Original Article

The Relationship Between Diaphragmatic Thickness and Pulmonary Function In Patients with Chronic Obstructive Pulmonary Disease: A Cross-Sectional Study

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Abstract

Background: Diaphragm dysfunction plays an important role in the pathophysiology of chronic obstructive pulmonary disease (COPD) and may contribute to respiratory symptoms and functional limitation. Ultrasonographic assessment of diaphragm thickness has emerged as a non-invasive method to evaluate respiratory muscle involvement, but its relationship with pulmonary function remains unclear. This study aimed to assess diaphragm thickness and its correlation with pulmonary function in patients with COPD.

Method: A cross-sectional study was conducted involving patients with COPD. Demographic and clinical characteristics, including symptom severity based on the modified Medical Research Council (mMRC) scale and GOLD classification, were recorded. Diaphragm thickness was measured using ultrasonography. Pulmonary function was assessed using spirometry, including FEV_1/FVC, percent predicted FEV_1, and percent predicted FVC. Correlation analyses were performed to evaluate the relationship between diaphragm thickness and pulmonary function parameters.

Results: The study population consisted predominantly of older male patients with moderate to very severe airflow limitation. The mean diaphragm thickness was 0.30±0.08. Diaphragm thickness showed a weak negative correlation with FEV_1/FVC ($r = -0.184$; $P=0.201$), weak positive correlations with percent predicted FEV1 ($r = 0.188$; $P=0.190$), and percent predicted FVC ($r = 0.168$; $P=0.243$). None of these correlations were statistically significant.

Conclusion: Diaphragm thickness was not significantly associated with spirometric parameters in patients with COPD. These findings suggest that static diaphragm thickness alone may not adequately reflect impairment in pulmonary function, necessitating a multidimensional assessment of respiratory muscle involvement.

Keywords: COPD, diaphragm thickness, pulmonary function, spirometry, ultrasonography

INTRODUCTION

Chronic obstructive pulmonary disease (COPD) is characterized by persistent and progressive airflow limitation that is associated with an exaggerated chronic inflammatory response in the airways and lung parenchyma.[11] A significant cause of morbidity and mortality, COPD is a global public health concern. An estimated 3.2 million people worldwide lost their lives to COPD in 2015, and in 2030 it is expected to rank as the fourth most common cause of death.[2]

Beyond its pulmonary manifestations, COPD is increasingly recognized as a systemic disease with significant extrapulmonary consequences, reflecting the presence of chronic systemic inflammation, which arises through several mechanisms. One widely accepted hypothesis suggests that inflammatory processes originating in the airways and lung parenchyma may "spill over" into the systemic circulation, thereby inducing inflammatory responses in distant tissues.[3]

Another mechanism implicates cigarette smoking and smoke exposure as direct contributors to systemic inflammation, given their established role in triggering inflammatory pathways and promoting endothelial dysfunction. Even passive smoking has been associated with elevated oxidative stress markers and vascular endothelial impairment.[3]

In addition, hypoxemia and pulmonary hyperinflation, which commonly occur in COPD, have been shown to exacerbate systemic inflammation. One of the most clinically relevant consequences of systemic inflammation in COPD is skeletal muscle dysfunction. Muscle impairment affects both respiratory muscles within the thoracic cavity and peripheral muscles, particularly those of the lower extremities. Interestingly, inflammatory involvement of respiratory muscles appears to be less pronounced than that observed in peripheral muscles, as evidenced by lower levels of pro-inflammatory cytokines in respiratory muscle biopsy specimens.[4–6]

Among respiratory muscles, the diaphragm has received particular attention due to its central role as the primary generator of tidal volume. Dyspnea and exercise intolerance are hallmark symptoms of COPD and are closely associated with an imbalance between respiratory muscle load and capacity.[6,7]

Chronic airflow limitation and pulmonary hyperinflation increase the mechanical burden on the diaphragm, resulting in diaphragmatic flattening, altered chest wall geometry, and reduced capacity to generate contractile force. These mechanical disadvantages contribute to diaphragmatic dysfunction, which has been increasingly recognized as a key factor in the pathophysiology of COPD-related respiratory impairment.[6,7]

Several studies have demonstrated that diaphragmatic mobility is significantly reduced in patients with COPD compared with healthy individuals and that this reduction is closely associated with air trapping and pulmonary hyperinflation.

