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Original Article

Correlation between diaphragm thickness and respiratory synergist muscle activity according to severity of chronic obstructive pulmonary disease

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Abstract. [Purpose] This study aims to analyze the effect that moderate to severe chronic obstructive pulmonary disease (COPD) has on the respiratory synergist muscles. The results will provide basic data that can be used in the clinical management of COPD. [Subjects and Methods] The subjects in the study were 47 male patients with COPD between 55 and 70 years old who were treated in a medical institution located in Jeollanam-do Province, South Korea, from October 2015 to December 2016. Measurements were analyzed to determine the correlation between the diaphragm thickness and the respiratory synergist muscle activity in patients with mild COPD. [Results] The results showed that there was a negative correlation between the diaphragm thickness and the sternocleidomastoid muscle and between the diaphragm thickness and the scalene muscle; however, there was a positive correlation between the diaphragm thickness and the external intercostal. For patients with severe COPD, negative correlations were found between the diaphragm thickness and the sternocleidomastoid muscle and between the diaphragm thickness and the scalene muscle. [Conclusion] The mechanical deformation of the thoracic cage caused by severe COPD reduces the lung capacity of patients and, thus, increases the difficulty in breathing. As the disease worsens, the patients tend to maintain ventilation using the respiratory synergist muscles. Thus, offering early and aggressive treatment and a respiration rehabilitation program to patients with COPD can help to reduce the actions of the respiratory synergist muscles to ensure normal breathing.

Key words: Chronic obstructive pulmonary disease, Diaphragm, Respiratory synergist muscles

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INTRODUCTION

Chronic obstructive pulmonary disease (COPD) increases the dynamic hyperinflation of the lung volume due to chronic airflow limitation. To compensate for the increase, there is an increase in pressure produced by the respiratory muscles of the thoracic cage, which makes breathing more difficult[1)]. In addition, pulmonary hyperinflation augments respiratory activities and reduces muscular receptivity in order to overcome the burden of ventilation[2)].

Patients with COPD have difficulty inhaling as the diaphragm, the agonistic muscle of respiration, becomes short and flat[3)]. This weakened respiratory function is compensated by the increased activities of the respiratory synergist muscles. Patients with COPD will also change their posture to find more comfortable ways of breathing by transforming the functions of the involuntary muscles[4)].

Patients with COPD have difficulty breathing because their breathing capacity is reduced. This is because the energy

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Table 1. General characteristics of subjects

Items	Experimental group-I (n=15)	Experimental group-II (n=15)
Age (years)	65.6 ± 4.1	66 ± 3.8
Height (cm)	164.5 ± 4.2	163.9 ± 3.6
Weight (kg)	64.5 ± 4.9	61 ± 2.1
FEV1 (%)	61.8 ± 2.9	41.2 ± 5.1
FEV1/FVC (kg/m^2)	51.9 ± 3.1	31.5 ± 4.8

Data are presented as mean ± SD, obtained by using the Shapiro-Wilk test.
FVC: Forced Vital Capacity; FEV1: forced expired volume in one second.

supplied by the diaphragm is not consistent with the respiratory demands of the patient[5]. As the disease worsens, the muscle fibers in the chest wall and the respiratory synergist muscles transform to adapt to an increased capacity in order to maintain breath strength and to increase endurance[6].

In patients with mild COPD, it is important to manage the abnormal mobilization of the respiratory muscles and oxygen demand, from the moment of detection even though the patients may not feel uncomfortable and have any symptoms. However, it is difficult to diagnose aggravated pulmonary function before the symptoms appear, and most patients show symptoms when they are moderately ill. It is also unclear when the abnormality of the respiratory muscles begins. Therefore, it is important to identify the relationship between the thickness of the diaphragm, and the accessory respiratory muscles, such as the sternocleidomastoid muscle, the scalene muscle, and the external intercostal muscle.

COPD is classified according to the degree of airflow obstruction; thus, airflow obstruction is as an important factor in predicting, diagnosing, and classifying the prognosis of COPD[7]. Based on previous studies, we diagnosed patients with COPD based on the forced expired volume in 1 second, dividing the subjects into mild or severe groups as defined by the GOLD severity classification[8].

