

Correlation and Clinical Significance of COPD Quadriceps Contraction Index with Diaphragmatic Function

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Received: September 30, 2026; **Published:** October 09, 2026

Abstract

Objective: To analyze the correlation between quadriceps contraction index (Qci) and diaphragmatic function in patients with chronic obstructive pulmonary disease (COPD), and explore its clinical significance.

Methods: A total of 100 patients with stable COPD admitted to the Second Affiliated Hospital of Ningxia Medical University from October 2024 to December 2025 were enrolled. They were divided into the mild-to-moderate COPD group (48 cases) and severe-to-very severe COPD group (52 cases) according to pulmonary function grading criteria. Meanwhile, 50 healthy subjects undergoing physical examination during the same period were recruited as the healthy control group. General baseline data of the three groups were collected. The anterior thigh thickness in supine position, quadriceps thickness (Qthick), diaphragmatic thickness at end-inspiration (TDI) and end-expiration (TDE), and diaphragmatic excursion (DE) during quiet breathing were measured. The thickening fraction of the diaphragm (TFDI) and Qci were calculated. The differences in indicators among groups were compared, and the correlations of Qthick, Qci with TD, DE and TFDI were analyzed.

Results: Qthick, Qci, DE, TDE, TDI and TFDI in the mild-to-moderate COPD group and severe-to-very severe COPD group were significantly lower than those in the healthy control group ($P < 0.05$). Qci in the severe-to-very severe COPD group was lower than that in the mild-to-moderate COPD group ($P < 0.05$). Pearson correlation analysis showed that Qci was positively correlated with TDI, TDE, quiet breathing DE and TFDI, with statistically significant differences ($P < 0.05$). Qthick was only positively correlated with TFDI ($P < 0.05$).

Conclusion: Qci is correlated with diaphragmatic function in patients with COPD. B-ultrasound measurement of quadriceps function can indirectly assess diaphragmatic function, providing a simple and feasible examination method for the evaluation of respiratory function in patients with chronic respiratory diseases.

Keywords: COPD; Quadriceps Contraction Index; Diaphragm Function

Introduction

Chronic obstructive pulmonary disease (COPD) is a heterogeneous lung disease characterized by chronic respiratory symptoms resulting from abnormalities of the airways and/or alveoli, accompanied by persistent progressive airflow limitation. It carries high morbidity, disability and mortality rates [1,2], seriously endangering public health and imposing a heavy economic burden. COPD represents a major global public health challenge that urgently requires solutions. According to the Global Initiative for Chronic Obstructive Lung Disease (GOLD) reports, COPD has become one of the three leading causes of death worldwide [2]. The prevention and control of COPD in China faces an especially severe situation. The latest national epidemiological survey demonstrates that the prevalence of COPD among Chinese adults aged ≥ 40 years reaches 13.7% [3], corresponding to approximately 43 million patients. COPD has been incorporated into the National Basic Public Health Service Program by the National Health Commission of China [4] and listed as one of the four major chronic diseases prioritized for national prevention and management. Notably, our previous team study found that the COPD prevalence among residents aged ≥ 40 years in Yinchuan was 15.9% [5], markedly higher than the national average. The grim epidemiological status and regional high-incidence feature highlight the urgent need and necessity for precise assessment, early intervention and standardized prevention and treatment of COPD. Skeletal muscle dysfunction, especially diaphragm and quadriceps dysfunction, is a recognized extrapulmonary complication of COPD [6]. It has become a core link that directly determines the patient's quality of life and prognosis. Its severity worsens as COPD disease progresses, directly causing the patient's reduced mobility endurance, increased risk of motor imbalance and falls, aggravated respiratory difficulties, and ultimately worsening condition, accelerating patient death. Although its clinical harm is clear, in conventional clinical practice, quantitative evaluation, early identification, and standardized monitoring of skeletal muscle function have not been given sufficient attention, becoming a prominent shortcoming in COPD's whole-pathology management. Currently, no relevant domestic study has clarified the correlation between peripheral muscles and respiratory muscles in patients with COPD.

Aim of the Study

This study focuses on evaluating the condition of impairment of quadriceps and diaphragm function in patients with COPD, and exploring the correlation between them, assessing their potential clinical value.