Ultrasonographic studies have further shown that decreased diaphragmatic mobility correlates with reduced exercise capacity and increased dyspnea following physical activity, such as during the six-minute walk test.[7,8]

Additionally, diaphragmatic mobility has been linked to airflow obstruction, lung hyperinflation, ventilatory capacity, and subjective perception of dyspnea. However, some studies have failed to demonstrate a direct relationship between reduced diaphragmatic mobility and maximal inspiratory pressure, suggesting that diaphragmatic weakness may occur independently of overall inspiratory muscle strength due to compensatory activation of accessory muscles.[7,8]

Furthermore, limitations in earlier studies—including the lack of concurrent assessment of lung volumes, symptom severity, and exercise capacity—have hindered comprehensive interpretation of diaphragmatic dysfunction. The use of chest radiography in assessing diaphragmatic mobility also poses practical limitations, including radiation exposure and dependence on patient cooperation.[7,8]

Recently, thoracic ultrasonography has emerged as a valuable, non-invasive tool for assessing diaphragmatic structure and function. Measurement of diaphragmatic thickness and thickening fraction using ultrasonography offers several advantages, including safety, feasibility, repeatability, and reproducibility. Ultrasonographic assessment allows for detailed evaluation of diaphragmatic morphology and function using B-mode and M-mode imaging, enabling accurate visualization of diaphragmatic motion and thickness at specific phases of the respiratory cycle.[7,9,10]

Given the growing evidence of an association between diaphragmatic dysfunction and pulmonary mechanics, assessment of diaphragmatic thickness at end-expiration represents a practical and clinically applicable parameter in patients with COPD. Previous studies have reported variability in diaphragmatic thickness among COPD patients, with some demonstrating associations between reduced diaphragmatic thickness and impaired pulmonary function parameters such as forced expiratory volume in one second (FEV_1) and forced vital capacity (FVC).[11–13]

However, findings remain inconsistent, particularly across different degrees of disease severity. Therefore, further investigation is warranted to elucidate the relationship between diaphragmatic thickness and pulmonary function parameters in patients with COPD across a broad spectrum of disease severity.

METHOD

This study was an analytical investigation employing a cross-sectional design, conducted over 10 months, from March to December 2020, at the Pulmonary Clinic of Prof. dr. Chairuddin Panusunan Lubis Hospital, Universitas Sumatera Utara, Medan, and was approved by the Health Research Ethics Committee of Universitas

Sumatera Utara with ethical clearance number No. 480/KEP/USU/2021.

The study population consisted of all patients with chronic obstructive pulmonary disease who had been diagnosed based on clinical symptoms and confirmed by spirometric examination. The study sample comprised patients who met the inclusion criteria and exclusion criteria.

Inclusion criteria for this study were stable COPD patients receiving treatment at Universitas Sumatera Utara Hospital, aged 40 years or older, who had been diagnosed with COPD by a pulmonologist based on clinical symptoms and confirmed by spirometry. Exclusion criteria included COPD patients with concomitant lung cancer or interstitial lung disease or those experiencing acute exacerbations of COPD or acute respiratory failure.

The required sample size was calculated using the appropriate formula, where "p" represented the proportion of COPD patients experiencing reduced diaphragmatic thickness or diaphragmatic muscle atrophy. Based on previous studies, the proportion of diaphragmatic muscle atrophy was reported to be 28.5% and was rounded to 30% for sample size calculation. Using a Z_α value of 1.96 corresponding to a 95% confidence level and a Z_β value of 0.84 corresponding to a statistical power of 80%, the minimum sample size required for this study was determined to be 50 participants. Participants were recruited using a consecutive sampling technique.

Eligible participants underwent ultrasonographic assessment of diaphragmatic thickness. Ultrasonography was performed using B-mode imaging. The diaphragm was visualized by placing the transducer in the ninth intercostal space, perpendicular to the ninth and tenth ribs, between the anterior and midaxillary lines, where the zone of apposition could be optimally observed approximately 0.5 to 2 cm below the costophrenic sinus.

