Journal of Recent Advances in Medicine

JRAM
Journal of Recent Advances in Medicine
مجلة التطورات الحديثة في الطب
Print ISSN 2636-252X - Online ISSN 2636-2538

Original Article

Ultrasonographic evaluation of diaphragm function in patients with chronic obstructive pulmonary disease and patients with bronchial asthma: Comparative study

Pulmonology

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ABSTRACT

Background: Chronic obstructive pulmonary disease (COPD) and bronchial asthma lead to chronic airflow limitation, which is supposed to alter the position and shape of the diaphragm due to increased lung volume with subsequent impairment of diaphragm contractility.

Objective: To evaluate and compare diaphragm thickness (DT) using ultrasonography in COPD patients and asthma patients.

Methodology: A participants were subjected to measurements of spirometric indices and assessment of DT using ultrasound.

Results: The DT at residual volume (DT-RV) was significantly decreased in COPD patients than either asthma or control groups. The DT at total lung capacity (DT-TLC) and diaphragm thickness fraction (DTF %) were significantly reduced in COPD group than both asthma and control groups, and in asthma group than control group. In asthma group, the DTF% was inversely correlated with smoking index and asthma duration, and positively correlated with FEF25-75%. In COPD group, the DTF% was positively correlated with FEV_1/FVC ratio and FEV_1%. The DTF% cutoff 24.5% can discriminate between normal diaphragm function and diaphragm dysfunction (DD) in asthmatic patients with 74% sensitivity, 62.4% specificity, 67.3% PPV and 69.3% NPV. In COPD, the DTF% cutoff 23.7% can discriminate between normal diaphragm function and DD with 85% sensitivity, 66.3% specificity, 73.2% PPV and 79.3% NPV. DD was detected in 25% of asthmatic patients and 37% of COPD patients. The important predictive factors of DD in asthmatic patients were FVC%, age, FEV_1% and asthma duration, while in COPD it were COPD duration, age, smoking index, FEV_1% and body mass index (BMI).

Conclusion: DD is prevalent among both COPD and asthmatic patients. The DT and contractility were significantly declined in both asthmatic and COPD patients compared to healthy subjects, and in COPD patients than asthma patients.

JRAM 2023; 4(1):144-154

Keywords: Bronchial asthma, COPD, diaphragm thickness, DTF%, diaphragm function, diaphragm dysfunction.

Submission Date: 18 July 2023 **Acceptance Date:** 5 August 2023

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Please cite this article as: Eysa AM, Hafez MR , Moezn EM. Ultrasound evaluation of diaphragm function in patients with chronic obstructive pulmonary disease and patients with bronchial asthma: Comparative stud. JRAM 2023; 4(1):144-154. DOI: 10.21608/jram.2023.223449.1218

INTRODUCTION

Chronic obstructive pulmonary disease (COPD) and bronchial asthma are obstructive airway diseases that are characterized by airway obstruction, which is irreversible and progressive in COPD, and reversible and variable in asthma. Chronic airflow limitation, caused by COPD or asthma, is supposed to alter the position and shape of the diaphragm caused by an increased lung volume[1].

COPD affects all body systems and diaphragm dysfunction (DD) is one of the prominent manifestations of extrapulmonary effects of COPD. An imbalance of respiratory muscles load/ capacity generally exist in COPD[2]. In COPD, DD is manifested as structural changes, e.g., single-fibre dysfunction, sarcomere injury and fibre type transformation, diaphragm atrophy, and functional changes e.g. reduced muscle strength and endurance, decreased mobility, and diaphragmatic fatigue [3]. Muscle-fiber shortening occurs due to lung hyperinflation, which puts diaphragm under persistent mechanical disadvantage and reduces its contractility [4]. In asthmatic patients, airway obstruction increases airway resistance and generates a threshold load that should be overcome with each breath. As function of the respiratory muscles is frequently compromised, it may contribute to sensation of dyspnea, therefore,

evaluation of their functions is of major clinical importance [5].

Although numerous methods have been used in assessing diaphragm function, the diagnosis of DD is still challenging [6]. Several techniques are present to evaluate the diaphragm, based on the conditions under concern, these techniques can be performed to evaluate either function (i.e, force-generating capacity) or diaphragm contractility [7]. Most of the methods used to identify diaphragm weakness or paralysis are expensive, indirect, expose patients to radiation, invasive, time-consuming, and uncomfortable or complex [8].

Diaphragm ultrasound (US) is a promising technique and has expanded its importance because of its many advantages e.g., widely available, no radiation exposure, noninvasive, give immediate results, highly accurate, done and repeatable at the bedside, and inexpensive. Several investigators have described US techniques to evaluate diaphragm function at zone of apposition (ZOA) which is defined as area of the chest wall where the abdominal contents reach the lower thoracic cage, many studies have documented standardization of this technique [6]. The increase of diaphragm thickness (DT) during contraction, known as the diaphragm thickness fraction (DTF%) indicate diaphragm inspiratory contractile activity [9]. US measurements of diaphragm kinetics can help in the early detection of structural changes produced by higher respiratory demand experienced throughout the patient's life [10].

Although numerous studies evaluate diaphragm US in patients with COPD, few studies assess it in asthmatic patients [5] [10-11]. Also, there is no data available about comparison of (DT) of asthmatic patients to those with COPD. Therefore, we designed this study to evaluate and compare DT using US between asthmatic patients and COPD patients.

