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ORIGINAL ARTICLE

Voice changes in patients with chronic obstructive pulmonary disease



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KEYWORDS

Chronic obstructive pulmonary disease;
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Abstract *Introduction:* Voice changes are not a direct symptom of chronic obstructive pulmonary disease (COPD), but many COPD patients experience voice changes.

Aim of the work: The aim of this work was to establish the voice changes in patients with chronic obstructive pulmonary disease.

Patients and methods: Fifty COPD patients were conducted in this study. Patients were enrolled after obtaining informed consents. All patients were subjected to clinical diagnostic aids which include history taking (age, sex, smoking index and drug history), general and chest examinations, spirometry, arterial blood gases, chest X-ray, endoscopic examination of the larynx, auditory perceptual assessment and acoustic analysis of voice.

Results: The age of the patients ranged from 32 to 76 years, all patients were current or former smokers and the pack year index ranged from 20 to 66 with a mean \pm SD value of 41.16 ± 13.80 . Dysphonia was perceived in 25 (50%) patients. There was significant positive correlation between the smoking index with Jitter%, Shimmer% and the grade of dysphonia. There was significant positive correlation between Jitter%, Shimmer% and the grade of dysphonia with the large doses of ICSs usage and with pMDIs usage. Moreover, there was significant inverse correlation between Jitter%, Shimmer% and the grade of dysphonia with DPIs usage and with FVC, FEV1 and MMEF% of predicted values.

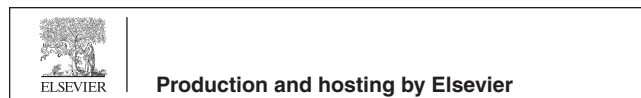
In conclusion: Dysphonia (hoarseness) in COPD patients is multifactorial. Successful analysis should depend on cooperation between pulmonologists, voice specialists, and laryngologists.

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Introduction

Chronic obstructive pulmonary disease (COPD) impacts life in many ways. Frequent wheezing and coughing, trouble breathing, coughing up mucus and shortness of breath are just a few COPD symptoms. According to the American Speech-Language-Hearing Association, these and other COPD symptoms can cause havoc on the throat and vocal cords, causing

problems like voice, communication and swallowing disorders. Voice changes are not a direct symptom of COPD, but many COPD patients experience voice changes due to COPD symptoms and even certain COPD medications [1].

Hoarseness is an abnormal deep, harsh voice. It can be described as raspy, breathy, soft, tremulous and even croaky or frog-like. Hoarseness may cause pain or a strained feeling when trying to speak normally. A hoarse voice can be caused by anything that interferes with the normal vibration of the vocal cords, such as swelling or inflammation [2].

The most common cause of hoarseness in an individual without COPD is acute laryngitis caused by an upper respiratory tract infection. Less common causes for those without COPD are misuse of the voice (such as from yelling or improper singing technique) and cold or flu [3].

For those with COPD, hoarseness may be caused by cold, flu or a COPD spell, but it may also result from certain COPD medications. Long-term use of inhaled corticosteroids, a category of inhalers used for COPD, and anti-cholinergics is known to cause hoarseness. Hoarseness is also associated with smoking tobacco [2].

The initiation of sound and voice begins with inhalation and exhalation. Thoracic and pulmonary disorders may serve to limit vital capacity, which in turn will limit breath support and control necessary for efficient speaking [3,4].

Appropriate prevention of controllable diseases such as COPD through early smoking cessation and early intervention for patients that develop these diseases will play a role in maintaining vocal strength and efficiency [5–7].

Dysphonia has been reported in 5–50% of patients using inhaled steroids. The wide range in this prevalence is a reflection of the means by which these data are calculated (i.e., as a coincidental finding in many studies that have ultimately set out to investigate a different, although associated, problem). It is also interesting that many studies use the terms dysphonia and hoarseness as different phenomena when, in fact, the difference is very subtle. Furthermore, it is apparent that dysphonia (or hoarseness) usually has been assessed only by questionnaires rather than by any clinical measurement [7–9].