Materials and Methods

Study subjects and grouping

A total of 100 patients with stable COPD who attended the Outpatient Department of Respiratory and Critical Care Medicine, the Second Affiliated Hospital of Ningxia Medical University from October 2024 to December 2025 were enrolled. According to the pulmonary function grading criteria in the 2022 GOLD guideline [7], the patients were divided into mild-moderate COPD group and severe-very severe COPD group. The mild-moderate group was defined as $FEV_1/FVC < 70\%$ and $FEV_1\%pred \geq 50\%$, while the severe-very severe group was defined as $FEV_1/FVC < 70\%$ and $FEV_1\%pred < 50\%$. Fifty healthy physical examinees with matched general information during the same period in our hospital were recruited as the healthy control group. All subjects signed informed consent forms, and this study was approved by the Ethics Committee of the Second Affiliated Hospital of Ningxia Medical University (Approval No.: KY-2024-016).

Inclusion and exclusion criteria

Inclusion criteria

Stable COPD: ① According to the 2022 Global Strategy for the Diagnosis, Management, and Prevention of Chronic Obstructive Pulmonary Disease [7], airflow limitation is defined as $FEV_1/FVC < 70\%$ after inhalation of bronchodilators. ② No psychiatric or psychological disorders, and the patients can actively cooperate to complete measurements of quadriceps function, diaphragmatic function and other indicators.

Exclusion criteria

Patients with other diseases that may affect pulmonary function, including but not limited to pulmonary infection, bronchial asthma, bronchiectasis, pulmonary malignant tumors, pulmonary tuberculosis, interstitial pulmonary fibrosis, etc.; complicated with severe cardiac, hepatic or renal diseases; complicated with malignant tumors and central nervous system diseases; complicated with chronic metabolic diseases and rheumatic immune diseases; patients with previous thoracic or knee joint surgery; patients with known myopathy or diseases causing diaphragmatic dysfunction; patients who recently received treatment with immunosuppressants, non-steroidal anti-inflammatory drugs or systemic corticosteroids.

Methods

Laboratory instruments and equipment

Weighing scale, measuring ruler, portable color doppler ultrasound (Model: PA12A).

Collecting general information

Basic information of all subjects was collected, including name, gender, age, height and body weight. Body Mass Index (BMI) was calculated by the formula: $BMI = \text{weight} / \text{height}^2$ (kg/m^2).

Measurement of diaphragm indicators

All subjects were placed in the supine position with the back inclined at an average angle of 45° . A convex array probe of B-mode ultrasound was placed at the junction of the right midclavicular line and the costal margin. The sampling line was kept perpendicular to the diaphragmatic dome to visualize the curved image of the diaphragm. Once the acoustic window was clearly displayed and the image stabilized, the mode was switched to M-mode ultrasound. After the curved trace of diaphragmatic motion with respiration was visualized, the vertical distance between the peak and trough of the curve was directly measured. Diaphragmatic excursion (DE) was acquired by maintaining 3-5 quiet spontaneous respiratory cycles under M-mode ultrasound and calculating the average value of three consecutive similar waves. A linear array probe of B-mode ultrasound was placed at the 9th or 10th intercostal space on the anterior axillary line, visualizing three white lines: the pleural line, peritoneal line, and the tendinous portion of the diaphragm. The distance between the pleural line and peritoneal line represents diaphragmatic thickness. Under M-mode, the sampling line was maintained perpendicular to the diaphragm, and measurements from three quiet spontaneous respiratory cycles were recorded for averaging. Diaphragmatic thickness at end-expiration (TDE) and end-inspiration (TDI) were measured respectively. The Diaphragm Thickening Fraction (TFDI%) was calculated using the formula: $TFDI\% = (\text{diaphragmatic thickness at end-inspiration} - \text{diaphragmatic thickness at end-expiration}) / \text{diaphragmatic thickness at end-expiration}$.

