# Chronic obstructive pulmonary disease in treated pulmonary tuberculous patients

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***Background/Aim*** To detect the prevalence of chronic obstructive pulmonary disease (COPD) as a sequel of treated pulmonary tuberculosis (PTB).

***Materials and methods*** A total of 50 adults, 28 men and 22 women, with a definite diagnosis of PTB and complete antituberculous therapy, with subsequent presentation of exertional dyspnea and/or cough, and expectorations for which no other alternative cause was found, were included in our study. All the patients underwent full history taking, full clinical examination, chest radiography, erythrocyte sedimentation rate, prebronchodilator and postbronchodilator forced vital capacity (FVC%), and forced expiratory volume ($FEV_1$%) in the first second of $FEV_1$/FVC%.

***Results*** Pulmonary function testing showed 22 patients (44%) with irreversible obstructive pattern denoting chronic obstructive pulmonary disease (COPD), seven patients had restrictive ventilatory defect, and three patients had mixed obstructive and restrictive pattern. Of those 22 patients with irreversible obstructive pattern (COPD), 11 patients (50%) had mild obstruction, nine patients (40.9%) had moderate obstruction, and two patients (9.1%) had severe obstruction. There is a positive correlation between dyspnea and post-tuberculous COPD patients, and a negative correlation between cough and post-tuberculous COPD patients. There is no correlation between the duration since the completion of antituberculous therapy and development of COPD.

***Conclusion*** COPD can be a sequel of PTB and should be overlooked, especially in those patients complaining of dyspnea even in the absence of any history of smoking. Post-tuberculous COPD as a cause of COPD in nonsmokers should be now more recognized in countries where the prevalence of PTB is still high.

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## Introduction

Chronic obstructive pulmonary disease (COPD) is estimated to affect 65 million people worldwide. It is currently the third leading cause of death, accounting for approximately three million deaths annually. Of the total number of deaths, 90% are in low-income and middle-income countries where the prevalence of pulmonary tuberculosis (PTB) remains high [1].

A relationship between PTB and the development of COPD has been suggested in several reports. However, a serious limitation is the confounding caused by concurrent exposure to risk factors such as tobacco smoking, dust and biomass fuel, and childhood respiratory illnesses, and a lack of diagnostic precision when distinguishing COPD from other forms of structural lung disease (e.g. bronchiectasis) found in patients who had PTB [2].

Chronic obstructive airway disease as a complication of PTB has been restudied recently in many regions of the globe [3,4]. In the executive summary of the 2006 update of the Global initiative for chronic obstructive lung disease (GOLD) guidelines [5], the role of tuberculosis (TB) in the development of chronic airway obstruction (CAO) has been recognized. According to the GOLD Workshop summary, chronic bronchitis or bronchiolitis and emphysema can occur as complications of PTB [6].

## Aim of the study

The aim of this study was to detect the prevalence of COPD as a sequel of treated PTB.

## Materials and methods

A total of 50 adults, 28 men and 22 women from TB clinic of Chest Diseases Department, Cairo University, previously diagnosed as having PTB based on clinical suspicious, chest radiography, and a positive sputum examination for acid fast bacilli by Ziehl Neelson, who had a complete antituberculous therapy, with subsequent presentation of chronic exertional dyspnea and/or cough and expectorations for which no other alternative cause was found, were included in our study.

Those patients having a probability of reactivated TB, having a history of current or previous smoking or occupational exposure, asthmatics, and cases of

interstitial lung disease and ischemic heart disease were excluded.

Patients were subjected to:

(1) Full history taking.
(2) Full clinical examination.
(3) Chest radiography and erythrocyte sedimentation rate.
(4) Sputum for acid fast bacilli by Ziehl Neelson.
(5) Prebronchodilator and postbronchodilator forced vital capacity (FVC%), forced expiratory volume in the first second ($FEV_1$%) and $FEV_1$/FVC %.

