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ORIGINAL RESEARCH

The Role of Diaphragmatic Ultrasound in Identifying Sarcopenia in COPD Patients: A Cross-Sectional Study

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Background/Aim: Chronic obstructive pulmonary disease (COPD) is often complicated by sarcopenia, a condition of reduced muscle mass and function that adversely affects quality of life, lung function, and exacerbation rates. Ultrasonography could be an effective tool for detecting sarcopenia, notably by assessing diaphragmatic function, which may indicate muscle health in COPD patients. This study aims to evaluate the effectiveness of diaphragmatic ultrasound in detecting sarcopenia among COPD patients.

Materials and Methods: Thirty-five patients with COPD, with a forced expiratory volume in one second (FEV1) between 30% and 80%, were consecutively enrolled in this cross-sectional and double-blind study. Sarcopenia was defined using the European Working Group on Sarcopenia in Older People 2 (EWGSOP2) criteria. Muscle mass was assessed with bioelectrical impedance analysis (BIA), muscle strength was assessed using the handgrip test and physical performance was assessed using a 4-meter gait speed test. Pulmonary function tests (PFT) (including maximum inspiratory pressure-MIP and maximum expiratory pressure-MEP) were performed. Diaphragm excursion and thickness at residual volume, functional residual capacity, and total lung capacity were measured using ultrasound. The diaphragm thickening fraction was calculated during normal (TF) and deep breathing (TLC-TF).

Results: Seventeen of 35 patients (48.6%) were found to be sarcopenic. Diaphragm thickness did not show significant variation between the groups. Both TF (27.43%) and TLC-TF (39.7%) were found to be lower in the sarcopenic group ($p<0.05$). The diaphragmatic excursion in the sarcopenic group was found to be 1.38 cm ($p=0.078$). There was no difference in median MIP and MEP values between the groups.

Conclusion: Diaphragmatic TF may be a valuable tool for detecting sarcopenia in COPD patients, which may vary independently of PFTs. This study highlights TF as a potential auxiliary measure, but further research with larger sample sizes and additional parameters is needed to confirm its clinical utility.

Keywords: excursion, frailty, obesity, respiratory failure, FEV1

Introduction

A multisystem and complicated illness, chronic obstructive pulmonary disease (COPD) presents a wide range of intra and extrapulmonary complaints.[1,2] It is becoming more well-acknowledged that extrapulmonary signs play a significant role in the functional decline observed in COPD patients.[3] Participants with COPD or during acute exacerbations of the disease may experience a more significant loss of muscle mass and strength, especially in the lower limbs.[4] A variety of factors, such as restricted movement, abnormalities in nutrition and energy, inflammation, hypercapnia, hypoxemia, electrolyte imbalances, and use of drugs like steroids, can cause muscle dysfunction. Muscle dysfunction has been linked to several unfavourable outcomes during a COPD exacerbation, including death, more extended hospital stays, and readmissions.

Sarcopenia manifests as a broader decline in skeletal muscle mass and strength, encompassing respiratory muscles rather than solely impacting the upper and lower limbs. Chronic inflammation-causing disorders like COPD or other underlying diseases can lead to sarcopenia.[5] Although sarcopenia has therapeutic implications for respiratory failure care and pulmonary

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rehabilitation in COPD patients, making its early identification potentially crucial, it still has not gained sufficient importance in the GOLD guidelines.

In the context of COPD, the standard tests defined in the sarcopenia consensus criteria should be applied, and the established cutoff points should be utilized. Combined use of respiratory function parameters such as Peak Expiratory Flow (PEF), maximum inspiratory pressure (MIP), and maximum expiratory pressure (MEP), which are indicative of respiratory sarcopenia, is recommended.[6,7] It is considered that diaphragm ultrasound may reveal respiratory dysfunction better than pulmonary function tests (PFT).[8] Previous studies have demonstrated that diaphragm thickness decreases in sarcopenic individuals as measured by ultrasound.[9] In this study, we evaluated the ability of diaphragm thickening fraction, thickness, and excursion to predict sarcopenia in individuals with COPD.

