Research Square

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Ultrasonographic Assessment of Diaphragmatic Function in Obstructive Sleep Apnea Patients

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Abstract

Background

Obstructive sleep apnea (OSA) is associated with altered respiratory mechanics, including diaphragmatic function. Ultrasonography provides a non-invasive tool to assess diaphragmatic structure and performance, which may correlate with OSA severity.

Objectives

To evaluate diaphragmatic function in OSA patients using ultrasonography and to investigate its relationship with OSA severity and other sleep-related parameters.

Methods

This case-control study was conducted at the Chest Medicine Department, Mansoura University Hospitals, Egypt, from July 2023 to July 2024. After ethical approval (MS.23.03.2352) and informed consent, 30 newly diagnosed OSA patients (AHI ≥ 5) and 30 non-OSA controls (AHI < 5) were included. Exclusion criteria included systemic organ failure, uncontrolled diabetes, chronic respiratory diseases, neuromuscular disorders, prior OSA treatment, and steroid use. Overnight, attended polysomnography (PSG) was performed following AASM guidelines. Diaphragm ultrasonography measured excursion, thickness at end-expiration and thickness at inspiration, and diaphragm thickening fraction (DTF).

Results

OSA patients had significantly higher BMI, comorbidities, and sleepiness scores compared to controls ($p < 0.05$). PSG confirmed mild (10%), moderate (33.3%), and severe (56.7%) OSA among patients. Diaphragmatic excursion was significantly lower in OSA patients, while diaphragm thickness and DTF were higher ($p < 0.05$). Diaphragm ultrasound parameters were independently associated with OSA presence. ROC analysis demonstrated excellent predictive performance for diaphragmatic excursion (AUC = 0.841) and inspiratory thickness (AUC = 0.874). AHI correlated inversely with excursion ($r=-0.39$) and positively with inspiratory ($r = 0.47$) and expiratory thickness ($r = 0.28$) as well as DTF ($r = 0.26$).

Conclusion

Diaphragmatic ultrasonography reveals significant structural and functional alterations in OSA patients and may used be as a reliable, non-invasive tool for predicting OSA presence and its severity.

Introduction

Obstructive sleep apnea (OSA) is a frequent sleep-disordered breathing state characterized by recurrent upper airway obstruction during sleep, causing intermittent hypoxia, sleep fragmentation, and fluctuations in intrathoracic pressure (1). The global prevalence of OSA accounts for about 9–38% in adults, with higher rates among males, individuals with obesity, and those with cardiovascular comorbidities (2).

While significant research has concentrated on upper airway obstruction, there has been less emphasis on the diaphragm's role in OSA pathogenesis. The diaphragm contributes significantly to ventilation, and its function can be altered in conditions affecting respiratory load, such as obesity, chronic hypoxia, and repetitive airway obstruction (3).

Currently, polysomnography (PSG) is still the best approach to diagnose OSA, providing objective measures of the apnea–hypopnea index (AHI), oxygen (O2) desaturation. However, PSG does not directly assess respiratory muscle function (4).

Ultrasonography offers a non-invasive, real-time method to evaluate diaphragmatic morphology and function, including diaphragm thickness, excursion, and thickening fraction (DTF) (5). Studies have demonstrated that diaphragmatic ultrasound correlates with respiratory performance in various pulmonary and neuromuscular conditions, yet its application in OSA remains limited. Preliminary research suggests that OSA may alter diaphragmatic excursion and thickness due to repetitive respiratory effort and negative intrathoracic pressure swings, but evidence is sparse and heterogeneous. Integrating diaphragmatic ultrasound with traditional PSG and clinical screening could enhance the understanding of OSA pathophysiology and potentially provide predictive markers for disease severity (6).

Despite the growing interest in respiratory muscle assessment, there is a notable gap in the literature concerning comprehensive evaluation of diaphragmatic mechanics in OSA patients using ultrasonography. Specifically, the correlation between diaphragm thickness, excursion, thickening fraction, and OSA severity indices such as AHI and oxygen desaturation has not been thoroughly investigated in adult populations. Understanding these associations may help detect cases at greater risk for respiratory complications, guide personalized therapeutic interventions and explore novel non-invasive monitoring strategies (7). The aim of the current study was to assess the effect of OSA on the ultrasonographic findings of the diaphragm and to evaluate the presence of diaphragmatic dysfunction among OSA patients and its relation to disease severity.

