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Impact of chronic obstructive pulmonary disease severity on diaphragm muscle thickness

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ABSTRACT

Background: Sonographic assessment of the diaphragm muscle thickness for disease progression in chronic obstructive pulmonary disease (COPD) patients was poorly evaluated.

Aim: To assess the impact of COPD severity on diaphragm muscle thickness measured by transthoracic ultrasound (TUS).

Methods: Case-control study was conducted on 100 clinically stable male COPD patients and 100 age-matched healthy subjects. Thickness of the diaphragm (TD) was measured by B-mode TUS. Measurements were taken on three different images to find the average static TD at residual volume (RV) and at total lung capacity (TLC). A thickness fraction (TF) was derived by subtracting the average thickness of the diaphragm at RV from the average thickness of the diaphragm at TLC/average thickness of the diaphragm at RV [TD at TLC-TD at RV/TD at RV]. COPD severity was measured by spirometry. Moreover, partial pressure of oxygen (PaO₂) and partial pressure of carbon dioxide (PaCO₂) were measured.

Results: The right and left TF were significantly lower in COPD group than control group. All static right and left TD indices didn't show any significant difference between COPD group and control group ($p > 0.05$). The right and left TF were significantly decreased through progression of COPD (mild, moderate, severe and very-severe grades) ($p = 0.020, 0.002$ respectively). TD at RV bilaterally were negatively correlated with the smoking index, also the left TD at TLC was negatively correlated with the smoking index and forced vital capacity (FVC%). The right and left TF were positively correlated with body mass index (BMI), forced expiratory volume in first second (FEV₁%), FEV₁/FVC ratio, and PaO₂, while it was negatively correlated with PaCO₂.

Conclusion: The static TD measured at RV and at TLC are preserved in COPD. The TF bilaterally was significantly decreased in COPD group and is decreased with increasing severity of COPD. The negative correlation of TF and PaCO₂ indicates that the diaphragm dysfunction is the main contributor to neuromuscular respiratory failure in COPD patients.

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Introduction

Chronic obstructive pulmonary disease (COPD) is a major cause of chronic morbidity and mortality throughout the world [1]. Inspiratory muscle weakness in patients with COPD is of major clinical relevance [2]. Studies dealing with inspiratory muscle weakness in COPD patients focus mostly on diaphragm as it is the main generator of tidal volume [3]. In COPD respiratory muscles weakness is

caused by hyperinflation and/or by generalized muscle weakness as a result of deconditioning, malnutrition, electrolyte disturbances, cardiac failure, the long-term administration of steroids and systemic inflammation [4,5]. Additionally, in these patients, the diaphragm works against an increased work load due to airflow limitation and geometrical changes in the thorax as a result of pulmonary hyperinflation [6]. Despite sarcomere adaptation that occurs in chronic hyperinflation, the inspiratory muscle length reduced. When compensated for reduced inspiratory muscle length, diaphragm function was suggested to be preserved or even improved in severe COPD [7]. Additionally, in moderate COPD the diaphragm would show physiologic, compensatory overuse

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hypertrophy. Contradicting to this view, oxidative stress and sarcomeric injury in COPD activate proteolytic machinery leading to contractile protein wasting and consequently loss of force-generating capacity of the diaphragm. This accelerated protein degradation actually leads to diaphragmatic atrophy and not hypertrophy. Although, several of these presumed pathologic alterations are present in mild-to-moderate COPD, these patients do not have limitations of their daily-life activities [6].

Ultrasound evaluation of the diaphragm is simple, non-invasive, readily available at the bedside and increasingly used both in the clinical and research settings [7]. B-mode transthoracic ultrasound (TUS) can be used to assess the thickness of the diaphragm (TD) over a wide range of lung volumes from residual volume (RV) to total lung capacity (TLC) in the zone of apposition (ZOA), with high reproducibility [8]. The TD measurements can be made either at the end of a normal expiration (FRC), during a breath-holding maneuver after maximal inspiration (TLC) or at the end of maximal expiration (RV), and they were reported as TDFRC, TTDLC and TDRV, respectively [7]. Diaphragm thickening fraction (TF) has been newly proposed to be more sensitive indicator of diaphragm contraction than measurement of thickness since the increase in diaphragmatic thickness during inspiration is used as an indirect measurement of muscle fiber contraction which is analogous to ejection fraction of the heart [9]. TF requires the measurement of both end-expiratory and end-inspiratory diaphragm thicknesses and is computed as [(inspiratory thickness – expiratory thickness)/end-expiratory thickness], expressed as a percentage [10,11]. The lower limit of normal for TD at RV has been reported to be 0.15 cm in healthy subjects and in patients with COPD [12]. US findings of diaphragm muscle in COPD patients is not concluded yet because of the varying results [3,4]. Therefore, this study was conducted to assess the impact of COPD severity on diaphragm muscle thickness measured by ultrasound.