Measurement of quadriceps indices in stocks

All subjects were placed in the supine position. The dominant lower limb was fully relaxed and naturally extended, with the great toe at 10° - 15° relative to the midline. All measurements were performed using a 4-12 MHz high-frequency linear array probe. Under B-mode ultrasound in musculoskeletal preset at a depth of 6 cm, the probe was placed perpendicularly at two-thirds of the distance between the anterior superior iliac spine and the superior pole of the patella along the longitudinal axis of the thigh. A transverse image of the anterior thigh of the dominant leg was acquired to measure the total anterior thigh thickness (the distance from the skin to the anterior border of the femur) and quadriceps thickness (the sum of the distance from the medial border of rectus femoris and the myofascial border of vastus intermedius) [8]. Quadriceps thickness (Qthick) was defined as the sum of the measured values of rectus femoris and vastus intermedius. The Quadriceps Contraction Index (Qci%) was calculated by the formula: $Qci\% = \text{quadriceps thickness} / \text{total anterior thigh thickness}$. The normal reference range of Qci was $\geq 60\%$. To assess reliability, the same operator performed repeated measurements on frozen images after an interval of 3 weeks, and the average value was taken.

Statistical analysis

SPSS 23.0 software was used for data processing. Measurement data were expressed as mean ± standard deviation and analyzed by t-test; enumeration data were analyzed by χ^2 test. For comparison between two groups, independent-samples t-test was adopted if the data followed a normal distribution. For comparison among three groups, one-way analysis of variance (ANOVA) was used when normality and homogeneity of variance assumptions were satisfied, and LSD test was applied for pairwise comparisons between groups. When the above assumptions were not met, the nonparametric Kruskal-Wallis H test was performed. Pearson correlation analysis was used for correlation analysis. A value of $P < 0.05$ was considered statistically significant.

Results

General data comparison

The differences in sex, age, height, and weight between the three groups were statistically insignificant ($P > 0.05$), while the differences in BMI between the three groups were at a critical level ($P = 0.05$), as shown in table 1.

Group	Healthy control group	Mild-moderate COPD group	Severe-very severe COPD group	F/ χ^2	P value
Gender (Male:Female)	33:17	34:14	30:22	1.945	0.378
Age (years)	68.30 ± 6.85	67.98 ± 6.57	66.63 ± 7.83	0.023	0.988
Height (m)	1.67 ± 0.08	1.69 ± 0.06	1.66 ± 0.07	2.261	0.108
Weight (kg)	66.85 ± 7.80	65.23 ± 10.71	64.19 ± 11.45	0.89	0.413
BMI (kg/m ²)	24.10 ± 2.67	22.73 ± 2.97	23.10 ± 2.93	3.052	0.05

Table 1: Comparison of general baseline data between groups.

Notes: Data are presented as mean ± SD.

Comparison of diaphragm function-related indicators (TD, DE, TFDI) among the healthy control group, mild-moderate COPD group and severe-very severe COPD group

Compared with the healthy control group, TDE, TDI, quiet-breathing DE and TFDI were significantly decreased in both the mild-moderate COPD group and severe-very severe COPD group, with statistically significant differences (all $P < 0.05$). However, there were no statistically significant differences in the above indicators between the mild-moderate COPD group and severe-very severe COPD group, as shown in table 2 and figure 1.

Group	Healthy control group	Mild-moderate COPD group	Severe-very severe COPD group	F/H	P value
TDE (mm)	3.40 ± 0.78	2.09 ± 0.50*	2.07 ± 0.60*	74.287	<0.001
TDI (mm)	4.91 ± 0.98	2.73 ± 0.68*	2.53 ± 0.75*	91.028	<0.001
DE at quiet respiration (cm)	2.09 ± 0.78	1.52 ± 0.59*	1.36 ± 0.59*	28.548	<0.001
TFDI	0.46 ± 0.12	0.32 ± 0.20*	0.23 ± 0.15*	51.774	<0.001

Table 2: Comparison of diaphragm function-related indicators among groups.

Notes: Data are presented as mean ± SD. * $P < 0.05$, compared with the healthy control group; # $P < 0.05$, compared with the mild-moderate COPD group.

