ORIGINAL RESEARCH PAPER

Pulmonary Medicine

OCCURRENCE OF OBSTRUCTIVE AIRWAY DISEASE IN TREATED PATIENTS OF PULMONARY TUBERCULOSIS : A RETROSPECTIVE STUDY

KEY WORDS:

Dr. Shivprasad Kasat

Dr. Ashish Deshmukh

Dr. Sunil Jadhav

Dr. Hafiz Deshmukh

Dr. Ashish Kendre

ABSTRACT

Aim: To study occurence of obstructive airway disease in treated patients of pulmonary tuberculosis **Materials and methods**: 50 patients data collected from Respiratory medicine department, MGM medical college, Aurangabad, who visited OPD for various respiratory symptoms. It is ensured that patients who are participated in the study are non smokers. Previous history of pulmonary tuberculosis and treatment history is noted. Evidences of previous treatment and radiological images were collected. Each patient was subjected to a thorough clinical examination and spirometry. **Results**: Based on clinical findings, radiological findings, and spirometry findings, of the 50 patients studied, it is observed that 11(22%) patients had developed obstructive airway disease.4 (8%) patients have developed extensive fibrosis post tuberculosis. 3 (6%) patients have developed cavitation with fibrosis. 4 (8%) patients have developed bronchiectasis.**Conclusion:** It is not uncommon that obstructive airway disease occurs as a complication of post tuberculosis sequale. 22 percent of the patients studied, have developed different types of obstructive airway diseases. Many factors may influence in post tuberculosis complications such as extensiveness of parenchymal involvement, duration of illness, socio-economic factors. Obstructive airway disease can be a sequel of pulmonary tuberculosis and should be overlooked, especially in those patients complaining of dyspnea even in the absence of any history of smoking. Post-tuberculous obstructive airway disease as a cause of COPD in nonsmokers should be now more recognized in countries where the prevalence of pulmonary tuberculosis is still high.

INTRODUCTION

Tuberculosis, a disease of great antiquity has become the most important communicable disease in the world. With over 8.8 million new cases occurring every year, 1.9 million belong to India (21%). It is estimated that 2 out of every 5 Indians are infected with TB and three lakh twenty two thousand Indians die of TB every year. [1] In other words, it is estimated that two persons die of tuberculosis every minute[1]. Obstructive airway disease has many causes. Tuberculosis, which can be a cause of this, has not been studied in detail. For many persons with tuberculosis, microbiological cure is just the beginning, not the end of their illness. Post tuberculosis pulmonary impairment has emerged as a distinct clinical entity, which is almost indistinguishable from other forms[2,3].

Pulmonary tuberculosis can lead to obstructive and restrictive lung disease resembling COPD. It can result in both reversible and irreversible airway obstruction. It is unclear whether there is a similarity in the pathology but clinically we see a post tubercular disease which is more or less similar to COPD.

Cavitation, extensive fibrosis, bulla formation and bronchiectasis implicated in the genesis of COPD caused by the destroyed lung due to pulmonary tuberculosis. Only a few studies have been done to identify this entity, but all the studies have definitely concluded that such an entity exists.

MATERIALS AND METHODS

This study was conducted at MGM Medical college and hospital, Aurangabad.

The study used data collected from 50 patients who visited OPD for various respiratory symptoms who have been treated for pulmonary tuberculosis in the past. All patients were subjected to a thourough clinical examination and spirometry. Previous treatment history, radiological images were noted.

Patients who are smokers and have previous history of obstructive airway disease prior to tuberculosis treatment were excluded.

RESULTS

Based on clinical findings, radiological findings, and spirometry findings, of the 50 patients studied, it is observed that 11(22%) patients had developed obstructive airway disease.

4 (8%) patients have developed extensive fibrosis post tuberculosis.

3 (6%) patients have developed cavitation with fibrosis.
4 (8%) patients have developed bronchiectasis.

DISCUSSION

Studies show that in patients with obstructive airway impairement, post pulmonary tuberculosis can be an important cause.