The diaphragmatic muscle appeared as a hypoechoic structure located between two parallel hyperechoic lines representing the diaphragmatic pleura and the peritoneal membrane. Diaphragmatic thickness was measured by placing the calipers within the hyperechoic boundaries. Participants were instructed to breathe spontaneously, and three measurements of right diaphragmatic thickness were obtained at end-expiration and averaged. The mean value was recorded as the diaphragmatic thickness.

Subsequently, all participants underwent spirometric examination to obtain pulmonary function parameters. The examination was performed with the participant in an appropriate upright posture. A nose clip and mouthpiece were applied, and the lips were sealed firmly around the mouthpiece. Participants were instructed to breathe normally, followed by a rapid and complete inspiration with a pause of less than two seconds at total lung capacity. This was followed by a maximal forced expiration until no further air could be expelled while maintaining an upright posture, and, finally, a maximal inspiration until full lung inflation was achieved.

The independent variables in this study were diaphragmatic thickness and diaphragmatic thickness fraction. Data on diaphragmatic thickness and pulmonary function parameters were obtained simultaneously in a single measurement session.[14] The dependent variables were pulmonary function parameters in patients with COPD, including the FEV_1/FVC ratio, percent predicted FEV_1, and percent predicted FVC.

Statistical analysis was performed using SPSS software version 20. Univariate analysis was conducted to describe diaphragmatic thickness and pulmonary function parameters among patients with COPD. Data normality was assessed using the Kolmogorov–Smirnov test, and a value of $P>0.05$ indicated a normal distribution.

Pearson's correlation test was used for normally distributed data. In contrast, Spearman's correlation test was applied for non-normally distributed data to evaluate the relationships between diaphragmatic thickness and pulmonary function parameters, including percent predicted FEV_1, percent predicted FVC, and the FEV_1/FVC ratio. A value of $P<0.05$ was considered statistically significant.

RESULT

A total of 50 COPD patients, consisting of 82% males and 18% females, with a mean participant age of 62.14±10.03 years, were enrolled in this study. Table 1 summarizes the demographic and clinical characteristics of the study participants.

Table 1. Characteristics of Patients with Acute Chronic Obstructive Pulmonary Disease

Characteristics	n	%
Age, years (mean±SD)	62.14±10.03	
Gender		
Male	41	82
Female	2	18
Body Weight (kg)	61.38±13.70	
Body Height (cm)	162.64±6.58	
Body Mass Index (kg/m^2)	23.17±4.69	
mMRC grading		
1	8	16
2	13	26
3	15	30
4	14	28
GOLD class		
A	5	10
B	23	46
C	0	0
D	0	0
E	22	44
GOLD grading		
GOLD 1	2	4
GOLD 2	5	10
GOLD 3	17	34
GOLD 4	26	52
FEV_1/FVC	0.60±0.08	
Percent predicted FEV_1	34.34±16.77	
Percent predicted FVC	44.70±17.31	
Diaphragm Thickness	0.30±0.08	

Note: GOLD=The Global Initiative for Obstructive Lung Disease; mMRC=modified Medical Research Council ; FEV_1=Forced Expiratory Volume in 1 second; FVC=Forced Vital Capacity

The mean body weight was 61.38±13.70 kg, with an average height of 162.64±6.58 cm, resulting in a mean body mass index of 23.17±4.69 kg/m^2. Based on the modified Medical Research Council (mMRC) dyspnea scale, most patients had moderate to severe symptoms, with grades 2 and 3 being the most frequent (26% and 30%, respectively), followed by grade 4 (28%) and grade 1 (16%).

Regarding GOLD classification, most patients were classified as group B (46%) and group D (44%), while a smaller proportion belonged to group A (10%), and no patients were classified as groups C or E. In terms of GOLD spirometric grading, the majority of patients were classified as GOLD 4 (52%) and GOLD 3 (34%), with fewer patients in GOLD 2 (10%) and GOLD 1 (4%). Pulmonary function tests showed a mean FEV_1/FVC ratio of 0.60±0.08, a mean percent predicted FEV_1 of 34.34±16.77%, and a mean percent predicted FVC of 44.70±17.31%, indicating predominantly severe airflow limitation.