Patients and Methods

Study Settings, Duration, and Design

This case-control study was conducted at the Chest Medicine Department, Mansoura University Hospitals, Egypt, from July 2023 to July 2024, following approval from the Institutional Research Board of the Faculty of Medicine, Mansoura University.

Inclusion Criteria: Patients visiting the Sleep Disordered Breathing (SDB) Unit at the Chest Medicine Department were included in the study if they had the next criteria: Age more than 18 years.; accept to participate in the study.; Newly diagnosed OSA confirmed by PSG with AHI > five events/hour in OSA group; Patients with AHI < five events/hour on PSG in Control group.

Exclusion Criteria: Patients were ruled out if they had any of the next situations: Age less than 18 years; Systemic organ failure, including heart failure, stroke, renal failure, or liver failure; Uncontrolled diabetes mellitus; Diagnosed obesity hypoventilation syndrome (OHS) or other sleep disorders apart from OSA; Chronic respiratory diseases which include COPD, bronchial asthma, or restrictive lung disease; OSA patients already receiving therapy (CPAP, oral appliances, or upper airway surgery); Neuromuscular disorders or other conditions affecting diaphragmatic function (malnutrition, chest trauma, or immobility); Use of steroids or uncontrolled autoimmune diseases; Refuse to participate in the study.

Methods:

I. Clinical Assessment: A comprehensive history was obtained from all participants. Medical history was carefully reviewed, focusing on co-morbidities and current medications. Special attention was given to sleep-related complaints, including snoring, dry mouth upon awakening, morning headaches, choking episodes during sleep, insomnia, excessive daytime sleepiness, observed apnea, and nocturia. A thorough physical examination was conducted for all subjects. General assessment which included measurement of blood pressure, height, weight, and body mass index (BMI). Local chest examination was performed to identify or exclude underlying pulmonary conditions that could influence respiratory function or study outcomes.

II. Laboratory and Imaging Investigations: All participants underwent laboratory and imaging investigations to evaluate systemic and respiratory status. Laboratory testing included arterial blood gases (ABG), liver function tests (LFT), serum creatinine, urea and hemoglobin A1c (HbA1c). Chest X-ray was performed to exclude structural abnormalities of the lungs or diaphragm.

III. Scales and Questionnaires: To assess sleepiness and evaluate the risk of OSA, all participants completed validated questionnaires that were translated into Arabic and culturally adapted for comprehension and relevance. The questionnaires used were Epworth Sleepiness Scale (ESS), Berlin Questionnaire (BQ) and STOP-Bang Questionnaire.

IV. Polysomnography (PSG): All participants underwent an overnight, attended polysomnography (PSG) using the SOMNOscreen D plus system (SOMNOmedics, Germany, SN: 3038). The diagnosis of OSA was confirmed when the AHI was ≥ 5 events per hour of sleep, in accordance with ICSD-3 criteria. The severity of OSA was classified as mild (AHI 5–14 events/hour), moderate (AHI 15–29 events/hour), or severe (AHI ≥ 30 events/hour). Scoring of apneas and hypopneas was performed according to AASM 2014 guidelines, allowing differentiation between obstructive, central, and mixed events based on respiratory effort and airflow patterns (8).

V. Ultrasonography Evaluation of the Diaphragm: Diaphragmatic structure and function were evaluated in all participants using a Digital Ultrasonic Diagnostic Imaging System, model 2200 (SN: ML-05108116, Germany). Diaphragmatic motion was assessed by measuring diaphragmatic excursion and thickness,

following established protocols. Right hemidiaphragm ultrasonography was performed due to superior image quality and reproducibility provided by the hepatic acoustic window. This approach is commonly used in diaphragmatic ultrasound studies and is considered representative of diaphragmatic function in the absence of unilateral pathology.

For excursion measurement, a 2–5 MHz phased-array transducer was used with the patient in a semi-recumbent position via the subcostal approach, placing the probe between the mid-clavicular (MCL) and anterior axillary (AAL) lines below the right costal margin and directing it medially, cephalad, and dorsally. The liver served as an acoustic window to visualize the right hemidiaphragm, which appeared as a thin, curvilinear, hyperechoic structure moving with respiration. M-mode ultrasonography was applied to record diaphragmatic motion, and the amplitude of excursion was measured from the basal value to the point of maximum height of inspiration on the graphs as shown in figure (1).

