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Original Article

Tuberculosis Airway Disease and Bronchiectasis- A Prospective Study

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Abstract

Background: Tuberculosis is very rampant in India accounting for 20% of the global burden. Patient of Pulmonary Tuberculosis inspite having completed the complete course of anti tuberculosis therapy many a time has to continue treatment for post tubercular complications like restrictive and obstructive lung disease and bronchiectasis leading to increased cost of treatment, hospital admissions and increased morbidity and mortality.

Materials & Methods: We studied 100 patients of Chronic obstructive Pulmonary disease above 20 years of age with FEV1/FVC of 0.7 or less with associated symptoms of cough with expectoration and breathlessness and studied the history of Tuberculosis. Studied 50 cases of bronchiectasis with history of Pulmonary Tuberculosis. We looked bronchiectasis by HRCT among patients admitted for moderate to severe COPD.

Results: In this study we found that 57% of patients diagnosed to have chronic obstructive pulmonary disease by clinical examination and spirometry were found to have a history of tuberculosis treatment. Post tubercular obstructive airway disease was more common in males(Male: female ratio 48:9). Among sixty patients of posttubercular bronchiectasis Male: Female ratio was 58:42. More than 50% of these patients were smokers. Among these cases of post tubercular bronchiectasis 28% had chronic obstructive pulmonary disease as co-morbidity. In our analysis of 50 cases of moderate to severe chronic obstructive airway disease we found 60% of them had associated bronchiectasis by HRCT. Mean age of these patients of bronchiectasis associated with COPD was 63±7 with male preponderance of 9:1. This combination of patients had more exacerbations, less serum albumin levels and more mortality.

Conclusion: Tuberculosis causes bronchiectasis and chronic obstructive and restrictive lung disease similar to COPD. Chronic obstructive pulmonary disease and Tuberculosis can coexist. Chronic obstructive pulmonary disease is associated with bronchiectasis in a significant number of patients. Chronic obstructive pulmonary disease patients are prone to develop secondary bacterial infections including tuberculosis. Bronchiectasis is associated with airway disease. The triad increases the complications, morbidity and mortality.

Key words: Bronchiectasis, COPD, Exacerbations, Morbidity, Mortality

INTRODUCTION

The triad of Pulmonary Tuberculosis, obstructive airways disease and brochiectasis is responsible for considerable morbidity and mortality in India. The speciality with this triad is Tuberculosis disease of the lungs leads to fibrosis and reactive airways and leads to crippling obstructive and restrictive lung disease similar to chronic obstructive pulmonary disease.[1-3] Chronic obstructive pulmonary disease is common in smokers and so is tuberculosis. Both Chronic obstructive lung disease and pulmonary tuberculosis may coexist. COPD patients are prone to secondary bacterial infections including mycobacterial disease. Pulmonary Tuberculosis leads to bronchiectasis. Bronchiectasis causes secondary infections and associated with airway disease. Chronic obstructive lung disease patients have associated bronchiectasis. The triad leads to crippling secondary infections, increased morbidity and mortality and increase in hospital expenditure. The problem is magnified if there is coexisting diabetes mellitus and or HIV disease which increases the suffering further.[4-6]

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MATERIALS & METHODS

The protocol was approved by the local ethics committee and written informed consent was obtained from each patient.

We have included the summary of three studies done in our tertiary care centre. Study one is the enumeration of patients of COPD with history of Tuberculosis. Second study is the study of post tubercular bronchiectasis and third a study of association of bronchiectsis in moderate to severe COPD patients. We analysed our results and compared with authenticated studies.

Study I

We have studied 100 cases of COPD diagnosed with FEV1/FVC 0.7 or less presenting with symptoms of cough with expectoration and breathlessness in our tertiary care centre.

Among them 57 (57%) gave the history of tuberculosis. 48 were male and 9 were females.

Study II

Post Tubercular Bronchiectasis: We did a 2 year study on the prevalence and clinical profile of post tubercular bronchiectasis in our tertiary care centre.

We included the following patients

1. Those who completed successful Anti-TB Treatment (ATT)
2. Symptomatology included
   (a) Cough with expectoration-recurrent
   (b) Dyspnoea
   (c) Fever
   (d) Hemoptysis
3. Thorough clinical examination eliciting the signs of bronchiectasis, cystic fi brosis, consolidation and coarse crepitations.
4. Sputum for acid fast bacilli (AFB) -spot and overnight specimen done and only sputum AFB negative cases were included.