Table 1 also presents the descriptive statistics of diaphragm thickness, with a mean of 0.30 and a standard deviation of 0.08, indicating moderate variability among patients. This finding reflects the overall distribution of diaphragm muscle thickness measured across the study participants.

Table 2. Correlations of diaphragm thickness with pulmonary function

Variables	Diaphragm Thickness	
	r	P
FEV_1/FVC	-0.184	0.201
Percent predicted FEV_1	0.188	0.190
Percent predicted FVC	0.168	0.243

Note: FEV_1=Forced Expiratory Volume in 1 second; FVC=Forced Vital Capacity

Table 2 illustrates the correlation between diaphragm thickness and pulmonary function parameters. Diaphragm thickness showed a weak negative correlation with the FEV_1/FVC ratio ($r = -0.184$), although this association was not statistically significant (P=0.201). Weak positive correlations were observed between diaphragm thickness and percent predicted FEV_1 ($r = 0.188$; P=0.190) and percent predicted FVC ($r = 0.168$; P=0.243). However, none of these correlations were statistically significant, suggesting that diaphragm thickness was not significantly associated with spirometric parameters in this study.

DISCUSSION

This study evaluated diaphragm thickness and its relationship with pulmonary function in patients with predominantly moderate to very severe chronic airflow limitation. The study population was largely composed of older adults with a high proportion of male patients and advanced disease severity, as reflected by the predominance of GOLD grades 3 and 4 and low mean values of percent predicted FEV_1 and FVC.

These characteristics are consistent with populations commonly affected by advanced COPD, in whom respiratory muscle involvement is expected. Patients with more advanced COPD were generally older, consistent with the progressive nature of chronic respiratory diseases. Male gender and advancing age may contribute to greater disease severity or represent the cumulative effects of long-term exposure to risk factors such as smoking.[15,16]

The mean diaphragm thickness observed in this study was 0.30±0.08, indicating measurable variability among patients. Kareem et al stated that diaphragm thickness and diaphragmatic thickening fraction were significantly

increased in patients with COPD compared with control subjects. On the right side, the mean inspiratory diaphragmatic thickness was higher in the COPD group than in controls (3.23±0.68 mm vs 2.80±0.55 mm; P=0.006), accompanied by a significantly greater right diaphragmatic thickening fraction (2.44±0.71 vs. 1.96±0.60; P=0.004). Similarly, the mean left inspiratory diaphragmatic thickness was significantly greater in COPD patients than in controls (3.22±0.63 mm vs. 2.79±0.51 mm; P=0.003), along with a higher left diaphragmatic thickening fraction (2.45±0.63 vs. 1.95±0.54; P=0.003).[17]

Jain et al stated that the mean diaphragmatic thickness during both inspiration and expiration, as well as the magnitude of thickness change and the zone of apposition, was significantly greater in patients classified as COPD Grade C compared with those in Grade A or B.[13] Diaphragmatic dysfunction is frequently observed in patients with COPD and may arise from multiple underlying mechanisms. Among these, pulmonary hyperinflation and malnutrition are the most prominent contributors to respiratory muscle weakness. Lung hyperinflation displaces the diaphragm caudally, resulting in a mechanical disadvantage that compromises its contractile efficiency. Therefore, diaphragm thickness should be interpreted cautiously as an isolated marker of diaphragmatic function.[18]

In this study, diaphragm thickness demonstrated only weak correlations with spirometric parameters. A weak negative correlation was observed between diaphragm thickness and the FEV_1/FVC ratio, while weak positive correlations were observed with percent-predicted FEV_1 and FVC. None of these associations reached statistical significance. These findings suggest that diaphragm thickness alone may not be a strong surrogate for airflow limitation or lung volumes as assessed by standard spirometry.