Figure (1): Showed diaphragmatic excursion which measured from the baseline to the point of maximum height of inspiration

Measurement of diaphragm thickness was c using a high frequency linear probe (12–13 MHz). Right diaphragm was visualized near their zone of attachment where the diaphragm abuts to the rib cage (zone of apposition). The probe was angled perpendicular to the chest wall, placed between the AAL and MCL at the eighth or ninth intercostal spaces. The diaphragm was recognized as a three-layered structure, consisting of the diaphragmatic pleura and peritoneal fascia encircling the hypoechoic muscle layer as shown in figure(2).Thickness measurements were obtained at end-expiration (FRC) and end of inspiration (TLC) from frozen M-mode images. DTF: calculated as (the difference between thickness at end maximum inspiration & thickness at end normal expiration) divided by thickness at end normal expiration.

figure (2): The diaphragm was identified as a three-layered structure and measurement of diaphragmatic thickness

Sample size:

Sample size calculation was based on mean diaphragm thickness on right side at rest between cases with obstructive sleep apnea group versus control retrieved from previous research. Using G*power version 3.0.10 to calculate sample size based on effect size = 1.05, 2-tailed test, α error = 0.05 and power = 90.0% then total sample size will be 30 patients at least.

Statistical analysis

All data were tabulated in SPSS sheet version 27. Categorical data were expressed as number and percent. Continuous data were tested for normality of distribution using Shapiro Wilk test. Normally distributed data were expressed as Mean ± SD. Non- parametric data were expressed as median. Chi square test was used for the comparison of categorical data. Student t- test and One way analysis of

variance (ANOVA) test were used for the comparison of normally distributed data between either 2 groups or more than 2 groups. U test and Kruskal Wallis test were used for the comparison of non-parametric data between either 2 groups or more than 2 groups. Receiver operator characteristics test was used for assessment of the diagnostic accuracy. Spearman correlation test was used for assessment of the correlation between 2 variables. A p value less than 0.05 was considered of statistical significance at level of 95% confidence interval.

Results

The current study included 30 obstructive sleep apnea patients (OSA) and 30 matched apparently healthy participants as control group. Mean age of OSA patients was 47.5 ± 10 years and 66.7% of patients were males. About 36.7% of patients were hypertensive, 23.33% were diabetics with significantly higher frequencies than controls **(p = 0.037; 0.023)**. Mean weight of the included OSA patients was 101.8 ± 13.94 kg and mean BMI was 36.8 ± 5.29 kg/m^2. OSA patients had significantly higher mean values of weight and BMI as compared to controls **(p < 0.001 for each) (**Table 1**)**.

Table 1
Sociodemographic and baseline characteristics differences between OSA group and control group:

	Group A (OSA group) (n = 30)	**Group B (control group) (n = 30)**	**Test of significance**
Age (Years) Mean ± SD	47.5 ± 10	45.8 ± 11.6	t=.39; p = 0.12
Sex No. (%) - Male - Female	20 (66.7%) 10 (33.3%)	17 (56.7%) 13 (43.3%)	χ^2=0.633; P = 0.426
Comorbidities - Hypertension - Diabetes	11 (36.7%) 7 (23.3%)	4 (13.3%) 1 (3.3%)	χ^{2FET}=4.36; **P = 0.037** χ^{2FET}=5.19; **P = 0.023**
Weight (kg) Mean ± SD	101.8 ± 13.94	76.9 ± 10.32	t = 7.79; **p < 0.001**
Height (cm) Mean ± SD	166 ± 10.65	172.4 ± 8.88	t=-2.52; **p = 0.015**
Body mass index (kg/m^2) Mean ± SD	36.8 ± 5.29	25.2 ± 3.72	t = 9.82; **p < 0.001**

t: Student t- test, χ^2: Chi-Square test; χ^{2FET}: Fisher exact test.

The current study showed that OSA patients had significantly higher median values of STOP BANG score, Berlin Score and Epworth scale than controls **(p < 0.001 for each)**. Also, The AHI showed statistically significant median values among OSA patients compared controls **(p < 0.001) (**Table 2**)**.