SUBJECTS AND METHOD

This case-control study was done at chest department, Al-Zahraa university hospital, Cairo, Egypt, during the period from March 2022 to May 2023. It was conducted after approval by the ethical committee of faculty of medicine for girls Al-Azhar university, Cairo, Egypt. An informed consent was gotten from all participants before included into the study.

Sample size

According to annual flow of COPD and its prevalence in Egypt (6.6%) [12] and the annual flow of asthma and its prevalence in Egypt (6.8%) [13], the sample size was calculated by Epi info, Atlanta, Georgia (US), and it was found that the statistically representive sample of either COPD or asthma was 96 patients in each group (margin of error 5% and confidence level 95%). Accordingly, this study was carried on 300 subjects, they were classified into 3 groups:

1. **Asthma group:** 100 known asthmatics patients, they were diagnosed several years ago based on typical asthma symptoms and signs with positive bronchodilator reversibility test at time of diagnosis.
2. **COPD group:** 100 known COPD patients, they were diagnosed several years ago based on typical COPD symptoms and signs with negative bronchodilator reversibility test at time of diagnosis.
3. **Control group:** included 103 healthy individuals.

Exclusion criteria: participants under 18 years and those with the following diseases were excluded from the study; neuromuscular disorders, diaphragmatic hernia, malignancies or central bronchogenic carcinoma, phrenic nerve injury, recent abdominal or thoracic surgery, traumatic lesion possibly affecting diaphragm, and severe malnutrition.

Data regarding sex, age, smoking index, body mass index (BMI), and disease duration were recorded. Pulmonary function test was done using (MEDISOFT-HYPERAIR compact + flow meter pulmonary function testing-Belgium). The FEV_1/FVC ratio, FEV_1%, FVC%, and FEF 25-75% were reported.

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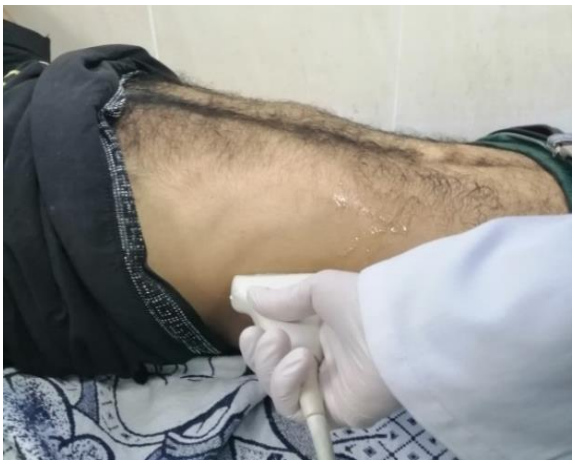
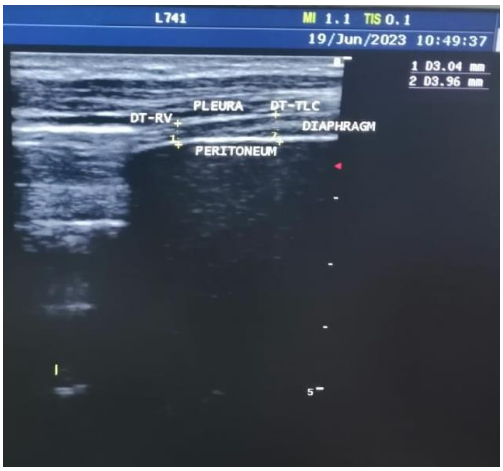


A. transducer was held perpendicular to the chest wall in the 9th intercostal space perpendicular to the 9th and 10th ribs, between the anterior and the midaxillary lines



B. ZOA is an area of the chest wall where the abdominal contents reach the lower rib cage

Figure (1): US measurement of diaphragm thickness

Diaphragm US was assessed by Sonoscape SSI-6000 (Medical Systems, Shenzhen, China). Optimal gain, depth of placement, and compression were adjusted individually for each participant. We assess DT of right hemi-diaphragm because evaluation of left one might be difficult due to small acoustic window of the spleen and interposition of gas in the stomach. The high frequency transducer (9.5-15 MHz) was putted in 9^{th} intercostal space perpendicular to 9^{th}-10^{th} ribs, midway between anterior axillary and midaxillary lines, the ZOA can be detected optimally 0.5-2 cm below the costophrenic sinus [6]. At the ZOA, the diaphragm is visualized as a structure composed of 3 distinct layers; 2 echogenic layers (peritoneum, and diaphragmatic pleura) surrounded central non-echogenic layer [14]. The DT was measured from the middle of the diaphragmatic pleura to the middle of the peritoneal membrane [15] (figure 1). The DT was measured at residual volume [DT-RV] and at total lung capacity (DT-TLC), then the DTF% was calculated using the following equation: $DTF\% = \frac{(DT-TLC) - (DT-RV)}{(DT-RV)} X100$ [16].