Materials and Methods

Participants

This study was designed as a cross-sectional, observational cohort study and conducted in the Department of Respiratory Diseases at Erciyes University. The study included patients aged ≥40 years who were admitted to a tertiary health center between March 2023 and May 2024. All procedures were performed in accordance with the ethical standards of the institution and the principles outlined in the Declaration of Helsinki. Ethical approval was obtained from the Ethics Committee of Erciyes University (Meeting Date: 08/03/2023, Decision Number: 2023/152). A total of 186 patients were prospectively evaluated for inclusion in the study. Patients were eligible if they had a confirmed COPD diagnosis by a specialist and either presented for follow-up or exhibited respiratory symptoms such as dyspnea and cough, along with risk factors in accordance with the GOLD guideline for COPD. Eligibility also required a post-bronchodilator spirometry result showing a forced expiratory volume in one second (FEV1) to forced vital capacity (FVC) ratio of less than 70%. Patients with an FEV1 value between 30–80% were consecutively enrolled. Exclusion criteria included individuals with known congestive heart failure, neuromuscular disease, history of cerebrovascular events, recent use of systemic steroids for a COPD exacerbation within the last month, and those receiving home oxygen therapy or noninvasive mechanical ventilation. Patients with conditions such as apnea, active malignancy, or use of medications affecting bone metabolism or muscle strength were also excluded. Furthermore, individuals unable to cooperate with deep inspiration and forced expiration manoeuvres and those for whom bioimpedance analysis (BIA) was contraindicated (eg, presence of a cardiovascular stent, pacemaker, joint prosthesis, or visible oedema) were omitted. After applying these exclusion criteria, 35 COPD patients were included in the final analysis. Informed consent was obtained from all patients who agreed to participate in the observational analytical study.

Respiratory Function Tests

Spirometry was used to quantify the FVC, FEV1, and FEV1/FVC (Vmax, ENCORE, Sensormedics, Yorba Linda, California, USA). Separate trials of spirometry were conducted while sitting and while lying down. A percentage was used to represent the change in FVC. Tests were conducted by a single, skilled respiratory physiotherapist using the guidelines provided by the American Thoracic Society (ATS) and European Respiratory Society (ERS).[10] To ensure the consistency of all spirometric parameters, three acceptable spirometry measurements were obtained in accordance with the guidelines of the ATS and the ERS. For each measurement, the best-recorded value was used for analysis, performed by a qualified respiratory physiotherapist in adherence to the established standards. This method was implemented to enhance the reproducibility and reliability of the spirometric parameters. The accuracy of the spirometry data obtained in this study was improved through this protocol.

Using a respiratory dynamometer (Vmax, ENCORE, Sensormedics, Yorba Linda, California, USA), MIP and MEP, which are used to quantify respiratory muscle force, were assessed by the ERS/ATS suggested technique.[10] Each measurement of MIP and MEP was performed three times in accordance with the ATS/ERS guidelines to ensure accuracy and reproducibility. The values of MEP and MIP are presented as percentages of the expected values.

Diaphragm Ultrasound

The ultrasound examination was performed by an experienced pulmonologist (NAY) with 12 years of ultrasound experience who has conducted studies in this field.[8] An ultrasound device was utilized to assess the indices of the diaphragm (FC1, Fujifilm-Sonosite, WA, USA). The mean value was finally calculated after recording each determined parameter three times.

For diaphragmatic excursion, we use a convex probe transducer (2–5MHz). Diaphragm movements were recorded in B-Mode while the participants were performing calm breathing. Ultrasonic waves were placed in the subcostal area towards the midpoint of the clavicle, allowing for vertical visualization of the diaphragm, followed by switching to the M mode.