Table 2
STOP BANG, Berlin, Epworth scale and AHI by PSG differences between OSA group and control group:

	Group A (OSA group) (n = 30) Median (IQRs)	**Group B (control group) (n = 30) Median (IQRs)**	**Test of significance**
STOP BANG	5.63 ± 1.65	1.7 ± 1.06	U = 24; **P < 0.001**
Berlin	4.5 ± 3.08	2.7 ± 0.75	U = 33; **P < 0.002**
Epworth scale	13.83 ± 4.04	3 ± 2.61	U = 32; **p < 0.001**
AHI	34.4 ± 2.7	3 ± 1.06	U = 31; **p < 0.001**

U= Mann Whitney U test; STOP BANG: Snoring- Tiredness- Observed apnea- Pressure- BMI- Age- Neck circumference- Gender ; AHI: Apnea–Hypopnea Index.

The results showed statistically significant differences between OSA, and control groups as regards diaphragm US findings as OSA patients were associated with a significant reduction in mean values of diaphragm excursion **(p < 0.001)** and significantly higher mean values of diaphragm thickness in inspiration **(p < 0.001)** and expiration **(p < 0.001)** and higher thickness fraction **(p = 0.03)** than controls **(**Table 3**)**.

Table 3
Diaphragm ultrasonographic findings differences between OSA group and control group:

	Group A (OSA group) (n = 30) Mean ± SD	**Group B (control group) (n = 30) Mean ± SD**	**Test of significance**
Diaphragm excursion (cm)	2.26 ± 0.49 cm	2.94 ± 0.47 cm	t = 5.42; **p < 0.001**
Diaphragm thickness Inspiration	0.309 ± 0.044 cm	0.249 ± 0.028 cm	t = 5.84; **p < 0.001**
Diaphragm thickness Expiration	0.215 ± 0.045 cm	0.173 ± 0.028 cm	t = 4.30; **p < 0.001**
Thickness fraction	47.70 ± 22.99	35.77 ± 13.89	t = 3.15; **P = 0.03**

t: Student t- test.

According to AHI results, 3 patients (10%) had mild OSA, 10 patients (33.3%) had moderate OSA and 17 patients had severe OSA (56.7%).However, these diaphragmatic US parameters did not show significant differences between OSA patients of different degrees **(Table 4)**. **Table 4. Diaphragm ultrasonographic findings between different OSA grades**:

	Mild (n = 3)	**Moderate (n = 10)**	**Severe (n = 17)**	**Test of significance**
Diaphragm excursion (cm) Mean ± SD	1.75 ± 0.21	2.32 ± 0.49	2.29 ± 0.49	F = 1.18 P = 0.321
Diaphragm thickness Inspiration Mean ± SD	0.260 ± 0.014	0.318 ± 0.044	0.309 ± 0.043	F = 1.54 P = 0.233
Diaphragm thickness Expiration Mean ± SD	0.185 ± 0.04	0.224 ± 0.05	0.214 ± 0.04	F = 0.619 P = 0.546
Thickness fraction Median (IQRs)	44 (19–69)	38 (19–69)	39(21–100)	KW = 0.02 P = 0.978

F: One Way ANOVA test, KW; Kruskal Wallis test.

The value of diaphragmatic US in prediction of OSA then was confirmed by multivariate binary logistic regression analysis and reported that decreased diaphragm excursion, increased diaphragm thickness and fraction were significant predictors of OSA **(**Table 5**)**.

Table 5
Predictor model of OSA based on diaphragm ultrasonographic findings:

	B	P value	Odds ratio (95% CI)
Diaphragm excursion (cm)	-4.14	**0.006**	0.016 (0.001–0.305)
Diaphragm thickness Inspiration	38.33	**< 0.0001**	Undefined
Diaphragm thickness Expiration	28.48	**0.001**	Undefined
Thickness fraction	0.363	**0.026**	1.44 (1.045–1.98)
Overall % predicted = 76.7%			

Binary logistic regression test.