Statistical analysis

Data was statistically analysed using SPSS program version 17.0 (Chicago, USA). The data was expressed as mean ±SD for parametric quantitative data, median with interquartile range (IQR) for non-parametric data, and percentages for qualitative data. Kruskal Wallis test was used to compare between the 3 groups to be followed by least significant test (LST) for multiple comparison between each two groups. Mann Whitney (MW) was used for comparison between two groups. Chi-square (X^2) test was used for comparison of qualitative data between the groups. Linear correlation coefficient test was used for detection of association between two quantitative variables in either asthma group or COPD group. Multivariate logistic regression analysis was used to identify the most significant predictive factors of DD in either asthma group or COPD group. Receiver operator characteristic curve (ROC) test was used to determine optimal cutoff value of DTF% and to assess the effectiveness of this cutoff for discrimination between normal diaphragm function and DD in either asthma group or COPD group [sensitivity, specificity, positive predictive value (PPV), and negative predictive value (NPV)]. For used tests the statistical significance was set at p-value ≤0.05 (95% confidence limit).

RESULTS

There was female predominance in asthma group and male predominance COPD and control groups (p= 0.001). COPD patients have significantly higher smoking index than asthmatics and controls, while asthma patients have lower smoking index than controls (p<0.05). Asthmatic patients have significantly longer disease duration than COPD patients (p =0.002). The FEV_1/FVC ratio, FEV_1%, FVC%, and FEF25-75% were significantly lower in COPD patients than either asthmatics or controls, and in asthmatic patients than controls (p<0.05). The majority of asthmatic patients (52.0%, 32.0%) have moderate or severe disease respectively, while the majority of COPD patients have moderate (43.0%), severe (28.0%) or very severe disease (25.0%). The DT-RV was significantly reduced in COPD group than either asthmatics or controls (0.001 and 0.003). The DT-TLC and DTF% were significantly reduced in COPD patients than asthmatics or controls (p < 0.05), and in asthmatics compared to controls (p < 0.05) (table 1).

In asthma, the DTF% was inversely correlated with smoking index and disease duration and positively correlated with FEF 25-75% (p<0.05). In COPD, the DTF% was positively correlated with FEV_1/FVC ratio and FEV_1% (table 2, figures (2a, 2b, 2c).

In asthma, the DTF % at a cutoff 24.5% can discriminate between normal diaphragm function and DD with 74% sensitivity, 62.4% specificity, 67.3% PPV and 69.3% NPV. In COPD group, the DTF% at a cutoff 23.7% can discriminate between normal diaphragm function and DD with 85% sensitivity, 66.3% specificity, 73.2% PPV and 79.3% NPV. Moreover, 25% of asthmatic patients and 37% of COPD patients had DD (table 4, figure 3).

The significant predictive factors for DD in asthma patients were FVC% (β= 0.74), age (β= -0.39,), FEV_1% (β= 0. 35) and disease duration (β= -0.15). The significant predictive factors for DD in COPD patients were; disease duration (β= -0.85), age (β= -0.68), smoking index (β= -0.63), FEV_1% (β= 0.41) and BMI (β= 0.15) (table 4).

Table (1): Comparison of studied variables between the studied groups

items		Groups			Stat. test	p- value	Post-Hoc analysis#		
		BA (n = 100)	COPD (n = 100)	Control (n = 103)			P1	P2	P3
Sex	Male	16 (16%)	91 (91%)	92 (89.3%)	X^2 = 163.5	0.001*	0.001*	0.001*	0.688
	Female	84 (84%)	9 (9%)	11 (10.7%)					
Age (yrs.)	Median (IQR)	58 (42-62)	59 (57-69)	62 (59-65)	KW = 4.8	0.071			
BMI (kg/m²)	Median (IQR)	30.3(27.4-32.9)	29.5(24.5-31.1)	29.2(27-31.1)	KW = 1.4	0.06			
Smoking index (p/y)	Median (IQR)	8 (6.5-10.5)	33.7 (28-50)	13 (8-17)	KW = 80.5	0.001*	0.002*	0.3	0.013*
Disease duration (yrs.)	Median (IQR)	16 (12-26)	12 (8-18)	----	MW = 3235.5	0.002*			
FEV_1/FVC ratio	Median (IQR)	71 (68-74)	61.5 (55-68)	39.5 (85-99)	KW = 239	0.001*	0.002*	0.021*	0.001*
FEV_1%	Median (IQR)	64 (47-70)	47 (29-66)	32.7 (79-86)	KW = 162.3	0.001*	0.010*	0.002*	0.001*
FVC%	Median (IQR)	83 (80-88)	63.5 (49-71)	92.6 (87-100)	KW = 190	0.001*	0.002*	0.003*	0.001*
FEF 25-75%	Median (IQR)	66.3 (59-70)	32 (27-50)	72 (68-77)	MW = 210.9	0.001*	0.003*	0.002*	0.001*
Severity	Mild	8 (8.0%)	4 (4.0%)	X^2 = 11.2	0.011*				
	Moderate	52 (52%)	43 (43%)						
	Severe	32 (32%)	28 (28%)						
	Very severe	8 (8%)	25 (25%)						
DT-FRC (mm)	Median (IQR)	2.9(2.7-3.1)	2.7(2.2-3.1)	3(2.6-3.6)	KW=21.8	0.002*	0.001*	0.322	0.003*
DT-TLC (mm)	Median (IQR)	3.8(3.5-4.5)	3.6(2.9-4.3)	3.9(3.4-4.8)	KW=15.5	0.003*	0.001*	0.038*	0.002*
DTF%	Median (IQR)	33.6(30-36)	31.8(28-33)	35.2(30-39)	KW=26.9	0.001*	0.004*	0.012*	0.001*