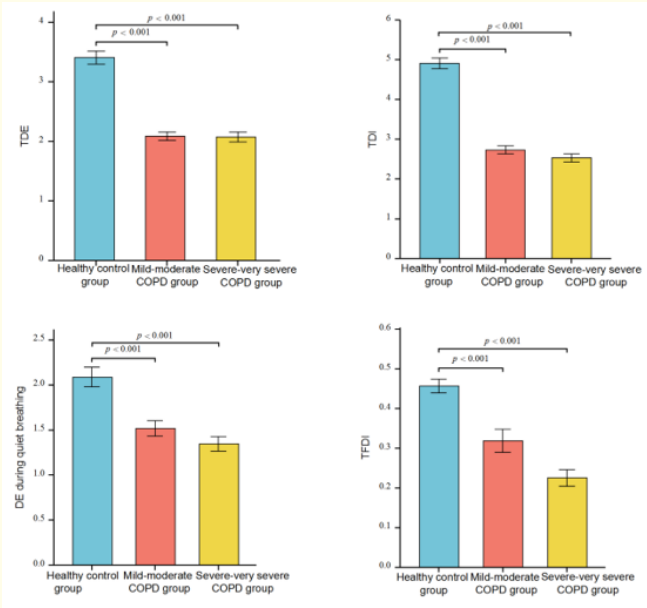


Figure 1: Comparison of TDE, TDI, DE and TFDI among groups.

Comparison of quadriceps muscle function-related indicators (Qthick, Qci) among the healthy control group, mild-moderate COPD group and severe-very severe COPD group

Compared with the healthy control group, Qthick and Qci were significantly decreased in both the mild-moderate COPD group and severe-very severe COPD group (all $P < 0.05$). Compared with the mild-moderate COPD group, Qci was significantly lower in the severe-very severe COPD group ($P < 0.05$), whereas Qthick was similar between the two groups with no statistically significant difference ($P > 0.05$), as shown in table 3 and figure 2.

Group	Healthy control group	Mild-moderate COPD group	Severe-very severe COPD group	F/H	P value
Anterior total thigh thickness (cm)	3.57 ± 0.64	3.30 ± 0.57	3.51 ± 0.90	3.124	0.21
Qthick (cm)	2.70 ± 0.58	2.30 ± 0.49*	2.19 ± 0.59*	11.541	0.001
Qci	0.75 ± 0.05	0.69 ± 0.53*	0.63 ± 0.07*#	59.889	0.001

Table 3: Comparison of quadriceps functional indicators among groups.

Notes: Data are presented as mean ± SD. * $P < 0.05$, compared with the healthy control group; # $P < 0.05$, compared with the mild-moderate COPD group.

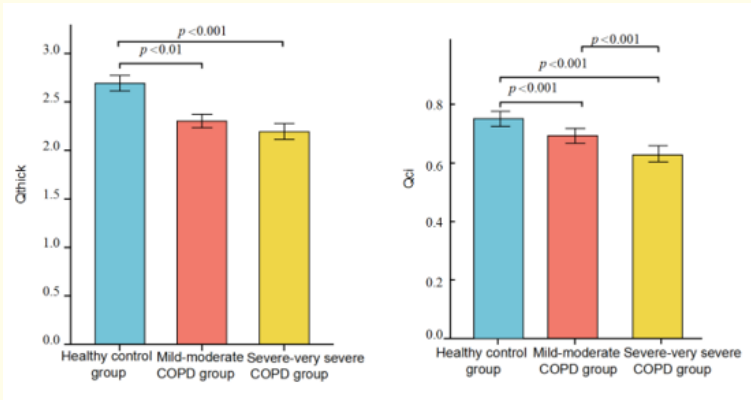


Figure 2: Comparison of Qthick and Qci among groups.

Correlation analysis of Qthick and Qci with TD, DE and TFDI indicators

Qthick was positively correlated only with TFDI ($P < 0.05$, $r = 0.253$), and showed no correlation with TDI, TDE and DE during quiet breathing (all $P > 0.05$). Qci was significantly positively correlated with TDI, TDE, DE during quiet breathing and TFDI (all $P < 0.05$), with correlation coefficients of 0.442, 0.363, 0.358 and 0.451, respectively (See table 4 and figure 3).

Group	TDI		TDE		DE during quiet breathing		TFDI	
	r value	P value	r value	P value	r value	P value	r value	P value
Qthick	0.086	0.295	0.070	0.393	0.084	0.309	0.253	0.002
Qci	0.442	0.001	0.363	0.001	0.358	0.001	0.451	0.001

Table 4: Correlation analysis between quadriceps indicators and diaphragmatic indicators.