Area under curve for diaphragmatic excursion is excellent (AUC = 0.841) with the best detected cutoff point is 2.75 yielding sensitivity (Sn) of 83.3% and specificity (Sp) 56.7%. Area under curve for Diaphragm thickness inspiration is excellent (AUC = 0.874) with the best detected cutoff point is 0.265 yielding Sn of 86.7% and Sp 76.7%. Area under curve for Diaphragm thickness expiration is good (AUC = 0.767) with the best detected cutoff point is 0.185 yielding Sn of 73.3% and Sp 70%. Area under curve for Thickness fraction is fair (AUC = 0.598) with the best detected cutoff point is 36.5 yielding Sn of 53.3% and Sp 56.7% (Table 6).

Table (6):Validity of ultrasound findings between OSA group and control group

	AUC (95%CI)	P value	CUT off point	Sensitivity %	Specificity %
Diaphragm excursion (cm)	0.841 (0.744–0.939)	< 0.001*	2.75	83.3	56.7
Diaphragm thickness Inspiration	0.874 (0.784–0.965)	< 0.001*	0.265	86.7	76.7
Diaphragm thickness Expiration	0.767 (0.642–0.893)	< 0.001*	0.185	73.3	70.0
Thickness fraction	0.598 (0.452–0.744)	0.193	36.5	53.3	56.7
AUC: Area under curve					

AHI correlated significantly and inversely with diaphragm excursion **(-0.39; p = 0.002)** and positively to diaphragmatic thickness in inspiration **(r: 0.47; p < 0.001)** and in expiration **(r: 0.28; p = 0.031)** and thickness fraction **(r: 0.26; p = 0.04).**Baseline and minimal saturation correlated positively with diaphragm excursion and inversely with diaphragmatic thickness in inspiration, in expiration and thickness fraction. Berlin score correlated inversely with diaphragm excursion and positively to diaphragmatic thickness in inspiration, expiration and thickness fraction **(**Table 7**)**.

Table 7
Correlation between ultrasound findings and demographic, clinical and anthropometric measurements of the studied cases:

		Diaphragm excursion	Diaphragm thickness inspiration	Diaphragm thickness expiration	Thickness fraction
Age	R	.312	− .155	− .010	− .124
	p value	.093	.414	.960	.515
Sex	R	0.16	-0.26	-0.16	-0.088
	p value	0.24	0.06	0.21	0.54
BMI	R	.129	− .156	− .104	.023
	p value	.498	.410	.585	.906
	p value	.060	.406	.324	.718
AHI	R	**-0.39**	**0.47**	**0.28**	**0.26**
	p value	**0.002**	**< 0.001**	**.031**	**0.04**
STOP.BANG	R	.119	.062	− .062	.058
	p value	.531	.744	.743	.761
BERLIN	R	**-0.46**	**0.53**	**0.39**	0.11
	p value	**< 0.001**	**< 0.001**	**< 0.001**	0.4
Baseline saturation	R	**0.28**	**-0.41**	**-0.29**	-0.24
	p value	**0.03**	**0.001**	**0.024**	**0.04**
Minimal saturation	R	**0.29**	**-0.39**	**-0.29**	**-0.21**
	p value	**0.02**	**0.002**	**0.024**	**0.04**

r: Spearman correlation coefficient, **statistically significant; categorical variables are dummy coded; BMI: Body mass index; AHI: Apnea–Hypopnea Index.

Discussion

To achieve this aim, the current study was conducted on 30 OSA patients and 30 non- OSA participants as healthy control. All participants underwent thorough history taking, clinical examination, BMI, in particular, and diaphragmatic ultrasound.

In this study, mean age of OSA cases was 47.5 ± 10 years with insignificant differences when compared to controls. In concordance with our study, *Wang et al.* in recent study compared 20 controls to 130 OSA patients did not find significant age differences between both groups (6). *Silva et al.* in another study included 321 patients did not report presence of significant differences between OSA patients and controls regarding age (9). Also, *Chien et al.* recorded that mean age of OSA patients was 44.3 ± 7.6 years with statistically insignificant differences as compared to controls (10).

The majority of OSA patients were males representing 66.7% of cases and control group was sex matched to OSA group. In contrast, *Silva et al.* recorded significant higher male predominance among OSA patients than controls **(p = 0.001)** (9). Molnár *et al.* demonstrated presence of significant sex differences between OSA and controls reporting higher male predominance among OSA patients **(p < 0.001)** (11).