We assessed the diaphragm thickness in the opposition zone using B-mode ultrasound imaging. A 6–13 MHz linear transducer was positioned on the chest wall in the eighth or ninth right intercostal space, between the anterior–axillary and midaxillary lines, while the subject was seated at a 30- to 45-degree angle.[11] The thickness of the diaphragm was measured during quiet breathing at both the expiratory (Functional residual capacity (FRC- Tdi) and the end of inspiration (TV-Tdi). Diaphragm thickness was recorded at the level of forced inspiration with total lung capacity (TLC-Tdi) and forced expiration at residual volume (RV-Tdi). The diaphragm thickening fraction was calculated using the following formula: TF = [(TV Tdi–FRC Tdi)/FRC Tdi] × 100. Similarly, the TLC-TF was calculated using the RV-Tdi and TLC-Tdi values.

Sarcopenia Definition

All geriatric assessments and anthropometric measurements were performed at baseline by a physician experienced in geriatric assessment. Obesity was defined as BMI ≥30 kg/m^2. We defined sarcopenia as low muscle strength plus low muscle mass consistent with The European Working Group on Sarcopenia in Older People 2 (EWGSOP 2) recommendation.[5] According to its 2018 definition, EWGSOP2 identifies reduced muscle strength as the principal criterion for sarcopenia; muscle strength is currently the most dependable indicator of muscle function. Sarcopenia is likely to occur when diminished muscle strength is observed. The identification of diminished muscle mass or functionality establishes a diagnosis of sarcopenia. Sarcopenia is classified as severe when there is a concomitant presence of diminished muscle strength, reduced muscle quantity or quality, and impaired physical performance. Muscle strength was assessed using a handgrip dynamometer (Takei TKK5401 Digital Handgrip Dynamometer, Niigata-City, Japan) in the sitting position with the elbow flexed. The mean value of three measurements in the dominant arm taken at least at one-minute intervals was recorded. Skeletal muscle mass (SMM; kilograms) was calculated using the BIA (Bodystat Quad Scan 1500, UK) equation by Janssen et al by using impedance (ohms) values obtained. It was calculated as follows: SMM (kg) = ([height^2/BIA resistance × 0.401] + [gender × 3.825] + [age × −0.071]) + 5.102. (Height in centimetres; resistance in ohms; sex: female = 0, male = 1; age in years.)

The SMM (kg) was normalized for squared height and defined as skeletal muscle mass index (SMMI) (kg/m^2). SMM was adjusted by BMI since adjustment SMM by height^2 in overweight and obese older adults, BMI ≥ 30 kg/m^2 for both genders, may underestimate sarcopenia. Sarcopenia was determined as <27 kg for males and <16 kg for females for handgrip strength, <1.049 kg/BMI for males, and <0.823 kg/BMI for females for SMMI. Four-meter walking speed was noted during participants' usual gait speed (sec) over a 4-meter-long course. Patients with sarcopenia and BMI ≥ 30 kg/m^2 were considered sarcopenic obese. In EWGSOP 2, "Probable sarcopenia" was diagnosed based solely on low muscle strength. "Sarcopenia" was classified by low muscle mass and strength coexistence. "Severe sarcopenia" was characterized by slow gait speed, low muscle mass and low muscle strength.

Statistical Analysis

This study primarily conducted all analyses using R software and Turcosa for statistical computing and graphics. A p-value was considered statistically significant if it was less than 0.05. The normality of the data was evaluated using the Shapiro-Wilk test. Data with non-normal distributions were compared using the Mann-Whitney U test, and the findings were reported as the median (min-max). Data that are normally distributed were subjected to the student's t-tests. The findings were displayed as mean ± standard deviation. Pearson correlation analysis was employed to assess

relationships between data sets that were normally distributed. In contrast, Spearman correlation analysis was utilized to examine associations involving data sets that were not normally distributed.

In this study, we conducted a one-tailed power analysis to determine if the sarcopenic group's thickening fraction (TF) value was significantly lower compared to the non-sarcopenic group. A post hoc power analysis was performed using G*Power with the following parameters: an effect size (Cohen's d) of 1.077, an alpha error probability (α) of 0.05, and sample sizes of 18 for the non-sarcopenic group and 17 for the sarcopenic group. The calculated achieved power (1-β) was 0.930, indicating that the study had sufficient power to detect a significant difference between the groups.