A dose-dependent hoarseness has been reported in 34% of patients treated with beclomethasone dipropionate (BDP) or budesonide (BUD) when both inhaled corticosteroids (ICSs) were administered via pressurized metered-dose inhalers (pMDIs) [10]. Other studies have reported an increased risk of hoarseness with the use of fluticasone propionate compared to BDP, and with pMDIs compared to dry powder inhalers (DPIs). It has been suggested that the etiology of dysphonia in some cases is due to a steroid myopathy affecting the vocal cord muscles. A closer examination using flexible laryngoscopy and videostroboscopy reveals varying degrees of myopathy in symptomatic patients. This problem can, however, be reversed when therapy with the inhaled steroid is stopped [11–14]. In contrast, Shaw and Edmunds [15] found that dysphonia not to be a problem when using regular inhaled BDP 100–1500 µg per day although no objective measure of dysphonia was used in this group. A comparable incidence of hoarseness was established in healthy control subjects and patients receiving long-term BUD therapy via turbuhaler [16].

Cough is an essential symptom of asthma and in COPD and has been correlated with worse control. The occurrence of cough during inhalation has been observed in more than

one third of the patients treated with ICSs [5]. It has been proposed that this side effect occurs as a result of a toxic role of inhaled excipients (oleic acid) from pMDIs, and from nonspecific irritant effects of ICSs [17].

Increased dose frequency is known to positively correlate with the incidence of local side effects [12,17,18]. Twice-daily regimens reduced the risk of dysphonia and candidiasis compared with administration four times per day. Once-daily use of BUD delivered via a turbuhaler is practically free from local side effects in patients starting to receive this treatment [18].

Aim of the work

The aim of this work was to establish the voice changes in patients with chronic obstructive pulmonary disease.

Subjects

Fifty adults with chronic obstructive lung disease were conducted in this study. Patients were admitted to the chest department, Alexandria University hospital and were enrolled after obtaining informed consents.

Methods

All patients were conducted to the following protocol:

- (1) Clinical diagnostic aids include history taking (age, sex, smoking index and drug history), local chest examination and general examination:
 - (a) Local examination:
 - To confirm the disease.
 - To exclude other chest diseases and exacerbation.
 - (b) General examination:
 - To exclude other organ involvement.
 - To exclude co-morbidities (gastro esophageal reflux disease and sinusitis).
 - To assess complications.
- (2) Arterial blood gases: using Nova biomedical (Phox S/N: UO/A 98010). USA measured the following parameters:
 - Arterial PH.
 - Arterial oxygen tension (PaO₂).
 - Arterial carbon dioxide tension (PaCO₂).
 - Oxygen saturation (SaO₂).
 - Level of serum bicarbonate (HCO₃) in mEq/L.
- (3) Spirometry: all patients underwent standard spirometry performed by trained personal. Techniques were carried out according to American Thoracic Society/European Respiratory Society standards [19].
- (4) Plain chest X-ray P-A view.
- (5) ENT examination and endoscopic examination of the larynx in order to assess any vocal fold pathology.
- (6) Aerodynamic measurements to assess the vital capacity and maximum phonation time for calculation of Phonatory Quotient.

- (7) Elementary diagnostic procedures include the patient's interview and auditory perceptual assessment of the patient's sample voice in order to assess the grade of dysphonia by using auditory perceptual assessment (APA): after careful listening to the patient's voice by three phoniatricians, any deviation of normal voice (i.e. dysphonia) was noted and reported in each group according to the following protocol of assessment: (modified GRBAS scale, Kotby, 1986) [20] Overall grade: (0) normal (1) slight (2) moderate (3) severe
- Character (quality): strained-leaky-breathy-rough (irregular).
 - Pitch: increase-decrease-diplophonia.
 - Register: habitual register-modal-falsetto-tendency of vocal fry at the end of phrase-register break.
 - Loudness: excessively loud-soft-fluctuating.
 - Glottal attack: normal-soft-hard.
 - Associated laryngeal functions: cough-whisper.