Subjects and methods

This study was conducted at chest diseases department, Al-Zahraa university hospital, Cairo, Egypt, in the period from January 2016 to December 2016.

Study participants

The study conducted on 100 male COPD patients, they were selected males to avoid the intersex variability on TD. All of them had spirometric criteria of irreversible/partially reversible airflow limitations [post-bronchodilator forced expiratory volume in first second / forced vital capacity ratio (FEV_1/FVC) <0.7 and an increase in FEV_1 of <200 ml or <12% of baseline value, 20 min after 4 puffs of inhaled Salbutamol (100 µg)]. They were classified according to $FEV_1\%$ as follow; 25 patients with mild COPD ($FEV_1 >80\%$), 25 patients with moderate ($FEV_1 60–80\%$), 25 patients with severe ($FEV_1 30–60\%$) and 25 patients with very severe ($FEV_1 <30\%$) [13]. They were recruited while attending chest outpatient clinic for regular follow up. All of them were clinically stable with no emergency visits, antibiotic use, increase medication, use of systemic steroid or theophylline or hospitalization in the previous 8 weeks. The control group includes 100 age and sex matched healthy subjects, all of them had no symptoms suggestive chronic chest diseases with normal spirometric-indices.

Patients with diaphragmatic injury, accident or surgery, stroke, muscular disorders, e.g. muscular dystrophy, multiple sclerosis, thyroid disorders, lupus, radiation therapy, infection, malnutrition, trauma to the muscle or nerve going through the chest to the muscle and other chest diseases affecting diaphragm were excluded from the study.

Ethical considerations

The study was approved by the ethical committee of A-Azhar University. All participant gave an informed written approval before enrollment into study. The data were coded to ensure confidentiality.

All subjects were subjected to the following:

- Age, smoking index (pack/year) [packs smoked per day × years of smoking] and body mass index (BMI) [weight (kg) / height (m²)] were recorded.
- 2. Spirometry

Spirometry was carried out on (Spirosift spirometry 5000 FUKUDA NENSHI, Japan). The following indices were recorded; $FEV_1\%$, forced vital capacity (FVC%), forced expiratory flow rate at 25–75% of vital capacity ($FEF_{25–75\%}$) and FEV_1/FVC ratio using best out of three technically satisfactory trials in accordance to the recommendations of the ATS [14].

Measurement of diaphragm thickness

- A. The diaphragmatic thickness was examined using Sonoscape A8 Medical Systems (Shenzhen, China). To abolish the caudal drag of gravity on diaphragm movement the patients were examined in the semi-recumbent position. A high frequency linear transducer (11 MHz) was placed on the chest wall at approximately the anterior axillary line, just cephalad to the lower costal margin. At the ZOA (observed optimally 0.5–2 cm below the costophrenic sinus), the diaphragm was visualized between the anterior and the midaxillary lines as a hypoechoic layer of muscle encased in two hyper-echoic layers of connective tissue (the parietal pleura and the peritoneum), deep to the intercostal muscles connecting the two ribs [15]. TD was measured just inside the hyperechoic connective tissue layers (Fig. 1). Measurements were taken on three different images to find the average static TD at RV and at TLC. The measurements were taken at RV and TLC as in clinical setting they were more reproducible than FRC. The TF was derived by subtracting the average TD at RV from the average TD at TLC /average TD at RV [TD at TLC-TD at RV/TD at RV] [16].
- B. Assessment of intra-rater agreement was done by statistical analysis of the intra-rater reliability of measurements. US imaging was done twice at the same anatomical position, 7 days apart for 20 participants as a pilot testing. Intra-rater reliability test was done by calculating Intra-class Correlation Coefficient (ICC) using a one-way random effect model and evaluation of absolute agreement. Confidence intervals were calculated at 95% confidence level for reliability coefficients. The estimated ICC = 0.85, with 95% CI (0.73–0.92). We had an evidence of good intra-rater agreement of measurements between scans by using this technique, therefore, it was used for all study participants. The measurements of pilot test were not included in data analysis.

Arterial blood gas (ABG) analysis

The PaO_2 mmHg and $PaCO_2$ mmHg were measured after 15 min resting period in ambient room air, by using blood gas analyzer (Rapid Lab 248, Siemens medical Solutions, Malvern, PA, US).

Statistical analysis

Data were statistically analyzed by SPSS version 17 for windows. Data were expressed as mean ± SD for quantitative variables

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