to previously validated sex-specific SMI cutoff values.

Muscle strength was assessed using handgrip strength testing with a calibrated handheld dynamometer (manufacturer, model, country). Handgrip strength was measured in the dominant hand with the participant in a seated position, the elbow flexed at 90°, and the forearm in a neutral position, according to standardized protocols. Participants performed 3 maximal voluntary contractions with brief rest intervals, and the highest recorded value was used for analysis. Sarcopenia was defined as the presence of low skeletal muscle mass in combination with low muscle strength.

All CT image analyses and handgrip strength

measurements were performed by trained personnel who were blinded to the participants' clinical, oncologic, and echocardiographic data.

Other Clinical and Laboratory Assessments

Baseline demographic and clinical data-including underlying diseases, type of cancer, chemotherapy regimen, anthracycline type and dosage, and baseline laboratory data-were extracted from the participants' electronic profiles.

Outcomes

The primary outcome of this study was the association between postchemotherapy sarcopenia and chemotherapy-related cardiotoxicity in patients with cancer. Cardiotoxicity was defined as systolic dysfunction based on European Society of Cardiology criteria and/or diastolic dysfunction according to ASE guidelines. Secondary outcomes included the association between postchemotherapy sarcopenia and changes in echocardiographic parameters, including LVEF, GLS, and indices of diastolic function, assessed before and after completion of chemotherapy; the incidence and prevalence of sarcopenia in patients with cancer following chemotherapy; and the association between postchemotherapy sarcopenia and health-related quality of life, assessed using a validated patient-reported outcome questionnaire.

Statistical Analysis

Statistical analyses were performed using IBM SPSS version 27.0. Crosstabulation analysis was used to examine the distribution of data and the percentage of outcome occurrence. The χ^2 test was employed to examine the significance of differences between groups. Odds ratios (ORs) were calculated to quantify the odds of outcome occurrence between the 2 groups. Finally, logistic regression analysis was performed to identify independent predictors and assess relationships among variables.

Results

The study included 163 patients with cancer who were candidates for chemotherapy. There were 98 women (mean 13.15 age, 60.4 years) and 65 men (mean 9.68 age, 58.4 years).[1] Of the 163 patients, 75 (46.0%) developed sarcopenia, and 88 (54.0%) did not.

Crosstabulation with χ^2 analysis was performed to examine group differences (Table 1).

Table 1. PMI_cut_sex * ECHO Crosstabulation

			ECHO	
		Norm	Abnor	Total
PMI_cut_sex With Sarcopenia	Count	84	4	88
	% Within PMI_cut_sex	95.5%	4.5%	100.0%
Without Sarcopenia	Count	27	48	75
	% Within PMI_cut_sex	36.0%	64.0%	100.0%
Total	Count	111	52	163
	% Within PMI_cut_sex	68.1%	31.9%	100.0%

Chi-Square Tests

	Value	df	Asymptotic Significance (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	65.883[a]	1	<.001		
Continuity Correction[b]	63.175	1	<.001		
Likelihood Ratio	73.561	1	<.001		
Fisher's Exact Test				<.001	<.001
Linear-by-Linear Association	65.479	1	<.001		
N of Valid Cases	163				

a. 0 cells (0.0%) have expected count less than 5. The minimum expected count is 23.93.

b. Computed only for a 2x2 table

According to the results of the χ^2 analysis, there was a significant difference in the echocardiographic results between the 2 groups (P<.05).

Individuals above the sex-specific psoas muscle index (PMI) cutoff had markedly higher odds of sarcopenia (OR, 37.3; 95% CI, 12.3 to 113.1). The overall logistic regression model was statistically significant (Omnibus test of model coefficients: χ^2=73.56; df=1; P<.001), indicating that the model including PMI category provided a significantly better fit than the null model. The model demonstrated substantial explanatory power, accounting for approximately 48.5% of the variation in sarcopenia status (Nagelkerke R^2=.485).

Individuals with PMI values above the sex-specific cutoff had markedly higher odds of sarcopenia than those below the cutoff (OR, 37.33; 95% CI, 12.32 to 113.09; P<.001). This association remained robust, with the confidence interval entirely above 1, which indicated a strong and statistically significant relationship. Using a probability cutoff value of 0.5, the model correctly classified 81.0% of cases overall, with a specificity of 95.5% and a sensitivity of 64.0% for the detection of sarcopenia (Table 2).

Table 2. Variables in the Equation

		B	S.E.	Wald	df	Sig.	Exp(B)	95% C.I. for EXP(B)	
								Lower	Upper
Step 1[a]	ECHO	3.620	.565	40.977	1	<.001	37.333	12.324	113.094
	Constant	-4.755	.683	48.454	1	<.001	.009		

a.Variable(s) entered on step 1: ECHO.

An interaction term between sarcopenia and trastuzumab use was added to the multivariable logistic regression model. The interaction was not statistically significant (OR, 0.093; 95% CI, 0.041 to 6.580; P=.13), indicating no evidence that the association between sarcopenia and cardiotoxicity differed between patients receiving trastuzumab and those with other malignancies (Table 3).

Table 3. Variables in the Equation

		B	S.E.	Wald	df	Sig.	Exp(B)
Step 1[a]	PMI_cut_sex	4.893	1.835	7.106	1	.008	133.308
	breast_cancer	-.511	1.177	.188	1	.664	.600
	interaction	-.659	1.297	.258	1	.612	.518
	Constant	-2.380	1.562	2.320	1	.128	.093

		95% C.I.for EXP(B)	
		Lower	Upper
Step 1[a]	PMI_cut_sex	3.652	4866.409
	breast_cancer	.060	6.025
	Interaction	.041	6.580
	Constant		

doxorubicin mean dose per usage	doxorubicin SD	Use of dexrazoxane	concomitant cardiotoxic therapies
67.5 mg/m^2	7.48	No	Trastuzumab for breast cancer

The mean usage of doxorubicin per dose was 67.5 mg/m^2, and each patient was given doxorubicin about 4-6 doses due to cancer type.