The findings of this study are consistent with those of previous research conducted by Ogan et al. Diaphragmatic thickness showed no significant association with spirometric indices of airflow limitation in patients with COPD. In this case–control study involving 34 COPD patients and 34 healthy controls, diaphragmatic thickness measured during tidal breathing (T_{min}) and deep inspiration (T_{max}) on both sides did not correlate with FEV_1/FVC, percent-predicted FEV_1, or percent-predicted FVC. Specifically, correlation analysis demonstrated no significant relationship between right-sided diaphragmatic thickness and FEV_1 (T_{min}R: r = 0.19; P=0.30; T_{max}R: r = −0.18, P=0.30), and similar non-significant findings were observed for left-sided measurements.[11]

Furthermore, diaphragmatic thickness did not differ across spirometric severity categories based on percent predicted FEV_1, nor was it associated with overall spirometric function (P=0.410). These results indicate that static diaphragmatic thickness, as assessed by ultrasonography, does not reflect airflow obstruction or spirometric severity, supporting the conclusion that diaphragmatic dysfunction in COPD is more

closely related to impaired mobility than to muscle thickness.[11] This may also be explained by the multifactorial nature of pulmonary function impairment, which depends not only on respiratory muscle characteristics but also on airway obstruction, lung compliance, and chest wall mechanics.[19]

The lack of significant correlation may also reflect the complexity of diaphragm adaptation in chronic respiratory disease. In some patients, chronic inspiratory loading may lead to compensatory hypertrophy of the diaphragm, whereas in others, muscle atrophy and dysfunction may predominate due to disuse, systemic inflammation, or corticosteroid exposure.

Additionally, spirometric indices such as FEV_1 and FVC primarily reflect airway and lung parenchymal pathology rather than respiratory muscle performance. Functional parameters such as diaphragmatic excursion, thickening fraction, or inspiratory muscle strength may better capture diaphragmatic contribution to ventilation than static thickness measurements.

Clinically, the predominance of higher mMRC grades and advanced GOLD classifications in this cohort highlights the substantial symptom burden and functional limitation of the study population. The absence of a clear association between diaphragm thickness and pulmonary function suggests that diaphragm ultrasound parameters should be interpreted in conjunction with clinical severity, symptom scores, and other functional assessments rather than as standalone indicators.

However, diaphragmatic ultrasonography represents a reliable modality for evaluating both structural and functional aspects of the diaphragm in patients with COPD. Accordingly, its routine incorporation into clinical management may be beneficial, particularly for patients presenting with dyspnea. As a non-invasive and easily accessible technique, ultrasound also has potential utility as an early screening tool to identify individuals at risk of diaphragmatic dysfunction, including those in the initial stages of COPD.[16,20,21]

Several limitations should be considered. The cross-sectional design limits causal inference, and the relatively small sample size may reduce statistical power to detect subtle associations. Additionally, diaphragm function was assessed only by thickness, without evaluation of dynamic parameters such as excursion or contractility. Future studies incorporating longitudinal designs and comprehensive diaphragmatic functional assessment may provide greater insight into the role of diaphragm morphology in disease severity and progression.

CONCLUSION

In this study, diaphragm thickness demonstrated moderate variability among patients with predominantly advanced airflow limitation. However, diaphragm thickness showed only weak and non-significant correlations with spirometric parameters, including FEV_1/FVC, percent

predicted FEV_1, and percent predicted FVC. These findings suggest that diaphragm thickness alone may not serve as a reliable indicator of pulmonary function impairment. A comprehensive assessment incorporating clinical severity, functional status, and dynamic diaphragmatic measurements may be necessary to better understand respiratory muscle involvement in patients with COPD.

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REFERENCES

1. Perhimpunan Dokter Paru Indonesia. Penyakit paru obstruktif kronik: diagnosis dan penatalaksanaan. Jakarta: Perhimpunan Dokter Paru Indonesia; 2016.
2. Raad S, Smith C, Allen K. Nutrition Status and Chronic Obstructive Pulmonary Disease: Can We Move Beyond the Body Mass Index? Nutrition in Clinical Practice. 2019;34(3):330–9.
3. Agustí A. Systemic Effects of Chronic Obstructive Pulmonary Disease: What We Know and What We Don't Know (but Should). Proc Am Thorac Soc. 2007;4(7):522–5.
4. Bailey KL, Goraya J, Rennard SL. The Role of Systemic Inflammation in COPD. In: Chronic Obstructive Pulmonary Disease. Totowa, NJ: Humana Press; 2012. p. 15–30.
5. Wouters EFM, Groenewegen KH, Dentener MA, Vernooy JHJ. Systemic Inflammation in Chronic Obstructive Pulmonary Disease: The Role of Exacerbations. Proc Am Thorac Soc. 2007;4(8):626–34.
6. Gea J, Pascual S, Casadevall C, Orozco-Levi M, Barreiro E. Muscle dysfunction in chronic obstructive pulmonary disease: update on causes and biological findings. J Thorac Dis. 2015;7(10):E418–38.
7. Boussuges A, Rives S, Finance J, Brégeon F. Assessment of diaphragmatic function by ultrasonography: Current approach and perspectives. World J Clin Cases. 2020;8(12):2408–24.
8. Santana PV, Albuquerque ALP de. Respiratory muscles in COPD: be aware of the diaphragm. Jornal Brasileiro de Pneumologia. 2018;44(1):1–2.
9. Lim SY, Lim G, Lee YJ, Cho YJ, Park JS, Yoon H Il, et al. Ultrasound Assessment Of Diaphragmatic Function During Acute Exacerbation Of Chronic Obstructive Pulmonary Disease: A Pilot Study. Int J Chron Obstruct Pulmon Dis. 2019;14:2479–84.