Results

Seventeen of 35 patients (48.6%) were found to be sarcopenic. The non-sarcopenic group had a mean age of 64.61±6.8 years, while the sarcopenic group had a mean age of 70.94±12.2 years, with no significant difference observed (p = 0.217). The median duration of COPD and cigarette consumption, measured in pack-years, shows no significant difference between the sarcopenic and non-sarcopenic groups (p= 0.867 and 0.464, respectively). The sarcopenic group had a slightly higher median BMI of 30.48 than the non-sarcopenic group, with a median BMI of 25.64 (p =0.643) (Table 1). Of the 35 individuals, 65.7% had normal BMI (n=23), while 34.3% were obese (n=12). Among sarcopenic individuals, 35.3% had normal BMI (n=6), and 64.7% were obese (n=11) (Table 1).

Gait speed did not differ significantly between the groups (p = 0.956), with both groups recording a median speed of 0.800, albeit with different ranges. A notable distinction was observed in the fat percentage, where sarcopenic participants had a significantly higher mean fat percentage (31.23%, SD =3.41) compared to their non-sarcopenic counterparts (24.4%, SD = 7.35) (p < 0.001). SMMI adjusted for BMI was significantly lower in the sarcopenic group (0.92± 0.07) compared to the non-sarcopenic group (1.26± 0.224) (p <0.001) (Table 2). However, no significant differences were detected in SMM, impedance, waist-hip ratio, water percentage, dry lean weight, total lean mass, and total fat mass between the two groups.

Table 1 Comparative Overview of Demographic and Physiological Attributes Across Sarcopenic and Non-Sarcopenic Groups

	Non-Sarcopenic (n=18)	**Sarcopenic (n=17)**	**Total**	**p**
Age	64.61± 6.8	70.94±12.2	67.68±14.99	0.217*
BMI	25.64±3.60	30.48±3.31	28.41±5.15	0.643*
BMI Status				
Normal (BMI<30), n (%)	17 (73.91%)	6 (26.09%)	23 (100%)	
Obese (BMI≥30), n (%)	1 (8.3%)	11 (91.67%)	12 (100%)	**p<0.001**
Cigarette consumption (pack-years), mean (SD)	33.8 ± 21.1	27.7±19.5	30.6±20.1	0.464*
COPD duration (year) (Min-Max)	3.17 (1.38–4.96)	5.41 (2.5–8.32)	4 (0–19)	0.867#
mMRC score (Min-Max)	1.61(1,2)	1.64(1–3)	1(1–3)	0.257#
Smoking Status				
Non-smoker	0	1 (5.91%)	1 (2.85%)	0.578
Smoker	7 (38.89%)	6 (35.29%)	13 (37.15%)	
Ex-smoker	11 (61.11%)	10 (58.8%)	21(60%)	

Notes: Values are expressed as n (%), mean± SD or median (minimum-maximum). Significant p values are shown in bold. *Student's *t*-test, #Mann-Whitney U test, Pearson's chi-square test.
Abbreviations: BMI, Body mass index; COPD, Chronic Obstructive Pulmonary Disease; mMRC score, Modified Medical Research Council score.

Table 2 Measurement Values for the Diagnosis of Sarcopenia in Patients

	Non-sarcopenic	Sarcopenic	Total	p
Gait Speed	0.800 (0.280–3.90)	0.800 (0.540–2.00)	0.800 (0.280–3.90)	0.956[#]
Fat percentage	24.4 (7.35)	31.23 (3.41)	26.74 (7.22)	**<0.001***
SARCF score	2.11 (2.61)	1.59 (2.27)	1.857 (2.43)	0.532[#]
SMM	30.5 (2.67)	27.6 (4.16)	29.0 (3.75)	0.055*
SMMI (BMI)	1.26 (0.224)	0.92 (0.07)	1.096 (0.240)	**<0.001***
SMMI length^2	10.52 (0.92)	9.76 (1.60)	10.12 (1.35)	0.167*

Notes: Values are expressed as n (%), mean± SD or median (minimum-maximum). Significant p values are shown in bold. *Student's *t*-test, [#]Mann-Whitney U test, Pearson's chi-square test.
Abbreviations: SARCF, Strength, Assistance in walking, rising from a chair, climbing stairs, and falling; SMM, Skeletal Muscle Mass Index.