The current study demonstrated that higher percentage of OSA patients were diabetics (p = 0.023) and hypertensive (p = 0.037). In contrast with the present study, a study by Wang et al, did not find significant differences between OSA and controls regarding frequency of hypertension (6). Silva et al. did not report presence of significant differences between OSA patients and controls regarding hypertension, ischemic heart disease and diabetes (9).

Our study illustrated that body weight and BMI were significantly higher among OSA group (101.8 ± 13.94 kg; 36.8 ± 5.29kg/m^2) than control group (76.9 ± 10.32kg; 25.2 ± 3.72 kg/m^2) **(p < 0.001; p < 0.001)**. Likewise, *Molnár et al.* demonstrated that BMI of OSA patients were 31 ± 2.3 kg/m^2 as compared to 23.1 ± 3.9 kg/m^2. in control group **(p < 0.001)** (11). Similarly, *Wang et al.* showed that OSA patients had significantly higher BMI than controls (27.7 ± 3.06 vs. 23.4 ± 3.07; **p < 0.001)**(6). In disagreement with the present study, *Chien et al.* did not detect significant differences in BMI between OSA and controls (10).

The present study showed that STOP BANG score was significantly increased among OSA patients compared to controls (5.63 ± 1.65 vs. 1.7 ± 1.06; p < 0.001). In agreement with the present study, Suliman et al. displayed that STOP- BANG points were significantly higher among OSA patients (5.03 ± 1.29) than non- OSA patients (3.5 ± 0.53) (p < 0.001) (12). Silva et al., in different research on 321 cases, reported that the STOP-BANG score was significantly higher among OSA cases than controls (9).

OSA patients had significantly higher Epworth scale than controls in the present study (13.83 ± 4.04 vs. 3 ± 2.6; p < 0.001). In agreement with the current results, Wang and colleagues displayed that OSA patients had significantly greater ESS points than controls (p = 0.04) (6). Also, Silva et al. and Dixit et al. found that ESS score of OSA patients were significantly higher than non- OSA patients (p = 0.04) (9&13).

The current study illustrated that OSA cases had significantly higher median value of Berlin score than controls ($p < 0.001$). In the same line, Thutell et al. demonstrated that OSA cases were associated with a significant increase in Berlin scale points compared to controls (14). Dixit et al. and Silver et al. also demonstrated similar findings and showed that Berlin questionnaire points were significantly higher among OSA than non- OSA patients ($p = 0.004$) (13&9).

This study included 30 OSA cases and 30 non-OSA subjects. In this study, diaphragmatic ultrasonographic findings exhibited significant differences both groups and these findings were confirmed in the multivariate analysis as all parameters showed statistical significance as predictors for OSA.

In this study, diaphragm excursion was significantly lower among OSA than non-OSA patients **(2.26 ± 0.49 vs. 2.94 ± 0.47; $p < 0.001$)**. However, *Wang et al.* did not find significant differences between OSA, and controls as regards diaphragm excursion (6).

The current examination reported that diaphragm thicknesses on inspiration and expiration showed significant increase in OSA patients (0.309 ± 0.044; 0.215 ± 0.045) than controls (0.249 ± 0.028; 0.173 ± 0.028) **($p < 0.001$; < 0.001)**. In concordance with the present study, **Wang et al.** reported higher diaphragm thickness in inspiration and expiration among OSA patients than controls **($p < 0.001$)** (6). Also, *Molnár et al.* illustrated that OSA cases had significantly thicker diaphragm on both sides than controls **($p = 0.02$)** (11). Comparable findings were recorded by *Pazarlı et al.* who found that diaphragmatic thickness in both expiration and inspiration on both sides was significantly raised among OSA patients than controls (15).

The present results showed that diaphragm thickening fraction was higher in OSA patients than controls (47.7 ± 26.99 vs. 35.77 ± 13.89; **$p = 0.03$)**. Thickening fraction reflects diaphragmatic contractility; however, increased values may represent compensatory hypertrophy rather than improved function. This interpretation is supported by studies reporting thicker diaphragms in OSA without proportional improvements in oxygenation (15). In contrast, Wang et al. reported reduced thickening fraction in OSA, attributing this to obesity-related fatty infiltration and demographic differences (6), highlighting the heterogeneity of diaphragmatic responses in OSA.