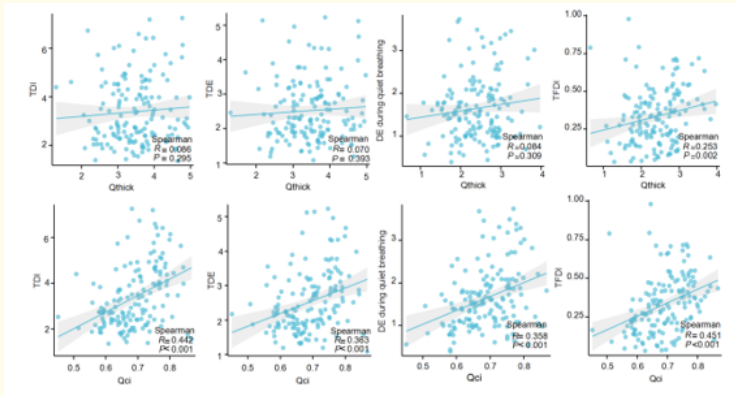


Figure 3: Correlation coefficient plot of Qthick and Qci with TDI, TDE, DE and TFDI.

Discussion

COPD is a systemic inflammatory syndrome. While damaging the airways and lung parenchyma, it can involve multiple organ systems throughout the body, among which skeletal muscle dysfunction represents an important extrapulmonary manifestation [9]. As a key comorbidity of COPD, skeletal muscle dysfunction is jointly driven by systemic inflammation, oxidative stress and mitochondrial dysfunction. It is characterized by muscle atrophy, reduced muscle strength and premature fatigue, and serves as an independent risk factor for deteriorated quality of life, increased hospitalization rate and elevated mortality risk in patients [10]. The diaphragm and quadriceps, as representatives of respiratory muscles and peripheral skeletal muscles respectively, develop characteristic muscle injury during the progression of COPD, which is a major contributor to dyspnea and decreased exercise tolerance in patients.

In this study, 50 subjects were enrolled in the healthy control group, 48 in the mild-moderate COPD group, and 52 in the severe-very severe COPD group. There were no statistically significant differences in gender composition, age, height and weight among the three groups. The baseline characteristics were balanced and comparable. The intergroup difference in BMI was at the borderline level ($P = 0.05$). Previous studies have generally reported lower BMI in COPD patients compared with healthy individuals [11]. The lack of significant difference in the present study may be limited by the relatively small sample size.

This study demonstrated that the mild-moderate COPD group already exhibited thinner TD, decreased DE and reduced TFDI, which was consistent with the findings reported by Kathleen Möller, Laxman Thakur, *et al.* [12,13]. This indicates that diaphragmatic dysfunction is not a terminal complication of COPD but an extrapulmonary injury that occurs in the early stage of the disease. Its core mechanism is related to diaphragmatic morphological changes and increased persistent mechanical load induced by pulmonary hyperinflation in COPD, as well as muscle atrophy and muscle fiber remodeling triggered by systemic inflammation and oxidative stress [14,15]. Long-standing pulmonary hyperinflation in COPD patients causes thoracic deformity and caudal displacement of the diaphragm. The normal domed shape of the diaphragm becomes shortened and flattened, which reduces the initial length of muscle fibers and markedly decreases contractile force under equivalent stimulation [14]. Such biological defects of the diaphragm caused by morphological alterations constitute the major adverse factor contributing to diaphragmatic dysfunction. Meanwhile, the diaphragm continuously counteracts elevated airway resistance, imposing chronic high workload on respiratory muscles and further inducing muscle fiber remodeling and atrophy. In response to these changes, the diaphragm undergoes adaptive structural transformation from type II (fast-twitch) muscle fibers to type I (slow-twitch) muscle fibers [16]. Compared with type II fibers, type I muscle fibers possess a richer peripheral vascular network, higher myoglobin content and more mitochondria. Increased capillary density, myoglobin level and mitochondrial density improve oxygen delivery efficiency of the diaphragm, thereby compensatorily enhancing diaphragmatic endurance and anti-fatigue capacity [15]. As a core indicator reflecting diaphragmatic contractile function, TFDI is impaired because of inherent muscle fiber atrophy and compromised contractile efficiency, compounded by thoracic mechanical restriction secondary to pulmonary hyperinflation. This impairs the capacity of the diaphragm for further thickening during inspiration. The above mechanisms explain why significant abnormalities in diaphragmatic morphology and function can emerge at the mild-moderate COPD stage.