(8) Additional instrumental procedures include acoustic analysis of the patient's voice to assess the fundamental frequency (average F0), jitter%, shimmer%, N/H ratio.

Statistical analysis

Statistical analysis was performed with Sigma Stat 2.0 (Systat Software Inc., Point Richmond, Calif) and SPSS 14 (SPSS, Chicago, Ill) for Windows.

Results

This study was carried on 50 COPD patients, 46 males (92%) and 4 females (8%) their age ranged from 32 to 76 years with a mean ± SD value of 49.88 ± 15.30 years, all patients were current or former smokers and the pack year index ranged from 20 to 66 with a mean ± SD value of 41.16 ± 13.80. **Table 1** shows the arterial blood gases, spirometric data and acoustic analysis of voice.

Table 1 The arterial blood gases, spirometric data and acoustic analysis of voice.

| | Minimum | Maximum | Mean | SD |
|-------------------|---------|---------|---------|--------|
| pH | 7.38 | 7.43 | 7.41 | 0.01 |
| PaO ₂ | 87 | 95 | 91.21 | 2.45 |
| PaCO ₂ | 44 | 50 | 46.32 | 1.76 |
| HCO ₃ | 24 | 29.32 | 27.44 | 2.08 |
| SaO ₂ | 94 | 97 | 96.04 | 1.06 |
| FVC | 53 | 88 | 73.580 | 10.012 |
| FEV1 | 50 | 84 | 69.360 | 10.203 |
| FEV1/FVC | 44 | 87 | 65.280 | 11.842 |
| MMEF | 43 | 80 | 64.560 | 11.172 |
| Average F0 | 100.65 | 125.43 | 115.987 | 7.598 |
| Jitter% | 0.02 | 1.98 | 1.157 | 0.616 |
| Shimmer% | 0.21 | 1.98 | 1.260 | 0.564 |

FVC: forced vital capacity, FEV1: forced expiratory volume after the first second, FEV1/FVC: forced expiratory volume after the first second/forced vital capacity and MMEF: maximum mid expiratory flow. Average F0: fundamental frequency.



Figure 1 Bilateral hyperaemia and inflammation of the vocal folds.

Dysphonia was perceived in 25 (50%) patients. Endoscopically, eight patients had bilateral hyperaemia and inflammation of the vocal folds (**Fig. 1**) and 3 patients had bilateral reinke's edema (**Fig. 2**). The most common predisposing factor of reinke's edema is smoking.

Table 2 shows significant positive correlation between the smoking index with Jitter%, Shimmer% and the grade of dysphonia ($p = 0.000$) and significant inverse correlation between the smoking index with fundamental frequency ($p = 0.001$).



Figure 2 Bilateral reinke's edema.

Table 2 Correlation between the smoking index with the fundamental frequency, Jitter%, Shimmer% and the degree of dysphonia.

| | Smoking index |
|--------------------|-------------------------------|
| Average F0 | $r = -0.930$ $p = 0.001^*$ |
| Jitter% | $r = 0.930$ $p = 0.000^*$ |
| Shimmer% | $r = 0.931$ $p = 0.000^*$ |
| Grade of dysphonia | $r = 0.966$ $p = 0.000^*$ |

Average F0: fundamental frequency.
* Significant correlation.

Table 3 represents significant inverse correlation between Jitter%, Shimmer% and the grade of dysphonia with the small to moderate doses of ICSs usage ($p = 0.004, 0.002$ and 0.000) respectively and significant positive correlation between fundamental frequency with the small to moderate doses of ICSs usage ($p = 0.001$). In addition, there was significant positive correlation between Jitter%, Shimmer% and the grade of dysphonia with the large doses of ICSs usage ($p = 0.000, 0.003$ and 0.002) respectively and significant inverse correlation between fundamental frequency with the large doses of ICSs usage ($p = 0.003$).