BA: Bronchial asthma, COPD: Chronic obstructive pulmonary disease, p/y: Pack/year, BMI: Body mass, X^2: Chi square test, KW: Kruskal Willis test, MW: Mann Whitney U test,:FEV_1%: Forced expiratory volume in first second %, predicted FVC: Forced vital capacity % predicted, Forced expiratory flow at 25-75% of vital capacity % predicted, DT-FRC: Diaphragm thickness at functional residual capacity, DT-TLC: Diaphragm thickness at total lung capacity, DTF %: Diaphragm thickness fraction %, #: Least significant difference, *: Significant p-value (< 0.05), P1:Asthma vs. COPD, P2: Asthma vs. control, P3: COPD vs. control.

Table (2): Correlation of diaphragm thickness fraction with other studied variables in bronchial asthma group and COPD group

Studied variables	Asthma group		COPD group	
	r	p-value	r	p-value
Age (years)	0.04	0.707	-0.06	0.546
BMI (kg/m²)	0.07	0.481	0.12	0.23
Smoking index (p/y)	-0.70	0.024*	-0.06	0.56
Disease duration (years)	-0.29	0.047*	-0.02	0.865
FEV_1/FVC ratio	0.05	0.618	0.32	0.001*
FEV_1%	0.04	0.664	0.26	0.008*
FVC %	-0.06	0.577	0.01	0.891
FEF 25-75%	0.30	0.003*	-0.04	0.716

BMI: Body mass index, p/y: pack/ year, FEV_1%: Forced expiratory volume in first second %, predicted FVC: Forced vital capacity % predicted, Forced expiratory flow at 25-75% of vital capacity % predicted, DFT% Diaphragm thickness fraction, ICS: Inhaled corticosteroid, r: Pearson correlation coefficient, *: Significant p-value (< 0.05).

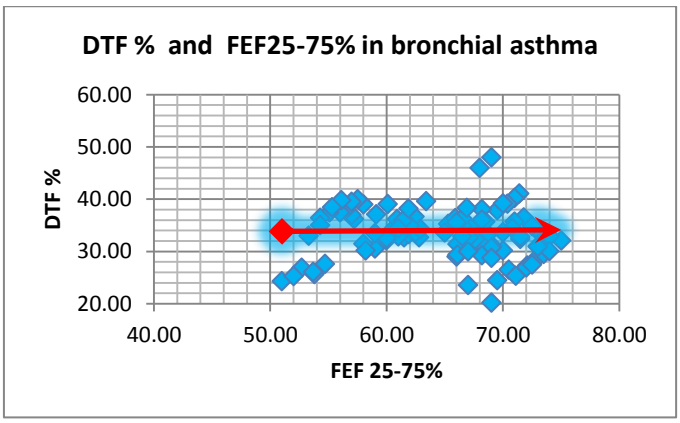


Figure (2a): Correlation of diaphragm thickness fraction with FEF25-75% in bronchial asthma group

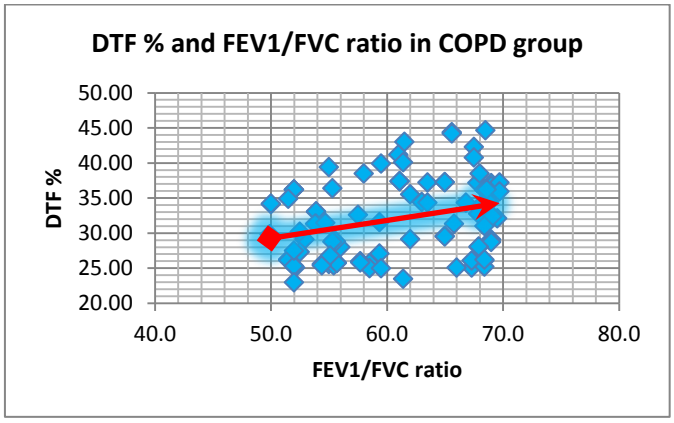


Figure (2b): Correlation of diaphragm thickness fraction with FEV_1/FVC ratio in COPD group

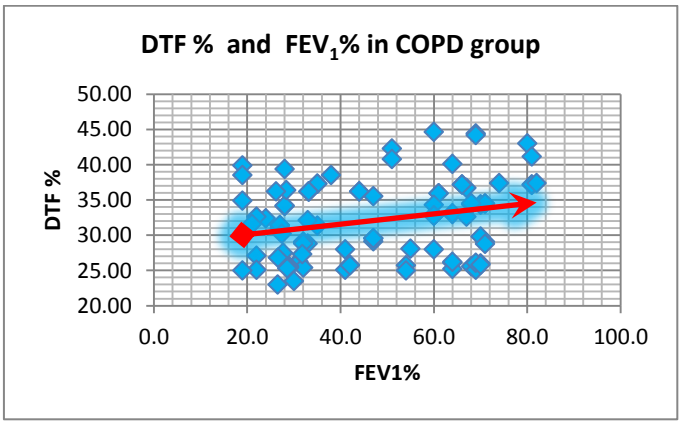


Figure (2c): Correlation of diaphragm thickness fraction with FEV_1 % in COPD group