Both groups showed no significant differences in FEV1 (1.92 lt, 63.14%), FVC (3.15 lt, 85.54%), and FEV1/FVC ratio (57.81%), indicating comparable pulmonary function between groups (for all p>0.05). The standing-to-supine decrease in FVC (FVC change %) did not differ significantly between groups, with median values of 3.07 and 4.12, respectively (p = 0.806). However, the MEP and MIP showed no significant differences between groups (p>0.05) (Table 3).

Table 3 Comparative Overview of Respiratory Functions Tests and Ultrasonographic Values Across Sarcopenic and Non-Sarcopenic Groups

	Non-sarcopenic	Sarcopenic	Total	p
FEV1 (lt)	1.92 (1.24–4.25)	1.91 (0.79–3.20)	1.92 (0.79–4.25)	0.990
FEV1 (%)	62.83±18.17	63.47±17.15	63.14±17.59	0.917
FVC (lt)	3.32±0.96	2.96±1.05	3.15±1.00	0.303
FVC (%)	92±23.91	78.85±21.31	85.54 ±19.00	0.093
FEV1/FVC	56.81±11.78	58.88±8.92	57.81±10.39	0.562
FVC in supine position (lt)	3.06 (1.06–4.03)	2.92 (2.64–5.34)	2.97 (1.06–5.34)	0.608
Standing-to-supine decrease in FVC (%)	3.07 (1.00–6.00)	4.12 (2.00–12.00)	4.19 (1.00–12.00)	0.806
MIP (cmH2O)	72.76 (47.00–90.00)	70.60 (39.00–80.20)	71.64 (39.00–90.00)	0.854
MEP (cmH2O)	69.92 (20.67)	61.70(25.36)	65.69 (23.24)	0.302
FRC-Tdi (cm)	0.19±0.05	0.17±0.03	0.18±0.04	0.836
TV-Tdi (cm)	0.25±0.06	0.23±0.04	0.24±0.05	0.332
RV-Tdi (cm)	0.17±0.03	0.16±0.03	0.17±0.03	0.332
TLC-Tdi (cm)	0.33 (0.24–0.57)	0.30 (0.22–0.36)	0.31 (0.22–0.57)	0.294
Excursion (cm)	1.54 (0.83–2.80)	1.38 (0.97–2.43)	1.48 (0.83–2.80)	0.078
TF (%)	48.20 (23.63)	27.43 (13.61)	38.11 (21.84)	**0.003**
TLC-TF (%)	49.35 (13.68)	39.76 (9.97)	44.69 (12.81)	**0.023**

Notes: Values are expressed as n, mean±SD, or median (minimum-maximum). Significant p values are shown in bold.
Abbreviations: FEV1, Forced Expiratory Volume in one second; FVC, Forced Vital Capacity; MIP, maximal inspiratory pressure; MEP, maximal expiratory pressure; FRC-Tdi, The diaphragm thickness at the level of functional residual capacity; TV-Tdi, The diaphragm thickness at the level of tidal volume; RV-Tdi, The diaphragm thickness at the level of residual volume; TLC-Tdi, The diaphragm thickness at the level of total lung capacity; TF, thickening fraction; TLC-TF, The change in thickness was assessed from total lung capacity level to residual volume level.