Incidences of obstructive airway disease in post tuberculosis patient was not studied extensively. It is difficult to predict that which patients are more likely to develop obstructive airway disease. Although some studies have shown that extensiveness of lung parenchymal involvement, duration of the illness, socio-economic factors may have an impact on likelihood of development of complications.

Cavitation, extensive fibrosis, bulla formation and bronchiectasis have been implicated in the genesis of obstructive airway disease caused by the destroyed lung due to pulmonary Tuberculosis[4,5]

Nefedov and Popova attributed that the main cause of better lung function was the resolution of fresh inflammatory changes and that of worse lung function was cicatricial transformation of lung tissue.[6]

The average duration of onset of obstructive airway disease has been found variable. Study undertaken by Hnizdo et al found that the average time between the diagnosis of the last episode of tuberculosis and the lung function test was 4.6 years (range one month to 31 years)[7]. The loss of lung function was highest within six months of the diagnosis of tuberculosis and stabilised after 12 months when the loss was considered to be chronic.

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. 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[8].

Allwood *et al.*[9] confirms an association between a past history of TB and the presence of chronic airway obstruction. This association is independent of cigarette smoking and biomass fuel exposure. The mechanisms underlying the development of airflow obstruction and its natural history and response to treatment require further study. Airflo obstruction 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 an exact correlation between the duration since the completion of antituberculous therapy and development of ventillatory defect . However we found that the obstructive changes become pronounced after 1 year of follow-up in treated cases and correlated with the residual scarring on chest radiograph, regardless of the findings on original chest radiographs. It is also observed that ventillatory defects on pulmonary function test have established before the chest radiography changes set in.

CONCLUSION

It is not uncommon that obstructive airway disease occurs as a complication of post tuberculosis sequale. 22 percent of the patients studied, have developed different types of obstructive airway diseases. Many factors may influence in post tuberculosis complications such as extensiveness of parenchymal involvement, duration of illness, socio-economic factors. Obstructive airway disease can be a sequel of pulmonary tuberculosis and should not be overlooked, especially in those patients complaining of dyspnea even in the absence of any history of smoking. Post-tuberculous obstructive airway disease as a cause of COPD in nonsmokers should be now more recognized in countries where the prevalence of pulmonary tuberculosis is still high.

Funding:None
Conflicts of interest : None Declared

REFERENCES

1. Global tuberculosis control: surveillance, planning, financing. WHO report 2007. Geneva, World Health Organization (WHO/ HTM/TB/2007.376).
2. Macnee W. Chronic Bronchitis and Emphysema: Seaton A, Seaton D, Leitch AG. Crofton and Douglas's Respiratory Disease, United Kingdom: Blackwell Science 2002; pp 616-617.
3. Leitch AG. Pulmonary tuberculosis: Clinical features in: Seaton A, Seaton D, Leitch AG, editors. Crofton and Douglass's Respiratory Disease, United Kingdom: Blackwell Science 2002; 523.
4. Snider GL, Doctor L, Demas TA, Shaw AR: Obstructive airway disease in patients with treated pulmonary tuberculosis. *Am Rev Respir Dis* 1971; 103: 625-640.
5. Rajasekharan S, Savitri S, Jeyaganesh D. Post tuberculosis bronchial Asthma. *Ind J Tub* 2001; 48: 139.
6. Nefedov VB, Popova LA. Study on patients with recurrent tuberculosis and ineffective primary treatment in the intensive phase of controlled chemotherapy: Probl Tuberk 2002; 12: 29-32.
7. Hnizdo E, Singh T, Churchyard GJ. Chronic pulmonary function impairment caused by initial and recurrent pulmonary tuberculosis following treatment. *Thorax* 2000; 55: 32-38.
8. Menzez AMB, Hallal PC< Perez-Padilla R, Jardim JRM, Muin OA, Loped MV et al. Tuberculosis and airflow obstruction: evidence from the PLATINO study in latin America. Eur Respir J 2007; 30: 1180-1185.
9. BW Allwood, L Myer, ED Bateman. A systematic review of the association between pulmonary tuberculosis and the development of chronic airflow obstruction in adults. *Respiration* 2013; 86: 76–85.