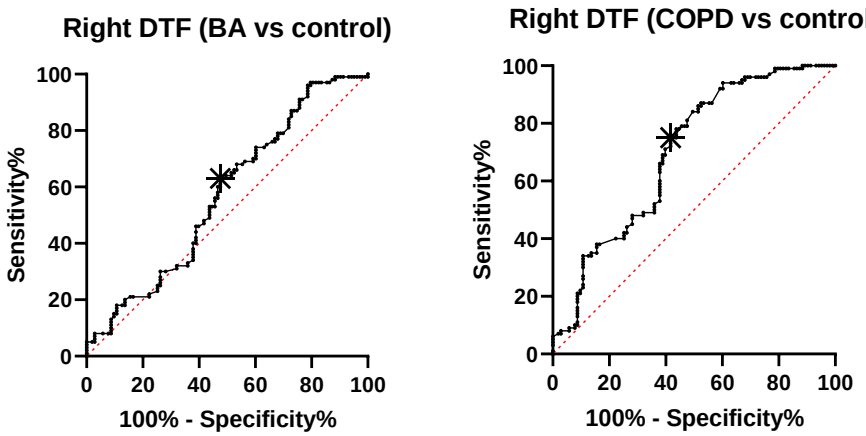


Figure (4): ROC curve for DTF% cutoff for detection of diaphragm dysfunction in bronchial asthma group and COPD group

Table (3): Diagnostic performance of DTF% cutoff for detection of diaphragm dysfunction in bronchial asthma group and COPD group

DTF%	Cutoff	AUC	Sensitivity	Specificity	PPV	NPV
Bronchial asthma	< 24.5%	0.56	74%	62.4%	67.3%	69.3%
COPD	< 23.7%	0.69	85%	66.3%	73.2%	79.3%

BA: Bronchial asthma, COPD: Chronic obstructive pulmonary disease, AUC: Area under curve, DTF %: Diaphragm thickness fraction %, PPV: Positive predictive value, NPV: Negative predictive value.

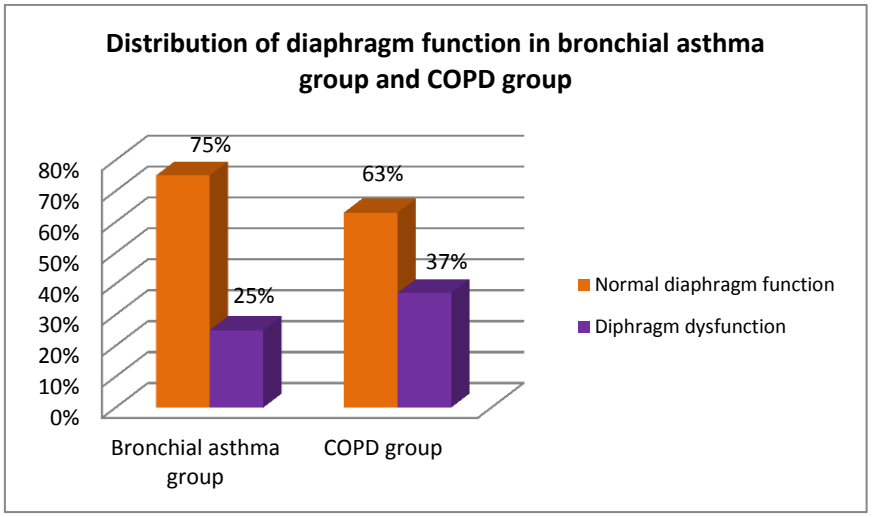


Figure (3) Distribution of diaphragm function in bronchial asthma group and COPD group

Table (4): Multivariate logistic regression analysis for factors predictive of diaphragm dysfunction in bronchial asthma group

Variables	β	SE	p-value	Odds	95% CL	
Bronchial asthma group						
Age (years)	-0.39	0.19	0.045*	1.47	1.0	2.1
Asthma duration (yrs.)	- 0.15	0.041	0.001*	0.86	0.79	0.93
FEV_1%	0. 35	0.016	0.023*	1.03	1.0	1.06
FVC%	0. 74	0.037	0.047*	1.07	1.0	1.15
COPD group						
Age (yrs.)	- 0.68	0.29	0.02*	0.5	0.28	0.89
COPD duration (yrs.)	- 0.85	0.38	0.027*	0.42	0.2	0.9
Smoking index (p/y)	- 0.63	0.25	0.011*	0.53	0.32	0.86
BMI (kg/m^2)	0.15	0.049	0.001*	0.85	0.77	0.94
FEV_1%	0.41	0. 11	0.012*	1.08	0.9	0.99

FEV_1%: Forced expiratory volume in first second % predicted, FVC: Forced vital capacity % predicted, CS: Inhaled corticosteroid, β: Regression coefficient, SE: Standard error, CL: Confidence limit

DISCUSSION

In severe COPD, the strength of respiratory muscles is an independent factor of patient's survival [17]. As respiratory muscles function compromised in asthmatic patients, it may contribute to sensation of dyspnea, accordingly, evaluation of their functions are of clinical importance [5].