Notably, the measured values of diaphragmatic excursion, thickness at end-expiration and end-inspiration, and diaphragmatic thickening fraction in the present study were comparable to ranges reported in previous ultrasonographic studies of healthy individuals and patients with obstructive sleep apnea, supporting the external validity of our findings (6&11&15).

Diaphragmatic ultrasonographic findings did not show significant differences between OSA patients of different disease severity. This came in agreement with *Pazarlı et al.* study which did not detect showed in diaphragm thickness between cases with mild, moderate or severe OSA (15). In contrast with the present study, *Wang et al.* found that diaphragmatic thickness and thickening fraction were significantly

varied between cases with mild- to moderate OSA and severe OSA with more pronounced changes in severe OSA cases (6). .

The current study demonstrated that the diaphragm ultrasonographic findings had good accuracy in differentiation between OSA patients and controls. In agreement with the present study, Wang et al. in recent study showed that diaphragmatic thickness, thickening fraction but not, diaphragmatic excursion were significant predictors for OSA (6). Similarly, Pazarlı et al. reported diaphragmatic thickness and thickening fraction as significant predictors for OSA (15). The studies explored the role of diaphragmatic US in diagnosis of OSA are limited and none of the published studies explored the diagnostic accuracy of the diaphragmatic US parameters except that one previous study demonstrated that diaphragmatic thickness beside age, sex and BMI had 91% sensitivity and 81% specificity in diagnosis of OSA (11).

Assessment of the right hemidiaphragm is widely accepted as representative of global diaphragmatic function in individuals without unilateral diaphragmatic pathology, which was excluded in the present study. Previous studies have demonstrated strong symmetry between both hemidiaphragms in terms of thickness and excursion in healthy subjects and in patients with systemic respiratory disorders (6&11&15).

The current study showed significant inverse correlation between Berlin score and diaphragm excursion and positive correlation between Berlin score and diaphragm thickness in inspiration and expiration and thickness fraction in OSA patients. Likewise, *Wang et al.* and Pazarlı et al. demonstrated presence of significant positive correlations of diaphragm thickness to OSA disease severity as detected by Berlin score **(6&15)**.

The present results showed that diaphragm excursion correlated significantly and positively with oxygen saturation. Meanwhile, diaphragm thickness and thickness fraction correlated inversely with oxygen saturation. Our observation that greater diaphragm excursion is associated with higher oxygen saturation is consistent with the physiological role of excursion as a surrogate of inspiratory strength and tidal volume generation. Previous ultrasonographic studies in both healthy individuals and patients with chronic respiratory disorders have shown that reduced diaphragmatic mobility is associated with hypoxemia, impaired ventilatory efficiency, and reduced lung volumes (11). The inverse correlation between diaphragm thickness/thickness fraction and oxygen saturation aligns with studies suggesting maladaptive structural remodelling of the diaphragm in OSA. **Wang et al.** displayed that cases with severe OSA had significantly thicker diaphragms but lower thickening ratios, with nocturnal desaturation emerging as the main driver of these alterations(6). Similarly, **Pazarlı et al.** reported thicker diaphragms in OSA cases compared with controls, but no improvement in oxygenation, supporting the view that increased thickness may reflect hypertrophy or pseudohypertrophy rather than improved contractility (15).

Assessment of the right hemidiaphragm is widely accepted as representative of global diaphragmatic function in individuals without unilateral diaphragmatic pathology, which was excluded in the present study. Previous studies have demonstrated strong symmetry between both hemidiaphragms in terms of

thickness and excursion in healthy subjects and in patients with systemic respiratory disorders (6&11&15).

The study had some limitations. Small sample size could negatively affect the generalizability and reliability of the results. However, calculation of sample size based on the effect size of previous studies could overcome this limitation. One single-center study was another limitation.

In conclusion, OSA is accompanied by significant diaphragmatic structural and functional alterations detectable by ultrasonography. Diaphragm ultrasound represents a simple, non-invasive, and clinically valuable tool for identifying diaphragmatic involvement in OSA and shows meaningful correlations with disease burden and hypoxemia, supporting its role as an adjunctive assessment modality in OSA patients.

