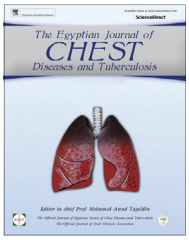
Egyptian Journal of Chest Diseases and Tuberculosis (2014) **63**, 561–567

The Egyptian Society of Chest Diseases and Tuberculosis

Egyptian Journal of Chest Diseases and Tuberculosis

www.elsevier.com/locate/ejcdt
www.sciencedirect.com



ORIGINAL ARTICLE

Voice changes in patients with chronic obstructive pulmonary disease

Enas Elsayed Mohamed [a,*], **Riham Ali El maghraby** [b]

[a] *Chest Diseases Department, Faculty of Medicine, Alexandria University, Egypt*
[b] *Phoniatrics Department, Faculty of Medicine, Alexandria University, Egypt*

Received 15 February 2014; accepted 4 March 2014
Available online 27 March 2014

KEYWORDS

Chronic obstructive pulmonary disease;
Dysphonia;
Smoking;
Inhaled corticosteroids

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 ± 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.

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

* Corresponding author. Tel.: +20 0 1224474317.
E-mail address: enas_elsayed73@yahoo.com (E.E. Mohamed).

Peer review under responsibility of The Egyptian Society of Chest Diseases and Tuberculosis.

ELSEVIER Production and hosting by Elsevier

0422-7638 © 2014 The Egyptian Society of Chest Diseases and Tuberculosis. Production and hosting by Elsevier B.V.
Open access under CC BY-NC-ND license. http://dx.doi.org/10.1016/j.ejcdt.2014.03.006

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 (oleicacid) 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 (PaO_2).
   - Arterial carbon dioxide tension ($PaCo_2$).
   - Oxygen saturation (SaO_2).
   - Level of serum bicarbonate (Hco_3) 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.