In this study, the significant decrease of DT-FRC and DT-TLC in COPD patients than asthmatics and controls, the significant decrease of DT-TLC and DTF% in COPD than both asthmatic and controls, and in asthmatics than controls, indicate that the diaphragm inspiratory contractile activity were altered in patients with either COPD or asthma. However, it is more altered in COPD than asthma. A possible explanation for these findings would be the nature of COPD as a disease characterized by chronic irreversible airway obstruction with subsequent dynamic air trapping and chronic hyperinflation which put the diaphragm under mechanical disadvantages and impaired its contractility. On the other hand, it is well known that asthma is an intermittent disease with reversible airway obstruction especially in its early stages, therefore the diaphragm in some asthmatic patients might maintain its contractility. However, the DT-TLC and DTF% are lower in our asthmatic patients than controls which may be due to that considerable proportion of our asthmatic patients have advanced disease (32% have severe asthma and 8% have very severe asthma), which may be associated with irreversible airway obstruction and chronic hyperinflation.

In COPD, Topcuoğlu.et al. [18] concluded that based on the studies in the literature, we think that the DD may be due to many etiological factors such as systemic inflammation, oxidative stress, drugs that cause respiratory muscle dysfunction, and hyperinflation. Lung hyperinflation has both static and dynamic components, static component resulting from lung parenchyma destruction and loss of elastic recoil of the lungs, while dynamic component occurs when COPD patients inhale before finishing full exhalation [19]. Lung hyperinflation leads to mechanical changes with subsequent impairment of force-length relationship of diaphragm that limits its contractility as a result of the shortening of the diaphragm, which is positioned in a non-optimal position [80]. In addition to airway obstruction and hyperinflation, myopathy due to systemic inflammation and oxidative can cause DD in COPD. Also, muscle wasting, decreased protein production, and enhanced apoptosis contribute to DD [8]. However, the exact mechanism of development of DD in COPD is still blurry, which to some extent, made treatment and rehabilitation more challenging [3].

In asthma, many mechanisms lead to imbalance between respiratory load and capacity with consequent respiratory muscle weakness. Numerous inflammatory mediators in asthma can reduce diaphragm contractility by inducing synthesis of oxygen free radicals which can cause oxidative damage to the myofilaments and regulatory proteins of sarcoplasma. However, hyperinflation was documented by many researchers to be the principle underlying cause of DD [5]. In addition to small airway closure occurring at lower lung volumes during slow exhalation, there may be more closure during forced exhalation due to dynamic airway compression [21].

Similarly, Hafez and Abo-Elkheir [17], found that the right DTF% was significantly reduced in COPD, while DT-RV and DT-TLC were non-significant differed between COPD patients and controls. Elsawy [22] reported that the DTF% bilaterally were significantly reduced in COPD patients than healthy controls. However, the static TD indices did not differ between patients and controls. They explained these findings by that at the end inspiration and end expiration DT were preserved in COPD patients due to sarcomere adaptation of the diaphragm muscle fiber that protecting the static DT with development of compensatory overuse hypertrophy. Essawy et al. [2] study showed a significant decrease of DT-TLC and DT-FR, and DTF% in COPD group compared to control group. Many previous researches have documented that COPD patients have a significantly reduced DT than healthy subjects [18] [23 - 29]. Additionally, Hua-Rong et al. [28] in their systematic review reported that the DT was significantly decreased in COPD than controls. Different findings were reported by Baria et al. [29] as they documented that no significant difference between COPD patients and controls regarding DT except the left DTF% that was significantly higher in COPD patients with air trapping than COPD patients without air trapping and controls. They concluded that the reduced diaphragm function in COPD is due to mechanical disadvantage secondary to lung hyperinflation, and not due to physiologic alteration of contractility. Ogan et al. [32] reported that the DT-TLC and DT-RV were normal in COPD patients due to the adaptation of the diaphragm because of excessive work against increased mechanical load. While Okura et al. [39] revealed that DT-TLC was decreased in COPD patients than healthy individuals, and there was no difference in DT-FRC and DT-RV between the 3 groups in their study conducted on COPD patients, young healthy, and elderly healthy male individuals. Jain et al. [34] found that the DT-TLC and DT-FRC were decreased in mild and moderate COPD, and DT-FRC was increased in patients with severe COPD. While the reason for the increase in DTs could not be fully explained, it was stated that it might be due to the development of some adaptations such as collagen deposition in severe obstruction.

Regarding DTs in asthma, similar result was reported by Fouda et al. [5] who showed significant decrease in DT in asthmatic children than controls. However, Tenório et al. [10] assess DTs among obese youths, youth with mild persistent asthma, and healthy non-obese youths, they reported that the obese youths had higher DT-FRC than either asthmatics or healthy youths. On the other hand, no difference was found in DTF% among obese, asthmatic, and healthy youths.

In the current study the DTF % cutoff <24.5% have fair sensitivity (74%), specificity (62.4%), PPV (67.3%), and NPV (69.3%) for discrimination between normal diaphragm function and DD in asthmatic patients. Additionally, at cutoff <23.7%, it have good sensitivity (85%), specificity (66.3%), PPV(73.2%) and NPV (79.3%) for discrimination between normal diaphragm function and DD in COPD patients. Sarwal et al. [35] in their study, used cut-off value <0.2 of DT, to define diaphragm atrophy in COPD. A higher cut-off point was used by Essawy et al. [2] study to differentiate between COPD groups and control group as DTF 0.927 with 75% sensitivity, 80% specificity, 93.7% PPV and 44.4% NPV. All these studies indicate that the cutoff point for discrimination of DD among either asthmatics or COPD patients is not determined yet.