From the perspective of molecular pathological mechanisms, multiple systemic inflammatory pathways are involved in the progression of COPD. A variety of inflammatory factors, including tumor necrosis factor- α (TNF- α), interleukin-6 (IL-6), nuclear factor- κ B (NF- κ B), are released into the bloodstream. On the one hand, they activate the ubiquitin-proteasome system to accelerate hydrolysis of contractile proteins and inhibit the proliferation and repair of satellite cells [17,18]. On the other hand, they disrupt mitochondrial structure and sarcoplasmic reticulum calcium transport homeostasis, induce muscular and mitochondrial dysfunction [19,20], and accelerate the degradation of contractile proteins such as actin and myosin. Ultimately, these changes drive structural remodeling of diaphragmatic fiber types, manifested as abnormal muscle fibers, reduced cross-sectional area of muscle fibers, decreased sarcomere number and diminished muscle mass, leading to impaired contractile efficiency and compliance of the diaphragm.

Notably, there was no statistically significant difference in diaphragmatic ultrasound indicators between the mild-moderate COPD group and the severe-very severe COPD group. This finding suggests that diaphragmatic injury does not decrease linearly with the deterioration of pulmonary function, and diaphragmatic impairment reaches a relatively prominent level in the early course of COPD. This phenomenon can also be explained from the perspective of disease mechanisms: sustained systemic inflammation in the early stage can initiate diaphragmatic remodeling. After entering the severe-very severe stage, confounding factors such as inter-individual differences in respiratory compensatory capacity and heterogeneous distribution of emphysema may mask the true intergroup differences in diaphragmatic ultrasound indicators across COPD severity grades [20,21]. These results indicate that ultrasound can serve as a bedside noninvasive tool for identifying diaphragmatic dysfunction in COPD. Diaphragmatic protection and respiratory muscle rehabilitation interventions should not be limited to end-stage patients but should be advanced to the early stage of COPD. This finding provides direct evidence supporting early comprehensive evaluation and early rehabilitation interventions for patients with COPD.

Furthermore, this study also compared the anterior thigh thickness among the three groups, and no statistically significant difference was observed. This parameter includes subcutaneous fat and muscle tissue and is confounded by interindividual variations in fat thickness; therefore, it cannot reflect COPD-related skeletal muscle wasting. The present study found that Qthick was significantly lower in the mild-moderate and severe-very severe COPD groups than in the healthy control group, indicating that quadriceps muscle mass loss also occurs in the early stage of COPD. This conclusion is consistent with a European study [22]. Mechanistically, persistent chronic inflammation and long-term hypoxia in COPD synchronously alter the internal and external microenvironment of muscle. On the one hand, they trigger mitochondrial dysfunction and loss of contractile proteins; on the other hand, they induce hypoxia and hypercapnia. The combined effects promote the transformation of quadriceps muscle fibers from type I oxidative fibers to type II glycolytic fibers. This process is accompanied by decreased oxidative capacity, microvascular regression and muscle fiber atrophy, collectively resulting in reduced muscle endurance and increased susceptibility to fatigue [23]. In the late stage of COPD, superimposed risk factors such as senescence, deteriorated nutritional status and systemic corticosteroid therapy further aggravate muscle fiber atrophy and continuously worsen peripheral skeletal muscle injury. Meanwhile, chronic inflammation inhibits the differentiation of satellite cells and promotes their transformation into adipocytes [24]. Hypoxia, oxidative stress and other factors disrupt fat metabolism [25], and fat preferentially accumulates in regions with muscle atrophy and expanded intermuscular spaces, ultimately leading to fatty infiltration of the quadriceps femoris. Nevertheless, there was no significant difference in Qthick between the mild-moderate group and the severe-very severe group, suggesting that this indicator can only identify the reduction in muscle thickness and fails to reflect intrinsic muscular lesions during disease progression. Subsequent detection and research combining indicators such as muscle echo intensity and shear wave elastography can be performed to further verify the above conclusions.