Prebronchodilator and postbronchodilator FVC%, $FEV_1$%, and $FEV_1$/FVC% were recorded in each case through simple spirometry on ZAN Messgeraete, 1999 GmbH (Schlimpfhofer Strasse 14, Oberthulba, 97723, Germany). Stages and pattern of COPD were recorded; classification of the severity of airflow limitation was done as per revised GOLD 2013 [7]. This was carried out in the Chest Department of Kasr El Aini Hospital, from February 2013 to March 2014.

Data were statistically described in terms of mean ± SD, median and range, or frequencies (number of cases) and percentages when appropriate. Comparison of numerical variables between the study groups was made using Student's *t*-test for independent variables. Correlation between various variables was carried out using Spearman's rank correlation equation for non-normal variables. *P*-value less than 0.05 was considered statistically significant. All statistical calculations were done using computer program SPSS (SPSS Inc., Chicago, Illinois, USA) version 15 for Microsoft Windows (California, USA).

## Results

This study included 50 patients, 28 men and 22 women, their age ranging from 23 to 63 years old, with a mean age of 40.70 years. All patients successfully completed their antituberculous chemotherapy from a period ranging from 5 to 18 years with a mean of 10 years ago.

A total of 39 patients complained of dyspnea, 22 patients complained of cough, and 16 patients complained of both dyspnea and cough.

Pulmonary function testing showed 22 patients (44%) with irreversible obstructive pattern denoting COPD, seven patients had restrictive ventilatory defect, and three patients had mixed obstructive and restrictive pattern (Table 1).

Of those 22 patients with irreversible obstructive pattern (COPD), 11 patients(50%) had mild obstruction, nine patients(40.9%) had moderate obstruction, and two patients (9.1%) had severe obstruction, as per revised GOLD classification of 2013 [7] (Table 2).

Table 3 shows a positive correlation between dyspnea and pulmonary functions.

Table 4 shows no correlation between cough and pulmonary functions.

Table 5 shows no correlation between the duration since the completion of antituberculous therapy and development of COPD.

## Discussion

Our study found that of the 50 symptomatic patients,

**Table 1 Sex, ventilatory defect on pulmonary functions, and complaints of patients**

| | Number of patients [*n* (%)] |
|---|---|
| Sex | |
| Male | 28 (56.0) |
| Female | 22 (44.0) |
| Total number | 50 (100.0) |
| Ventilatory defect type | |
| Normal | 18 (36.0) |
| Restrictive | 7 (14.0) |
| Obstructive | 22 (44.0) |
| Mixed | 3 (6.0) |
| Total number | 50 (100.0) |
| Dyspnea | |
| No | 11 (22.0) |
| Yes | 39 (78.0) |
| Total number | 50 (100.0) |
| Cough | |
| No | 28 (56.0) |
| Yes | 22 (44.0) |
| Total number | 50 (100.0) |

**Table 2 Degree of airflow limitation among chronic obstructive pulmonary disease patients**

| | Count [*n* (%)] |
|---|---|
| Stage | |
| Mild | 11 (50.0) |
| Moderate | 9 (40.9) |
| Severe | 2 (9.1) |
| Group total | 22 (100.0) |

**Table 3 Correlation between dyspnea and pulmonary functions**

| | Dyspnea [count (row %)] | | *P*-value |
|---|---|---|---|
| | No | Yes | |
| Pulmonary functions | | | |
| Normal | 8 (44.4) | 10 (55.6) | |
| Restrictive | 2 (28.6) | 5 (71.4) | |
| Obstructive | 1 (4.5) | 21 (95.5) | 0.015 |
| Mixed | | 3 (100.0) | |
| Group total | 11 (22.0) | 39 (78.0) | |

**Table 4 Correlation between cough and pulmonary functions**

| | Cough [count (row %)] | | *P*-value |
|---|---|---|---|
| | No | Yes | |
| Pulmonary functions | | | |
| Normal | 10 (55.6) | 8 (44.4) | |
| Restrictive | 4 (57.1) | 3 (42.9) | |
| Obstructive | 12 (54.5) | 10 (45.5) | 1.0 |
| Mixed | 2 (66.7) | 1 (33.3) | |
| Group total | 28 (56.0) | 22 (44.0) | |