Table 4 demonstrates significant positive correlation between Jitter%, Shimmer% and the grade of dysphonia with pMDIs usage ($p = 0.004, 0.003$ and 0.000) respectively and significant inverse correlation between fundamental frequency with pMDIs usage ($p = 0.003$). Moreover, there was significant inverse correlation between Jitter%, Shimmer% and the grade of dysphonia with DPIs usage ($p = 0.000, 0.002$ and 0.001) respectively and significant positive correlation between fundamental frequency with DPIs usage ($p = 0.001$).

Table 5 shows significant inverse correlation between FVC, FEV1 and MMEF% of predicted values with Jitter%, Shimmer% and the grade of dysphonia and significant positive correlation between FVC, FEV1 and MMEF% of predicted values with fundamental frequency.

Discussion

The voice problems can vary between patients depending on co-morbidities, prescribed medications and severity of COPD. Hoarseness is often present in COPD secondary to Gastroesophageal Reflux Disease (GERD). Up to 80% of COPD patients present with symptoms associated with GERD. The acidic content of the refluxed material causes changes to the mucosal layer of the laryngeal tissue, including the true vocal folds, resulting in a change in vocal quality [21]. Effects of long term smoking which could contribute to changes in voice quality and pitch variation, upper respiratory tract infections, vocal abuse, and laryngeal cancer, could also cause hoarseness [22].

Xerostomia or dry mouth can also be related to vocal hoarseness. The presence of dry mouth, and therefore hoarse vocal quality, can commonly be attributed to inhaled corticosteroids or mouth breathing. The side effects associated with inhaled steroids may be due to inflammation or irritation of the oropharyngeal and laryngeal mucosa, caused by residue

Table 4 Correlation between the pressurized metered dose inhalers usage and the dry powder inhalers usage with the fundamental frequency, Jitter%, Shimmer% and the degree of dysphonia.

| | pMDIs usage | DPIs usage |
|--------------------|-------------------------------|-------------------------------|
| Average F0 | $r = -0.684$ $p = 0.003^*$ | $r = 0.684$ $p = 0.001^*$ |
| Jitter% | $r = 0.811$ $p = 0.004^*$ | $r = -0.811$ $p = 0.000^*$ |
| Shimmer% | $r = 0.886$ $p = 0.003^*$ | $r = -0.886$ $p = 0.002^*$ |
| Grade of dysphonia | $r = 0.878$ $p = 0.000^*$ | $r = -0.878$ $p = 0.001^*$ |

Average F0: fundamental frequency.

pMDIs: pressurized metered dose inhalers.

DPIs: dry powder inhalers.

* Significant correlation.

from the inhaled substance, oropharyngeal candidiasis and steroid-induced vocal cord myopathy. Increased hoarseness, rough voice, and loss of speech volume were reported in patients on higher doses of inhaled steroids [23].

In this study dysphonia was identified by high Jitter%, Shimmer% and grade of dysphonia and low fundamental frequency (average F0). There was significant positive correlation between the smoking index with Jitter%, Shimmer% and the grade of dysphonia ($p = 0.000$) and significant inverse correlation between the smoking index with fundamental frequency ($p = 0.001$).

Smoking may result in a far more dramatic loss of lung function and dysphonia. The tobacco-associated hoarseness can arise from disease of the larynx or vocal cords, either inflammation or tumor growth. Matsuo et al. [24] found that hoarseness in tobacco smokers is associated with an increased frequency of polypoid vocal fold lesions and head and neck cancer. Other study reported the acute and chronic effects of cigarette smoke on oropharyngeal function; Dua et al. [25] compared pharyngo-upper-esophageal sphincter contractile reflexes in 10 healthy smokers and 10 healthy non-smokers, and showed that smokers had an increased threshold required for the initiation of the pharyngo-upper-esophageal sphincter contractile reflex and high prevalence of GERD, micro-aspiration and hoarseness.