In contrast, Qci decreased progressively with increasing COPD severity, and pairwise comparisons among the three groups all showed statistically significant differences. These results indicate that simple measurement of anterior thigh thickness cannot identify peripheral skeletal muscle injury in COPD. In the early stage, there may be no obvious change in the apparent thickness of muscle, while the muscle parenchyma has already been damaged. This suggests that structural injury and functional impairment of peripheral skeletal muscle do not occur synchronously, and simple thickness parameters cannot reflect intrinsic muscle function. Qthick, with fat interference excluded, can only detect early muscle mass loss, whereas the functional indicator Qci is more sensitive to reflect progressive peripheral skeletal muscle dysfunction mediated by disease. Since Qci integrates muscle thickness and echo features of muscle parenchyma, it is capable of detecting subtle internal lesions that cannot be captured by muscle thickness parameters alone. Therefore, Qci can serve as a more sensitive ultrasonic indicator for dynamically monitoring skeletal muscle lesions in COPD and reflecting the dynamic deterioration of peripheral skeletal muscle function with disease progression. The results of this study showed that Qci was moderately positively correlated with TDI, TDE, DE during quiet breathing and TFDI; while Qthick was only weakly positively correlated with TFDI and exhibited no significant correlation with other diaphragmatic indicators. These findings not only suggest the characteristic of synergistic injury of respiratory

muscles and peripheral skeletal muscles in patients with COPD, but further verify the sensitivity of Qci. With the progression of COPD, multiple mechanisms including systemic inflammation, oxidative stress, chronic hypoxia and malnutrition can simultaneously affect both the diaphragm and quadriceps femoris, leading to concurrent muscle fiber atrophy and impaired contractile function in the two muscles. The study by Maynard-Paquette., *et al.* [26] demonstrated a significant correlation between Qci and TFDI, which is consistent with our results. Compared with Qthick, Qci showed stronger and more significant correlations with diaphragmatic indicators. The presumed underlying reason is that Qthick is susceptible to confounding factors such as subcutaneous fat thickness, BMI and individual body habitus, whereas Qci reflects quadriceps contractile function, can more truly reflect the proportion of muscle in soft tissues of the thigh, represents the actual work capacity of skeletal muscle, and is more suitable for evaluating sarcopenia in COPD patients. The above results indicate that diaphragmatic dysfunction is accompanied by reduced contractile function of peripheral skeletal muscle in COPD patients, and Qci has a better correlation with diaphragmatic indicators than Qthick. Therefore, this study suggests that clinicians can indirectly assess respiratory muscle function by measuring peripheral skeletal muscle function. Meanwhile, both respiratory and peripheral skeletal muscles should be jointly regarded as targets for respiratory rehabilitation interventions.

Conclusion

This is a cross-sectional study. Limited by its single-center sample and confounding factors, it has certain limitations, and further verification through longitudinal cohort studies is warranted. Ultrasonographic assessment of quadriceps and diaphragmatic function in COPD patients is rapid, convenient, easy to perform and reliable. By comparing diaphragmatic and quadriceps functional indicators between the healthy group and COPD groups, this study identified varying degrees of diaphragmatic and quadriceps dysfunction in COPD patients, and Qci was positively correlated with diaphragmatic functional parameters. Clinically, ultrasound measurement of quadriceps function can be used to indirectly evaluate diaphragmatic function in COPD patients. This assessment strategy can circumvent the inconvenience of direct diaphragmatic examination and provide new references for precise clinical evaluation of disease status, early identification of extrapulmonary complications, dynamic monitoring of respiratory muscle function, and guidance of individualized treatment. It carries important clinical implications for improving patients' respiratory function, slowing disease progression and preventing respiratory failure.

Funding Support

This study was supported by the following funding: Yinchuan Science and Technology Program: Correlation of Quadriceps Function Assessed by Ultrasound with Disease Severity and Diaphragmatic Contractility in Patients with COPD (No. 2024SFZD001); Science and Technology Innovation Team for Respiratory Disease Rehabilitation and Standardized Management of Yinchuan (No. 2023CXTD05); Key Scientific and Technological Achievement Transformation Project of Ningxia Hui Autonomous Region: Transformation and Application of Intellectually Controlled Respiratory Support Technology in Capacity Improvement of Respiratory Support Therapy for Critically Ill Patients (No.2024CJE09048); 2021 Ningxia Hui Autonomous Region Science and Technology Innovation Leading Talent Project (No.2021GKLRLX03).

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Volume 15 Issue 10 October 2026

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