10. Scarlata S, Mancini D, Laudisio A, Benigni A, Incalzi RA. Reproducibility and Clinical Correlates of Supine Diaphragmatic Motion Measured by M-Mode Ultrasonography in Healthy Volunteers. 2018;96(3):259–66.
11. Ogan N, Aydemir Y, EVrin T, Ataç GK, Baha A, Katipoğlu B, et al. Diaphragmatic thickness in chronic obstructive lung disease and relationship with clinical severity parameters. Turk J Med Sci. 2019;49(4):1073–8.
12. Cimsit C, Bekir M, Karakurt S, Eryuksel E. Ultrasound assessment of diaphragm thickness in COPD. Marmara Medical Journal. 2016;29(1):8–13.
13. Jain S, Nair G, Nuchin A, Uppe A. Study of the diaphragm in chronic obstructive pulmonary disease using ultrasonography. Lung India. 2019;36(4):299–303.
14. Dong Z, Zhao M, Zheng S, Gu H, Cao N, Sun X, et al. Ultrasound Evaluation of Diaphragmatic and Intercostal Muscle Function as an Indicator of COPD Severity: A Prospective Cross-Sectional Study. Int J Chron Obstruct Pulmon Dis. 2025;20:4115–28.
15. Gulnaz H, Sardar M, Jafri SR, Zakia S, Saeed K. Investigating the influence of Chronic Obstructive Pulmonary Disease severity on the thickness of the Diaphragm Muscle. Pakistan Journal of Chest Medicine. 2025;30(2):203–10.
16. Meridj A, Belaala R, Djeghri Y. The Importance of Measuring the Fraction of Diaphragmatic Thickening in Patients with COPD. Global Journal of Medical and Clinical Case Reports. 2025;12(2):047–54.
17. Kareem JM, Al-Waely NK, Al-Hashimi AF. Diaphragmatic Thickness in Chronic Obstructive Pulmonary Disease. Medical Journal of Babylon. 2022;19(3):427–33.
18. Chandrakumar AK, Baby J, Xavier ESC, Valsalan P, Puthanveetti R. Correlation Between Ultrasonographic Evaluation of Diaphragm Excursion, Thickness and Spirometry in COPD patients: A Case-Control Study. Indian Journal of Respiratory Care. 2023;12(1):33–7.
19. Ibrahim AA, Abd-Eldayem AMM, Hamid HEMA. Relation between pulmonary function changes and diaphragmatic ultrasound in patients with idiopathic pulmonary fibrosis. The Egyptian Journal of Bronchology. 2025;19:7.
20. Schulz A, Erbuth A, Boyko M, Vonderbank S, Gürleyen H, Gibis N, et al. Comparison of Ultrasound Measurements for Diaphragmatic Mobility, Diaphragmatic Thickness, and Diaphragm Thickening Fraction with Each Other and with Lung Function in Patients with Chronic Obstructive Pulmonary Disease. Int J Chron Obstruct Pulmon Dis. 2022;17:2217–27.
21. Abd El-hay AE hay I, Abd Elnaby HEH, El-gamal MMME. Ultrasonographic Assessment of Diaphragmatic Function and Its Correlation with Chronic Obstructive Pulmonary Disease Severity. Egypt J Hosp Med. 2019;76(7):4524–32.