Table 3 Correlation between the small to moderate doses of inhaled corticosteroids usage and the high doses of inhaled corticosteroids usage with the fundamental frequency, Jitter%, Shimmer% and the degree of dysphonia.

| | Small to moderate doses of inhaled corticosteroids usage | High doses of inhaled corticosteroids usage |
|--------------------|--|---|
| Average F0 | $r = 0.684$ $p = 0.001^*$ | $r = -0.651$ $p = 0.003^*$ |
| Jitter% | $r = -0.811$ $p = 0.004^*$ | $r = 0.684$ $p = 0.000^*$ |
| Shimmer% | $r = -0.886$ $p = 0.002^*$ | $r = 0.659$ $p = 0.003^*$ |
| Grade of dysphonia | $r = -0.878$ $p = 0.000^*$ | $r = 0.664$ $p = 0.002^*$ |

Average F0: fundamental frequency.

* Significant correlation.

Table 5 Correlation between the spirometric data of the patients with the fundamental frequency, Jitter%, Shimmer% and the degree of dysphonia.

| | FVC | FEV1 | FEV1/FVC | MMEF |
|--------------------|-------------------------------|-------------------------------|-------------------------------|-------------------------------|
| Average F0 | $r = 0.738$ $p = 0.001^*$ | $r = 0.723$ $p = 0.000^*$ | $r = 0.733$ $p = 0.003^*$ | $r = 0.723$ $p = 0.000^*$ |
| Jitter% | $r = -0.792$ $p = 0.000^*$ | $r = -0.856$ $p = 0.003^*$ | $r = -0.864$ $p = 0.004^*$ | $r = -0.837$ $p = 0.002^*$ |
| Shimmer% | $r = -0.840$ $p = 0.005^*$ | $r = -0.915$ $p = 0.001^*$ | $r = -0.919$ $p = 0.000^*$ | $r = -0.891$ $p = 0.000^*$ |
| Grade of dysphonia | $r = -0.828$ $p = 0.004^*$ | $r = -0.890$ $p = 0.000^*$ | $r = -0.833$ $p = 0.000^*$ | $r = -0.863$ $p = 0.003^*$ |

Average F0: fundamental frequency.

FVC: forced vital capacity, FEV1: forced expiratory volume after the first second, FEV1/FVC: forced expiratory volume after the first second/forced vital capacity and MMEF: maximum mid expiratory flow.

* Significant correlation.

Hoarseness can also occur in response to post-nasal drip secondary to cigarette-smoke induced chronic sinusitis [22].

The frequent use of ICSs, especially at higher doses, has been accompanied by concern about both systemic and local side effects. The systemic complications of ICSs have been extensively studied and are well-documented in the literature. There are comparatively few studies reporting on the local complications of ICSs. Compared with systemic side effects, the local side effects of ICSs are considered to constitute infrequent and minor problems. However, while not usually serious, these local side effects are of clinical importance.

In this study there was significant inverse correlation between Jitter%, Shimmer% and the grade of dysphonia with the small to moderate doses of ICSs usage ($p = 0.004$, 0.002 and 0.000) respectively and significant positive correlation between fundamental frequency with the small to moderate doses of ICSs usage ($p = 0.001$). In addition, there was significant positive correlation between Jitter%, Shimmer% and the grade of dysphonia with the large doses of ICSs usage ($p = 0.000$, 0.003 and 0.002) respectively and significant inverse correlation between fundamental frequency with the large doses of ICSs usage ($p = 0.003$).

However, the anti-inflammatory steroid preparation would cause inflammation in the upper airway, the problem is probably multifactorial, depending on the following factors: the steroid (e.g., preparation, carrier substance, dose of steroid, and regime), the manner in which it is propelled into the airways (i.e., the inhaler device), intrinsic inflammation of the upper airway in COPD patients, mechanical irritation because of cough, inflammatory disease (e.g., rhinitis and postnasal catarrh) and inflammatory stimuli (e.g., smoking and noxious agents in the workplace).