As COPD progresses, it can lead to respiratory failure due to the degenerative changes that take place in the ventilation mechanism caused by the overuse of the respiratory synergist muscles; respiratory failure can have negative influences on other body functions[9]. In this study, we aimed to analyze the effect of moderate to severe COPD on the respiratory synergistic muscles to provide basic data that can be used for the clinical management of COPD.

SUBJECTS AND METHODS

The subjects of this study were 47 male patients with COPD aged 55 to 70 years old (approval No. SH-IRB 2017-05). They were treated in a medical institution in Jeollanam-do Province, South Korea, from October 2015 to December 2016. The subjects did not have a history of major medical conditions, including ischemic heart disease, intermittent claudication, and other complications determined to be impractical in performing this program, concomitant injuries, such as congenital deformity in the thoracic cage or a rib fracture, and radical symptoms of COPD. In addition, the subjects understood the purpose of this study and agreed to participate (Table 1).

Pulmonary functions were measured with a Chestgraph HI-701 (Chest M.I., Inc., Japan). While sitting, the subjects placed the respiratory apparatus in their mouths and inhaled and exhaled as much as possible. The pulmometry was performed at least three times; the measurement was recorded when the difference between the two largest values was within 5% or 200 ml.

Ultrasonic measurements were performed using a B-mode M12L high-frequency linear transducer (5.0–14.0 MHz) (Logiq 7; GE Healthcare, USA). When the subjects stood upright, the mid-axillary line between the eighth and ninth ribs was identified. When they sat at 90 degrees, the linear transducer was used to scan the chest wall at a right angle to obtain 2D images of the area between the eighth and ninth ribs. The thickness of the diaphragm was the distance between the two parallel lines that appeared in the middle of the pleura and in the middle of the peritoneum. The measurements were performed three times to calculate the average value[10].

The activities of the respiratory muscles were measured with a surface EMG MP100 system with four channels (Biopac Systems, Inc., USA). The sampling rate for the EMG signal collection was set at 1,000, and the frequency band filter was set at 20–450 Hz. To minimize the skin resistance to the EMG signals, we removed the hair and dead cells from the subjects' skin using fine sandpaper and cleaned the skin using alcohol cotton. Two Ag/AgCl surface electrodes were attached to the belly of each muscle at 2 cm intervals parallel to the muscle fibers. The reference electrodes were attached to the spine of the scapula and the spinous process of the twelfth spine. The EMG signals were collected from the right sternocleidomastoid muscle, the scalene muscle, and the external intercostal muscle. The EMG signals from the muscles were converted into the root mean square (RMS). The EMG signals were analyzed with the Acqknowledge 3.9.1 software program (Biopac Systems, Inc., USA). In order to normalize the EMG signals of the subjects, the subjects were asked to sit comfortably and to breathe naturally ten times. The average RMS value was determined from eight breaths; the initial and the final breaths were excluded during the reference action. Using a Threshold Inspiratory Muscle Trainer (NJ, USA), we quantified the RMS values when

Table 2. Correlation between groups on diaphragm thickness and the respiratory synergist muscle activity

Experimental group I	Dia-Th	SCM	Scale-A	Ex-costal	Experimental group II	Dia-Th	SCM	Scale-A	Ex-costal
Dia-Th	1				Dia-Th	1			
SCM	–0.71*	1			SCM	–0.79*	1		
Scale-A	–0.65*	0.48	1		Scale-A	–0.71*	0.51	1	
Ex-costal	0.63*	–0.51	–0.48	1	Ex-costal	0.54	–0.54	–0.51	1

*p<0.05.
Data are presented as mean ± SD, Pearson correlation.
Dia-Th: Diaphragm Thickness; SCM: sternocleidomastoid muscle; Scale-A: Anterior Scalene muscle; Ex-costal: External intercostal muscle.

the subjects breathed at a level of 30% of the maximum inspiratory pressure[11].