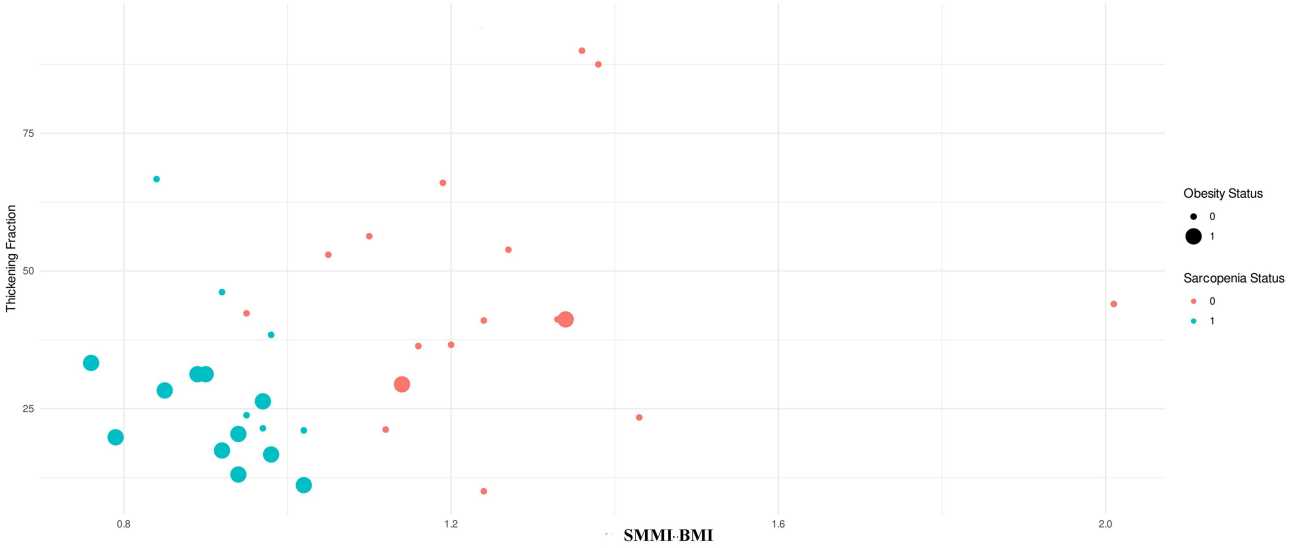

Figure 1 Scatter plot illustrating the relationship between skeletal muscle mass index (SMMI-BMI) and diaphragm thickening fraction in patients grouped by sarcopenia and obesity status. Sarcopenia status is represented by red (1, present) and cyan (0, absent) dots, while obesity status is indicated by the size of the dots (small for non-obese, large for obese). This visualization aids in assessing potential correlations between muscle metrics and diaphragm functionality across different patient groups.

There were no significant differences in diaphragm thickness between the two groups at the levels of FRC, TV, RV, and TLC ($p > 0.05$) (Table 3).

The sarcopenic group exhibited a notably lower TF of 27.43%, whereas the non-sarcopenic group showed a higher TF of 48.20% ($p = 0.003$). A positive, moderate, and statistically significant correlation was observed between TF and SMMI-BMI variables ($r=0.3972$, $p=0.045$) (Figure 1). Additionally, TLC-TF showed a significant difference between the groups ($p=0.023$).

Also, we found that diaphragmatic excursion value for the non-sarcopenic group was 1.54 cm, while it was 1.38 cm in the sarcopenic group. The group differences were insignificant ($p=0.078$) (Table 3).

Discussion

The present study reveals that diaphragm TF, as measured by ultrasound during both quiet and deep breathing, could be a valuable and auxiliary indicator of sarcopenia in COPD patients. Although the diaphragm excursion value was markedly reduced in the sarcopenic group, it was insufficient to distinguish between sarcopenic and non-sarcopenic groups. Our results also indicate a high prevalence of sarcopenia (48.6%) among COPD patients with moderate to severe airway obstruction, underscoring the need for early recognition and management. Notably, the study highlights the challenge of sarcopenic obesity in COPD, with a substantial number of sarcopenic patients also classified as obese, which exacerbates respiratory impairment. This underscores the importance of addressing not only cachexia in COPD but also "sarcopenic obesity", given its potential impact on prognosis and treatment strategies.