Declarations

Ethical Approval

All participants provided written informed consent. Confidentiality of data was ensured. The study was reviewed and approved by Institutional Research Board (IRB) at faculty of medicine Mansoura University, code number: MS.23.03.2352.

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Figures

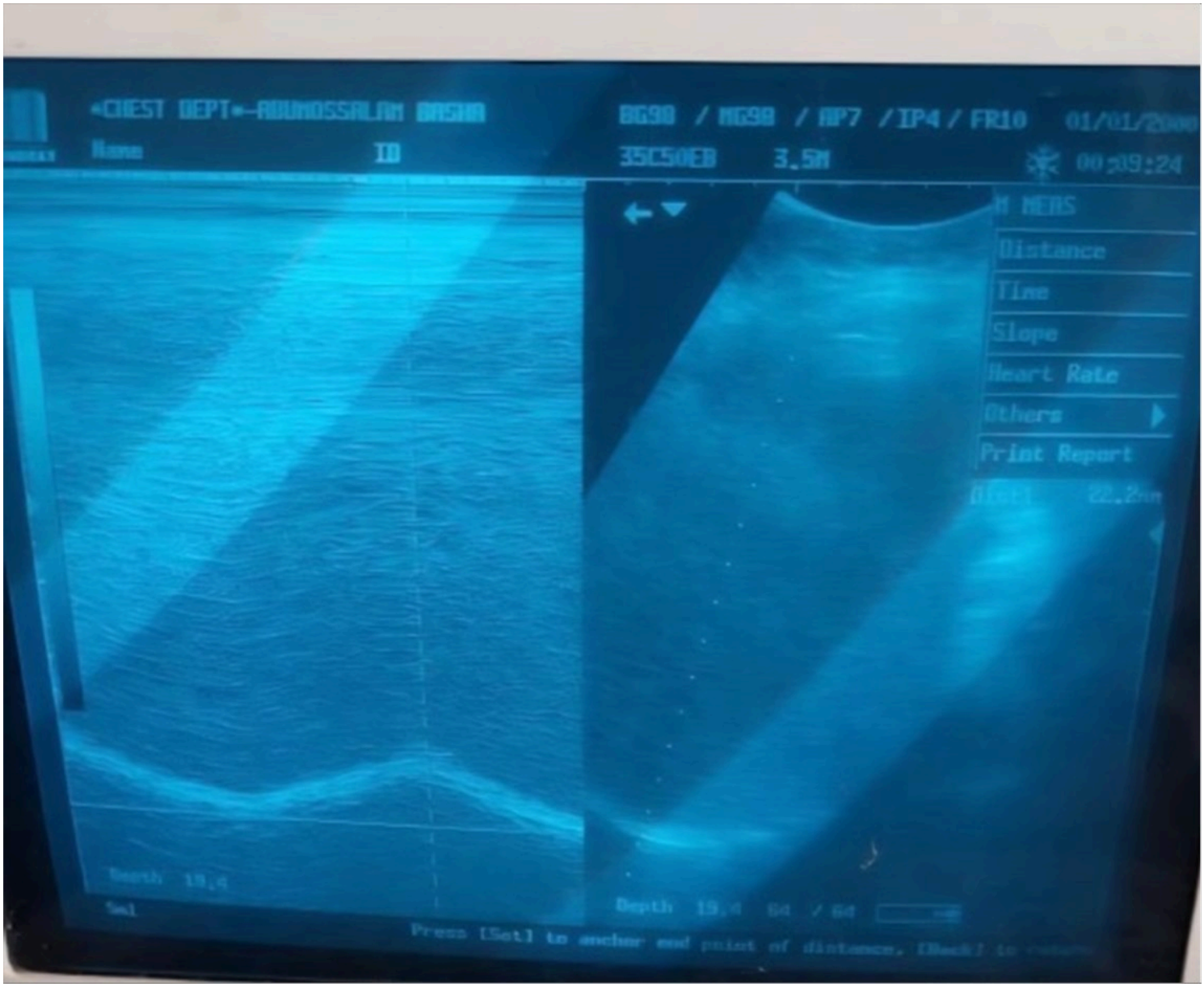


Figure 1

Showed diaphragmatic excursion which measured from the baseline to the point of maximum height of inspiration



Figure 2

The diaphragm was identified as a three-layered structure and measurement of diaphragmatic thickness