The DD is highly prevalent among our COPD patients (37%) than asthmatic patients (25%) which might be attributed to the presence of different mechanisms in COPD does not present in asthma. In COPD, systemic inflammation, prolonged use of systemic cortico-steroids, and lung hyperinflation may serve as synergetic mechanisms for development of DD. Additionally, the early closure of small airways with air trapping resulting in intrinsic positive end-expiratory pressure that act as an extra load that the respiratory muscles must overcome before creating inspiratory flow which may lead to DD [36]. Muscle dysfunction is very prevalent among COPD patients [37], and appears to be caused by multiple factors,

although lung hyperinflation appears to be the main contributor for deterioration of respiratory muscle [38]. DD is relatively common in 20-45% stable COPD patients, reaching 80-90% in patients with repeated hospital admissions due to frequent exacerbations [39]. Cao et al. [3] reported that DD occurs early in COPD course and may be present in all GOLD stages. Although positive adaptive changes in diaphragm structure could be identified in COPD patients, negative structural changes still dominate in COPD patients [40].

Our study demonstrated the DTF% was positively correlated with FEF25-75% in asthma group, and with FEV_1%, FVC% in COPD group. Moreover, the reduced FEV_1% and FVC% were predictive factor of DD in asthma and FEV_1% was predictive factor of DD in COPD. These findings can be explained by that with increasing asthma and COPD severity, air trapping occurs that put the diaphragm at mechanical difficulties and negatively impacts its contractility. Similarly, Elsawy [22] reported that the DTF% bilaterally were positively correlated with FEV_1% and FEV_1/FVC ratio. Moreover, the FEV_1%, FVC%, and FEF25-75% were significant predictive factors impacting DT in COPD patients. Topcuoğlu et al. [18] reported that in COPD patients, the FEV_1% and FVC%, were correlated with DTF% bilaterally. Previous studies did not identify significant association between DTF% and FEV_1 [32] [41]. Another study reported significant positive correlation between DT and FEV_1% in mild COPD patients, but not in moderate and severe COPD patients [42].

Our study showed that although the BMI of the 3 studied groups belong to overweight category, and it was not correlated with DTF%. However, it is a predictive factor of DD in COPD patients. These findings indicate that obesity could negatively impact diaphragm function in COPD and asthma patients as the presence of obesity affects the diaphragm mechanics due reduction of diaphragm movement by increased abdominal pressure and fat. Hellebrandová et al. [1] reported that there was reduced efficacy of the diaphragm function due to an increased abdominal pressure and reduced abdominal compliance. Moreover, the diaphragm may be overstretched in patients with abdominal obesity, thereby placing it at mechanical difficulty. On the other hand, Cao et al. [3] reported that consumption of fat and protein causes muscle wasting and reduce which decreases muscle strength and endurance. In COPD, similar results had been reported by Cimsit et al. 42] and Eryüksel et al. [41] as DT was not correlated to BMI. Previous studies reported dissimilar result as the DTF% were related to BMI [17] [22] [43]. In asthma, Tenório et al. [10] reported that obesity led to increased DT-FRC and the DTF% was positively correlated with fat mass and body fat percentage in both asthmatic and obese youth. From all of these studies including the current one we can say that this double sword-edge of nutritional status on diaphragm function is still not well clarified.

The present study shows that the DTF% was inversely correlated with smoking index in asthmatics, and it is a predictive factor of DD in COPD patients. These findings indicate that smoking negatively affects diaphragm contractility in patients with either COPD or asthma. Similarly, another study reported that DT was related to smoking [44]. Elsawy [22] reported that in COPD patients the DT-RV bilaterally were negatively correlated with the smoking index, also the left DT-TLC was inversely correlated with the smoking index. Therefore, we can encourage patients to quit smoking as this simple measure can improve diaphragm contractility and it will prevent deleterious effects of tobacco on muscle function and structure.

Our asthmatic patients have significantly longer disease duration than COPD patients. The DTF% was negatively correlated with disease duration in asthmatics. Additionally, longer disease duration is a predictive factor of DD in patients with either asthma or COPD. These findings indicate that the diaphragm function might be influenced by asthma and COPD duration as longer disease duration is always associated with more advanced airway pathology and hyperinflation that put diaphragm at mechanical disadvantage and impair its function.

Our study encountered some limitations, firstly, this study did not matched baseline characteristics between asthmatics and COPD patients e.g., sex, smoking index, disease duration and severity which are confounders influences DT. Secondly, it was a hospital- based study, performed at a single centre, thus, the studied patients might not be representative of all asthmatic or COPD patients.

CONCLUSION

Diaphragm dysfunction is prevalent in COPD and asthmatics patients; accordingly, evaluation of diaphragm function is mandatory for early staring of pulmonary rehabilitation program / diaphragmatic training programs to improve diaphragm function, especially in patients with more advanced COPD or asthma who are most likely to have hyperinflation with subsequent DD. We recommend adding US evaluation of diaphragm function to a routine assessment and pulmonary function test in patients with either COPD or asthma would be of great clinical relevance to give additional contributions to determining the progress of the disease. Future studies are needed to determine the best cutoff point of DTF% for discrimination between normal diaphragm function and DD in patients with either COPD or asthma.