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Discussion

Overall Interpretation of Results

Nearly half of anthracycline-treated patients developed sarcopenia. A recent meta-analysis of 26 studies reported that sarcopenia prevalence in patients with esophageal cancer receiving neoadjuvant therapy (which includes chemotherapy) was 48%.[20] A comprehensive review of studies in patients with cancer (not limited to 1 type) reported a baseline sarcopenia prevalence ranging from 14% to 78.7%, with many studies showing prevalence above 30% to 40% after treatment exposure. This indicates that sarcopenia is common before and increases after chemotherapy.[21]

Sarcopenia was significantly associated with adverse echocardiographic changes. Bas et al[17] used CT to quantify skeletal muscle mass at the third lumbar vertebra (L3) level (L3-SMI) and PMI before anthracycline treatment. They found that patients with low muscle mass had a markedly higher incidence of subsequent cardiotoxicity. A recent study in patients with lymphoma found that CT-derived myosteatosis was an independent predictor of cardiotoxicity. After adjustment for conventional risk factors, myosteatosis conferred approximately 3.4-fold greater odds of anthracycline-induced cardiac dysfunction.[22]

Pro-inflammatory cytokines (tumor necrosis factor α [TNF-α] and interleukin 6 [IL-6]) activate nuclear factor κB (NF-κB), which upregulates muscle-specific E3 ubiquitin ligases (muscle RING finger protein 1 [MuRF-1] and muscle atrophy F-box protein [MAFbx]/atrogin) and the ubiquitin-proteasome system, accelerating myofibrillar protein degradation.[23,24] Concomitantly, aging and chronic illness impair mitochondrial quality control: defective biogenesis and mitophagy lead to accumulation of dysfunctional mitochondria, elevating ROS and energy deficits in muscle fibers.[24] These ROS further amplify inflammation and proteolysis, creating a vicious cycle of muscle atrophy.[24] In anthracycline-treated myocardium, similar stressors are triggered by the drug's pharmacology. Anthracyclines intercalate cardiac DNA and poison topoisomerase 2β (Top2β), leading to mitochondrial DNA damage and suppression of peroxisome proliferator–activated receptor γ coactivator 1α/β (PGC-1α/β), master regulators of mitochondrial biogenesis.[25]

In individuals with established heart disease, sarcopenia is associated with worse cardiac function and outcomes. For instance, the loss of muscle mass and strength is more prevalent in heart failure with preserved ejection fraction (HFpEF), and sarcopenia predicts higher 1-year mortality in both HFpEF and heart failure with reduced ejection fraction (HFrEF).[26] In patients with cancer, sarcopenia correlates with frailty and chemotoxicity. Meta-analyses show that frail (sarcopenic) patients have higher all-cause mortality, treatment toxicity, and intolerance.[27]

Clinical and Guideline Implications

The foregoing evidence has several practical implications. First, sarcopenia assessment (via imaging or functional tests) could be incorporated into baseline risk stratification for cardiotoxic chemotherapy. CT-based muscle metrics (eg, L3-SMI, psoas area, and muscle attenuation) are readily available on staging scans.[22] Simple measures of strength, such as handgrip dynamometry or gait speed, might also serve as surrogate markers of muscle reserve. Identifying individuals with sarcopenia could flag a subgroup needing "permissive cardiotoxicity" strategies. By way of example, these patients might be considered for prophylactic cardioprotective therapy (angiotensin-converting enzyme [ACE] inhibitors, β-blockers, statins, and dexrazoxane) as recommended by guidelines for high-risk patients.[28] Indeed, the 2022 European Society of Cardiology (ESC) Cardio-Oncology Guidelines explicitly advise prophylaxis with ACE inhibitors or angiotensin receptor blockers, β-blockers, statins, and/or liposomal anthracyclines or dexrazoxane in patients at very high risk of toxicity.[28] We propose that sarcopenia, reflecting systemic vulnerability, be viewed as an additional high-risk feature. Finally, recognizing sarcopenia underscores the value of multidisciplinary care. Interventions such as tailored exercise training, nutrition, and physical therapy-which improve muscle mass and function-might not only enhance quality of life but could theoretically bolster cardiac reserve.

Echocardiographic Surveillance and Imaging

In all patients with cancer, standardized imaging is critical. Guidelines define cardiotoxicity,

or cancer therapy–related cardiac dysfunction, as a reduction in LVEF of 10% or more to below the lower limit of normal (typically <50%), or a relative decrease in LV GLS of more than 15%.[29] Both American Society of Echocardiography/European Association of Cardiovascular Imaging (ASE/EACVI) consensus recommendations and recent reviews confirm that GLS is an independent predictor of eventual LVEF reduction in patients receiving cardiotoxic therapy.[29] In practice, patients with sarcopenia, who may have subclinical myocardial damage, could particularly benefit from strain surveillance to detect early dysfunction.

Conclusions

Postchemotherapy sarcopenia is strongly associated with anthracycline-induced cardiac dysfunction. CT-based muscle assessment and functional testing may enhance cardio-oncology risk stratification. Recognizing sarcopenia as a marker of systemic vulnerability could guide intensified surveillance and cardioprotective strategies in high-risk patients with cancer.

Declarations:

Ethical Approval

The Research Committee of Rajaie Hospital in Tehran has ethically approved this study. (Ethics Committee approval: IR.RHC.REC.1404.088).

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Conflict of Interest

The authors report no conflict of interest.

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