**Table 5 Correlation between the duration of stoppage of antituberculous treatment and pulmonary functions changes**

| Pulmonary functions | *N* | Mean | SD | Median | Minimum | Maximum |
|---|---|---|---|---|---|---|
| Normal | 18 | 9.56 | 2.915 | 10.0 | 5 | 14 |
| Restrictive | 7 | 11.29 | 3.546 | 12.0 | 5 | 15 |
| Obstructive | 22 | 10.82 | 3.568 | 10.0 | 5 | 18 |
| Mixed | 3 | 11.33 | 3.215 | 10.0 | 9 | 15 |
| Total | 50 | 10.46 | 3.296 | 10.0 | 5 | 18 |

22 patients (44%) who successfully completed their antituberculous regimen, presenting mainly with dyspnea with or without cough, developed COPD, denoting that COPD can be a sequel of treated PTB.

Lee and Chang [8] found that CAO is a common finding owing to TB-destroyed lung. Patricio Jiménez *et al.* [9] found that CAO is a common sequel with TB.

PLATINO study, a latest large population-based multicenter study, carried out in five Latin American countries ($n$ = 5571 participants) included patients on the criteria of a past diagnosis of PTB by a physician and performed spirometry in the field. It included only those patients presenting to the hospital with dyspnea. Along with the exclusion of other possible confounding factors, smokers and patients with age more than 65 years were also excluded; it was found that $FEV_1$ is reduced compared with FVC in most cases [10]. However, another previous study had found that, after 15 years' follow-up of 40 patients, there was a higher yearly decline in FVC compared with $FEV_1$ [11].

Kim *et al.* [12] conducted a study to assess the impact of PTB on the prevalence of COPD, and found that the prevalence of COPD increased from 3.7 to 5% by including participants with a history of TB treatment.

COPD can occur as one of the chronic complications of PTB, and the obstructive ventilatory defect appears more common among various pulmonary function derangements [13].

Verma *et al.* [14] concluded that there is indeed an important contribution of TB to airflow obstruction (AFO), linking two of the most common ailments in the world. For many persons with TB, microbiological cure is just the beginning, not the end of their illness. The prevention and adequate treatment of TB would reduce the burden of AFO in all countries, especially the developing countries. However, the exact abnormality that results from tuberculous infection has to be considered in detail with future studies, and a better understanding of the pathophysiology of airflow limitation may point the way to therapeutic strategies for control of symptoms in these patients.

Allwood *et al.* [2] confirms an association between a past history of TB and the presence of CAO. This association is independent of cigarette smoking and biomass fuel exposure. The mechanisms underlying the development of AFO and its natural history and response to treatment require further study. AFO may progress after the completion of PTB treatment. In view of the large number of patients with PTB worldwide, and the rising incidence of COPD globally, the contribution of PTB as a contributory cause in the pathogenesis of COPD is important both to epidemiologists and health-care providers.

In our study, we could not find a correlation between the duration since the completion of antituberculous therapy and development of COPD. Willcox and Ferguson [15] found that the obstructive changes become pronounced after 10 years of follow-up in treated cases and correlated with the residual scarring on chest radiograph, regardless of the findings on original chest radiographs.

Obstructive airway disease has many causes. TB, which can be a cause of this, has not been studied in detail. Even the organizations, such as GOLD and GINA, authority figures in COPD and asthma, respectively, have not recognized or treated PTB as an etiological factor, which again shows that post-pulmonary tuberculous obstructive airway disease is a clinical entity in its infancy. Post-tubercular impairment can be manifested as reversible or irreversible obstructive airway disease, mixed defect, or as pure restrictive defects [14].

COPD can be a sequel of PTB and should be overlooked, especially in those patients complaining of dyspnea even in the absence of any history of smoking. Post-tuberculous COPD as a cause of COPD in nonsmokers should be now more recognized in countries where the prevalence of PTB is still high.

## Acknowledgements

### Conflicts of interest

None declared.

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