For data analysis, we used SPSS 18.0 for Windows (IBM Corporation, USA) to provide the descriptive statistics about the general characteristics of the subjects, the pulmonary functions, the diaphragm thickness, and the average activities of the respiratory synergist muscles. We used the Pearson's correlation analysis to identify the correlations among the pulmonary functions, the diaphragm thickness, and the activities of the respiratory synergist muscles. The significance level was set at α=0.05.

RESULTS

The correlation between the diaphragm thickness and the respiratory synergist muscle activity in patients with mild COPD was determined. There were negative correlations between the diaphragm thickness and the sternocleidomastoid muscle (r=–0.71, p<0.05) and the scalene muscle (r=0.68, p<0.05). However, there was a positive correlation between the diaphragm thickness and the external intercostal muscle (r=0.65, p<0.05). For patients with severe COPD, negative correlations were found between the diaphragm thickness and the sternocleidomastoid muscle (r=–0.72, p<0.05) and the scalene muscle (r=–0.71, p<0.05) (Table 2).

DISCUSSION

The diaphragm in patients with COPD tends to be shortened by 40% due to pulmonary hyperinflation[12]. This results in a shortened apposition zone, which leads to ineffective contractions because of reduced piston-like motions of the diaphragm. In addition, pulmonary hyperinflation increases the use of the accessory respiratory muscles. The respiratory muscle movements might be reduced by an excessive overlap of actin–myosin filaments[13].

According to Calverley et al.[3], hyperinflation causes the diaphragm to not function properly because of the shortened contractile fibers. These abnormalities in the diaphragm lead to difficulties with inhalation, which can be alleviated using the accessory respiratory synergist muscles. Loring et al.[2] reported that the abnormal shape of the diaphragm is associated with low exercise tolerance and low functional capacity.

Orozco-Levi[9] found that as COPD worsens, the rapidly elevated oxygen demand can no longer be met through the excessive use of the respiratory synergist muscles, and that respiratory failure occurs because of deformities in the ventilatory mechanism. In this study, the thickness of the diaphragm in both patients with mild and severe cases of COPD had a negative correlation with the sternocleidomastoid muscles and the scalene muscles, which means that as the value of one variable increased, the other variable decreased. These results suggest that the importance of the respiratory synergist muscles increases as the diaphragm becomes weaker. Shah and Herth[14] reported that, when the movements of the diaphragm are limited due to the abnormal breathing caused by the deterioration of the pulmonary function, the respiratory synergist muscles, such as sternocleidomastoid muscles and the scalene muscle, are used to lift the thoracic cage upward to compensate for the limitations. These results support the results of this study.

According to Kisner and Colby[15], as the severity of COPD increases, the activities of the diaphragm decrease and the involuntary muscles are transformed to facilitate breathing. In our study, the activity of the external intercostal muscles was more significant in patients with severe COPD than in patients with mild COPD, which indicates that an abnormal respiratory system was mobilized through the increased actions of the respiratory synergist muscles rather than the diaphragm. Mercadier et al.[16] reported that an increase in the respiratory rate in patients with COPD was adjusted by changes in the fiber type of the diaphragm; there was a consequent increase in the proportion of type I muscle fibers, which are associated with fatigue resistance.

Levine et al.[17] reported that the intercostal muscle fibers changed from type II to type I in patients with severe COPD. This change was a response to the activities of the other respiratory synergist muscles. These observations may indicate that COPD causes muscle deformities, a major cause of the differential adaptation of the peripheral and the respiratory muscles, and that the respiratory agonistic muscles are concomitantly deformed as the COPD becomes severe.

According to Mariana et al.[1], patients with severe COPD have more difficulty breathing due to their reduced lung capacity

because of the mechanically deformed thoracic cage. In these cases, the respiratory synergist muscles play a role in breathing as the disease becomes severe. Thus, early and aggressive treatment and respiration rehabilitation programs should be provided to patients with COPD to reduce the actions of the respiratory synergist muscles and to ensure that the patients are able to breathe normally.

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