Sarcopenia, characterized by a reduction in muscle mass and function, is associated with poor nutritional status, increased inflammation, and other systemic effects that may exacerbate COPD. Sarcopenia in individuals with COPD has become an increasingly important topic in recent years, as it is associated with exacerbations and poor prognosis.

Middle-aged and elderly individuals are typically COPD patients, but it has been shown that muscle wasting in COPD occurs in all age groups regardless of age.[12] Early detection of sarcopenia raises the possibility of treatment approaches, such as pulmonary rehabilitation, that can have a positive impact on this patient population. A meta-analysis on COPD patients found the prevalence of sarcopenia to be 16.5%, though there was substantial heterogeneity across studies.[13] Previous reviews have reported sarcopenia prevalence rates of 19% in patients with GOLD stages 1–2, increasing to 37.6% in those with GOLD stages 3–4.[14] In our study, we excluded COPD patients with very severe obstruction (FEV1<30%) and those with mild obstruction (FEV1>80%), as they could skew sarcopenia prevalence. Among the COPD patients with FEV1 between 30% and 80%, we found a sarcopenia prevalence of 48.6%.

In line with our study objectives, we found that sarcopenia-related reductions in diaphragm measures, particularly in TF and TLC-TF, correlated with the sarcopenic condition. Diaphragm TF is an indirect measure of muscle fibre contraction, similar to the heart's ejection fraction, but it reflects a higher degree of active diaphragmatic contraction. In healthy individuals, TF values typically range between 30% and 36%. Among individuals with COPD, the lower limit of normal TF is above 20%.[15] Another study reported the mean TF value as 41.7% for COPD patients with severe and mild airway restrictions.[16] In our study, the TF value was 38.11% across the patient group, reflecting the respiratory effort associated with increased ventilatory neural drive in COPD.[17] In the only study examining sarcopenic individuals, the TF value was reported as 15%.[9] In our sarcopenic COPD group, we observed a TF value of 27.43%, reflecting a reduced but not critically low diaphragmatic effort compared to sarcopenic individuals in the literature. This variability in TF highlights the need for further research to better understand the factors affecting diaphragmatic function in this population. Additionally, the TLC-TF value was significantly lower in the sarcopenic group ($p=0.023$), suggesting impaired diaphragmatic performance. In COPD patients, elevated TF values may indicate a compensatory response, similar to the hyperkinetic phase of the heart driven by increased respiratory demand. Conversely, a decrease in TF in sarcopenia could represent a "failure phase", similar to reduced ejection fraction in heart failure, reflecting diminished diaphragmatic function.

In healthy individuals, the mean diaphragmatic excursion value of the population has been reported to range from 1.8 to 3 cm during tidal volume.[18,19] In COPD patients, diaphragmatic excursion has been shown to decrease, with further reductions as the disease progresses. While some studies demonstrate a relationship between diaphragmatic excursion and COPD exacerbations as well as FEV1,[20] other publications report no correlation with FEV1.[19] In our study, although diaphragmatic excursion was lower in sarcopenic individuals, the difference was not statistically significant. For reference, healthy individuals generally exhibit an excursion value of around 1.7 cm, whereas in our COPD cohort, the mean excursion was 1.48 cm. Although this reduction was not statistically significant, it may still hold clinical relevance and warrants further investigation into larger samples.

It has been reported that a diaphragm thickness of 1.50 mm indicates diaphragm weakness,[21] though variable diaphragm thicknesses have been observed in individuals with COPD.[22] Also, in sarcopenic individuals, diaphragm thickness has been reported as 1.8 mm.[23] There is insufficient data regarding both COPD and sarcopenic groups. In our study, detecting a 1.7 mm thickness may indicate diaphragmatic weakness in sarcopenic patients. Data on diaphragm thickness in COPD and sarcopenic groups remain limited, but in our study, a thickness of 1.7 mm may suggest diaphragmatic weakness in sarcopenic patients. However, literature often associates diaphragmatic dysfunction more strongly with parameters like TF and excursion rather than thickness alone, as these metrics may better reflect functional diaphragm impairment.