Williamson et al. [10] reported dysphonia in 58% of patients using pressurized aerosol ICS preparations, compared with 13% of controls. In 2000, Lavy et al. [26] confirmed dysphonia via videostrobolaryngoscopy in 58% of patients who were receiving ICS therapy. Rachelefsky et al. [27] analyzed data from 23 studies published from 1966 through 2004 and determined that, compared with placebo, ICS at all dosages was associated with a 5.2-fold greater risk of dysphonia.

The wide range of incidence rates in these studies likely was due to methodologic factors, dosage variability and how the diagnosis was made, which included self-reported questionnaires, telephone surveys, clinical diagnoses, endoscopic

examinations, and histologic analyses of biopsy samples. Bearing in mind the limitations of the described studies, we believe that dysphonia caused by ICS therapy is not rare and should be considered a frequent cause of dysphonia.

Causes of dysphonia associated with ICS therapy have been poorly investigated, and the origins of dysphonia may have multiple confounding factors. Williams et al. [28] identified bowing in the vocal folds and proposed a possible association between dysphonia and ICS therapy. Subsequently, they postulated that this bowing was due to a bilateral adductor myopathy induced by local deposition of topical corticosteroids.

Lavy et al. [26] described 22 patients with ICS-associated dysphonia who underwent videostrobolaryngoscopy and an objective acoustic analysis. Seventeen of the 22 were affected by dysphonia on a daily basis: 9 had some evidence of poor vocal fold apposition, and supraglottic hyperfunction was identified in 8. Mucosal quality abnormalities were identified in 13 patients. The mucosal wave was difficult to evaluate; however, the authors identified 2 patients with mucosal wave asynchrony. The individual analysis found no correlation between the degree of vocal cord apposition and the speech evaluation findings. The authors suggested that atrophy with bowing was not likely the primary cause of dysphonia. Although they noted that the primary cause was difficult to establish because of various findings, they maintained that corticosteroids had a direct effect on the mucosa or on the mucus secreting glands of the ventricles or the trachea.

The use of ICS predisposes to the development of an inflammatory infiltrate; however, this does not necessarily have a clinical correlate. In a prospective observational study of 50 patients, 18 more inflammatory infiltrate was identified in ICS users compared with nonusers; however, pharyngeal erythema was not correlated with an inflammatory infiltrate. Dysphonia associated with ICS use likely results from deposition of active ICS in the oropharynx during administration of the medication, so that a specific cause or mechanism of this disorder has not yet been elucidated [29].

In this study there was significant positive correlation between Jitter%, Shimmer% and the grade of dysphonia with pMDIs usage ($p = 0.004$, 0.003 and 0.000) respectively and significant inverse correlation between fundamental frequency with pMDIs usage ($p = 0.003$). Moreover, there was significant inverse correlation between Jitter%, Shimmer% and the grade of dysphonia with DPIs usage ($p = 0.000$, 0.002 and

0.001) respectively and significant positive correlation between fundamental frequency with DPIs usage ($p = 0.001$).

Dysphonia may be affected by the method used to administer medication. Selroos et al. [12] described 154 patients who received ICS therapy for 2 years via an MDI and then switched to administration via a DPI. They noted that the frequency of dysphonia decreased from 21% to 6%. This change may be attributable to differences in vocal cord positioning when using a DPI compared with an MDI.

The meta-analysis of randomized controlled trials by Rachelefsky et al. [27] found that ICS MDI devices were associated with a 5-fold greater risk of dysphonia when compared with placebo MDI devices, whereas the ICS DPI devices had 3-fold greater risk vs. placebo DPI devices.