Fund: This work was not funded from any governmental or non-governmental agencies.

Conflict of interest declaration: The authors declared that there is no direct or indirect conflict of interest**.**

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الملخص العربي

تقييم وظيفه الحجاب الحاجز بالموجات فوق الصوتية في مرضى الضيق الشعبي الهوائي المزمن ومرضى الربو الشعبي: دراسة مقارنة

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ملخص البحث

الخلفية: يؤدي مرض ضيق الشعب الهوائية المزمن و الربو الشعبي إلى قلة تدفق الهواء، والذي يعتقد أنه يغير من موضع وشكل الحجاب الحاجز بسبب زيادة حجم الرئة مع وجود ضعف في انقباض الحجاب الحاجز.

الهدف: تقييم ومقارنة سمك الحجاب الحاجز باستخدام الموجات فوق الصوتية في مرضى ضيق الشعب الهوائية المزمن ومرضي الربو الشعبي.

الطرق: اشتملت دراسة الحالات والشواهد هذه 100 مريضا بالربو الشعبي و 100 مريضا بمرض ضيق الشعب الهوائية المزمن و 100 شخصا سليما. خضع جميع المشاركين لقياس وظائف التنفس وقياس سمك الحجاب الحاجز باستخدام الموجات فوق الصوتية

النتائج: كان سمك الحجاب الحاجز عند الحجم المتبقي أقل احصائيا في مجموعة مرضي ضيق الشعب الهوائية المزمن مقارنة بمجموعة الربو الشعبي و المجموعة الضابطة. كان سمك الحجاب عند سعة الرئة الكلية و النسبة المئوية لسمك الحجاب الحاجز أقل احصائيا في مجموعة مرضي ضيق الشعب الهوائية المزمن مقارنة بمجموعة الربو الشعبي والمجموعة الضابطة، وفي مجموعة الربو الشعبي مقارنة بالمجموعة الضابطة. في مجموعة الربو الشعبي، تناسبت النسبة المئوية لسمك الحجاب الحاجز سلبيا مع مؤشر التدخين و مدة المرض، و تناسبت إيجابيا مع تدفق الزفير القسري 25- 75%، بينما في مجموعة مرضي ضيق الشعب الهوائية المزمن تناسبت إيجابيا مع شدة المرض (حجم الزفير القسري في الثانية الأولى %) و النسبة ما بين السعة الحيوية القسرية / حجم الزفير القسري في الثانية الأولى %. في مجموعة الربو الشعبي؛ تسطيع القيمة الحدية (24.5٪) للنسبة المئوية لسمك الحجاب الحاجز التميز بين وظيفة الحجاب الحاجز الطبيعية خلل وظيفة الحجاب الحاجز بحساسية (74٪)، وخصوصية (62.4٪)، و قيمة تنبؤية ايجابية (67.3٪) و قيمة تنبؤية سالبة (69.3٪). في مجموعة مرضي ضيق الشعب الهوائية المزمن؛ تسطيع القيمة الحدية (23.7٪) للنسبة المئوية لسمك الحجاب الحاجز التميز بين وظيفة الحجاب الحاجز الطبيعية وخلل وظيفة الحجاب الحاجز بحساسية (85٪) وخصوصية (66.3٪) و قيمة تنبؤية ايجابية (73.2٪) و قيمة تنبؤية سالبة (79.3 ينتشر خلل وظيفة الحجاب الحاجز في مرضى ضيق الشعب الهوائية المزمن (37٪) ومرضى الربو الشعبي (25٪). العوامل التنبؤية ذات الدلالة الاحصائية لخلل وظيفة الحجاب الحاجز في مرضى الربو الشعبي مرتبة تنازليا هي: السعة الحيوية القسرية، جرعات الكورتيكوستيرويدات المستنشقة، عمر المريض، حجم الزفير القسري في الثانية الأولى %، ومدة المرض. في مرضى الشعب الهوائية المزمن العوامل التنبؤية ذات الدلالة الإحصائية لخلل وظيفة الحجاب الحاجز مرتبة بطريقة تنازليا هي: مدة المرض، العمر، مؤشر التدخين، جرعات الكورتيكوستيرويدات المستنشقة، حجم الزفير القسري في الثانية الأولى %، ومؤشر كتلة الجسم.

الاستنتاجات: خلل وظيفة الحجاب الحاجز منتشر بين مرضى ضيق الشعب الهوائية المزمن ومرضي الرب والشعبي. انخفض سمك الحجاب الحاجز وانقباضه بشكل كبير في كلا من مرضى الربو الشعبي ومرضي ضيق الشعب الهوائية المزمن مقارنة بالأشخاص الأصحاء ، وفي مرضى ضيق الشعب الهوائية المزمن مقارنة مع مرضى الربو الشعبي.

الكلمات المفتاحية: الربو الشعبي، مرض ضيق الشعب الهوائية المزمن، سمك الحجاب الحاجز، النسبة المئوية لسمك الحجاب الحاجز، وظيفة الحجاب الحاجز، ضعف الحجاب الحاجز

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