In our study, neither MIP nor MEP values differed between the groups. Although this might seem counterintuitive, the lack of correlation between TF and MIP/MEP in the sarcopenic group may be attributed to altered respiratory mechanics and hyperinflation, characteristic of COPD and could mask the expected reductions in these measures. Even though there was no difference between the groups, MEP was lower in the sarcopenic group than in the non-sarcopenic group, and the expected values for the healthy population were lower.[24] This observation of preserved MIP and partially reduced MEP in COPD patients may be due to the differential use of expiratory and inspiratory muscles, as expiratory muscles tend to be less engaged and may, therefore, be more vulnerable to sarcopenia. Although previous studies on sarcopenia have presented variable data, it is known that age, BMI, and gender influence MIP and MEP values. Studies report that MIP is more closely related to diaphragm thickness or excursion.[9,24] In our study, neither TF nor excursion showed a significant relationship with MIP and MEP ($p>0.05$ for all). This could be due to the limited number of patients in the study, as well as the influence of multiple factors. These findings align with existing literature indicating that expiratory muscles may be more susceptible to sarcopenia than inspiratory muscles. This could explain the slightly lower, yet statistically insignificant, MEP values observed in sarcopenic patients.

Sarcopenia in obese individuals has gained research interest in recent years, revealing a complex, paradoxical relationship between obesity and COPD. While cachexia has long been recognized as a marker of poor prognosis in COPD, recent findings suggest that obesity can also adversely impact COPD outcomes, including reduced performance in the six-minute walk test, lower quality of life, and increased exacerbation rates.[25,26] In our study, nearly all obese patients fell within the sarcopenia group, highlighting the need to address not only the traditional concept of "cachexia" in COPD but also the emerging issue of "obese COPD". This dual challenge underscores the importance of further investigating the interactions between obesity, sarcopenia, and COPD to optimize patient management strategies.

Several limitations should be considered when interpreting our results. This cross-sectional study involved a small sample size. It used bioelectrical impedance analysis (BIA) to measure skeletal muscle mass, which may overestimate values compared to Dual-Energy X-ray Absorptiometry (DXA).[27] CT and Magnetic Resonance Imaging (MRI) are the definitive methods for muscle mass assessment, but they are costly and involve radiation exposure. DXA, considered the gold standard for measuring body composition in both research and clinical settings, provides accurate differentiation of fat, bone mineral, and lean tissue with minimal radiation.[28] However, its lack of portability limits its use in large-scale epidemiological studies. Bioelectrical Impedance Analysis (BIA) offers a portable, cost-effective alternative feasible for ambulatory and bedridden patients. Although BIA's accuracy can vary, especially in estimating lean mass, studies under standardized conditions have shown strong correlations between BIA and MRI-derived muscle mass estimates.[29,30] Given its practicality, BIA is a reasonable alternative in studies where DXA is not accessible, though the limitations in precision should be acknowledged.

Additionally, we did not stratify patients by COPD stages or compare obese and non-obese groups within the same stage. The study did not evaluate sarcopenia's impact on quality of life, lung function decline, exacerbation frequency, or the need for long-term oxygen and NIV therapy. Lastly, musculoskeletal ultrasound was limited to the diaphragm, excluding other muscle groups, particularly the lower limbs, which could have provided a more comprehensive assessment of sarcopenia. These limitations inform future research directions in this field.

Conclusion

This study contributes to the growing evidence supporting ultrasound-derived diaphragm TF as a valuable tool for detecting sarcopenia in COPD patients, potentially aiding in developing targeted rehabilitation strategies. Future studies should evaluate these measures in larger samples to strengthen understanding the clinical relevance of diaphragm measures in sarcopenic COPD.

Disclosure

The authors report no conflicts of interest in this work.

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