As regard dysphonia in patients receiving combined corticosteroid and bronchodilator therapy, Mirza et al. [30] described voice and laryngeal changes in 5 patients who switched from corticosteroid and bronchodilator therapy administered separately to concurrent administration. Most patients had areas of hyperemia and a plaque pattern on the surface mucosa. The combination therapy was stopped to assess reversibility of the mucosal lesions. Twelve weeks after stopping the combination therapy, patients underwent a laryngeal examination; 3 showed substantial improvement in lesions, and 2 seemed to have complete recovery. More recently, a systematic review of randomized controlled trials by Frois et al. [31] found no differences in dysphonia or other local adverse effects when comparing fluticasone or budesonide combined with long-term bronchodilator therapy.

In a pMDI, the drug is dissolved or suspended in a propellant under pressure, and, when activated, a valve system releases a metered dose of the drug and propellant. The propellant provides the force to propel and disaggregate particles. pMDIs may be manually actuated or breath-actuated. They can be used alone or in combination with various devices or adaptations (e.g., spacers or extended mouthpieces) designed to slow the aerosol cloud, reduce oropharyngeal deposition, and promote ease of use. This reduces the need for coordination of actuation and inhalation, making the device easier to use [32].

DPIs do not require propellants but rely on the patient's inspiratory effort to disperse the drug into small particles and deliver it to the lungs. An inspiratory flow rate of 30 L/min is needed to work the most efficient DPIs, and nearly all adults can achieve this (adult average, 60 L/min), even when wheezy [33].

Common problems include the inability to coordinate actuation and inspiration precisely enough to use a pMDI, or the inability to inhale forcefully enough when wheezy to use a DPI.

A dry powder inhaler containing both a long-acting muscarinic antagonist (LAMA) and long-acting beta agonist (LABA) has been approved for use in COPD in the United States. The combination was compared with each of the individual agents and placebo in a 24-week randomized trial that included over 1500 subjects with COPD. The combination inhaler achieved greater increases in pulmonary function and improved symptom control compared with the individual agents and placebo. Adverse local events like hoarseness were infrequent [34].

In this study there was significant inverse correlation between FVC, FEV1 and MMEF% of predicted values with Jitter%, Shimmer% and the grade of dysphonia and significant positive correlation between FVC, FEV1 and MMEF% of predicted values with fundamental frequency.

The quality of voice is depending on breath support. Even subtle respiratory problems can lead to changes in voice. Aerodynamic measurements play a role in quantifying airflow during respiration and phonation. Pulmonary function tests may play a role in identifying subtle respiratory problems. Dysphonia can be directly linked to the disturbances in airflow volume and rate. The decreased lung volume associated with COPD and the common breathlessness contribute to dysphonia and reduce message length resulting in a decrease in vocalization efficiency [22].

Hypoxia causes deep structural and durational changes of voice and that the same degree of hypoxia associated with mental activities during test performance causes deeper degradation of voice structure. The soft phonation may be contributed to the narcotic effect of hypercapnia [35].

Laryngeal observation is the most common practical technique used in assessing all voice disorders to show muscle tension. Every instance of recent onset, persistent hoarseness should be thoroughly evaluated; never ignored or attributed to a benign cause. Persistent hoarseness must be taken seriously as it may be a sign of serious disease [36].

In conclusion: Dysphonia (hoarseness) in COPD patients is multifactorial. The voice problems can vary between COPD patients depending on the severity of smoking, prescribed medications, method used to administer medication and disease severity. However, inhaled corticosteroids are associated with an increased occurrence of dysphonia. Any evaluation of dysphonia in COPD patient should consider the use of ICS as an adverse effect includes a thorough examination of the larynx, and rule out vocal cord nodules, post tussive trauma and gastroesophageal reflux. We should use the lowest effective dose of ICS and patients should be instructed to rinse the mouth after inhalation. Instructions should be provided on the proper method of inhalation. Successful analysis should depend on cooperation and collaboration among pulmonologists, voice care specialists, and other laryngologists in the care of any COPD patient had hoarseness of voice.

Conflict of interest

None declared.

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