Exclusion Criteria

1. Sputum positive for AFB status
2 Extremely moribund conditions
3. Unwilling and non-cooperative clientele.

RESULTS

Sample size was 60 subjects (n=60) with male–female ratio of 35:25 (58%:42%), with in the age range of17–69 years, of which majority were in the 31–50 years group (43.3%). Initial presenting symptoms were productive cough (95%), dyspnoea (90%) and hemoptysis (35%). History of smoking was noted in 53% of total sample. It is worth noting that minority of the females were also smokers.

Chronic obstructive pulmonary disease (COPD) (28%) is the major co morbid condition associated with PTBX followed by hypertension (12%), type 2 diabetes (5%)and coronary artery disease (5%).

Majority of patients had bronchiectatic changes which were identifi able on chest X-ray (53%). One-fourth patients had fibrosis (25%). Features of destroyed lung were evident in 9 cases (15%). Fungal ball was seen in 4 cases (7%). Bilateral involvement was seen in 25 cases(42%) followed by right predominance (33%).

HRCT Thorax revealed the following types

1. Traction bronchiectasis:25 cases (42%)
2. Mixed bronchiectasis: 15 cases (25%)
3. Saccular bronchiectasis: 9 cases (15%)
4. Cystic bronchiectasis:7 cases (12%)
5. Central bronchiectasis: 4 cases (6%).

Secondary bacterial infections in our study included staphylococcal, klebsiella and pseudomonas species.

Our study of Post tubercular bronchiectasis has associated COPD in 28% of patients. Two patients in our study of sixty patients showed active tuberculosis in the bronchial washings.

BRONCHIECTASIS IN COPD PATIENTS

In our own study of association of bronchiectasis in COPD patients. 50 patients of COPD having moderate to severe COPD were analysed with clinical, chest x-ray, CT scan, serum c reactive protein and albumin levels and microbiological study by sputum microscopy for culture and sensitivity and gram stain. Mean age of the patients was 63±7.87 years. Out of 50 Patients 45 were men (90%) and the remaining were women (10%). Bronchiectasis was present in 30 patients (60%). H. influenza was the commonest organism isolated from sputum. Patients with bronchiectasis had significantly more exacerbations (p=0.0001), severe airway obstruction (p=0.037), higher CRP levels (p=0.0001) and low album In levels (p=0.007). Nine patients (30%) died in bronchiectasis group and only one patient (3.33%) died in patients without bronchiectasis. Our study showed an elevated prevalence of bronchiectasis in patients with moderate to severe COPD and was associated with severe airway obstruction. Increased exacerbations, inflammation, malnutrition and mortality in Indian patients.

DISCUSSION

China is a country of high population. The prevalence of tuberculosis in terms of absolute number is high in China. Adult patients who diagnosed as bronchiectasis at the hospital between 1995 and 2011 were recruited to this study. The amount of hospitalized patients with bronchiectasis in 2011 was 9.3 fold of that in 1995. The number was increase very fast during the seventeen years. The data tallied with the situation of pulmonary TB in China. 69.21% (4892 in 6977) patients were original from countryside. The main causes of bronchiectasis were pulmonary TB (31.17%), bacterial infection and pertussis. The peak age ranges of post-TB bronchiectasis were 30 to 39 and 60 to 69. Patients with post-TB bronchiectasis prone to have haemoptysis but less sputum. The chest radiography of patients with post-TB bronchiectasis represented upper lobes injury of the lung. Less pseudomonas aeruginosa culture positive and less acute exacerbation were recorded in post-TB bronchiectasis patients from the data of available follow-up patients.[7-8]

They concluded that the main cause of bronchiectasis in China was pulmonary TB, possiblly because of the grim pulmonary TB epidemic situation. Post-TB bronchiectasis patients have marked different clinical features and prognosis, compared with non-TB bronchiectasis.

Tuberculosis of the bronchi and bronchioles can cause destruction of the airways. Endobronchial and peribronchial obstruction of airways because of lymph nodes can cause obstruction and pooling of secretions with secondary infection and further destruction of the airways and can cause bronchiectasis. Bronchiectasis is associated with reactive airways because of secondary infection leading to obstructive and restrictive airway disease. Bronchiectasis patients are prone to secondary infections including mycobacterial disease. An association between chronic obstructive pulmonary disease (COPD) and tuberculosis (TB) has been described, mainly due to smoking and corticosteroid use. Whether inhaled corticosteroid (ICS) therapy is associated with an increased risk of TB remains unclear.[9-15]

Tuberculosis (TB) is a major cause of death worldwide. The World Health Organization estimated that there were 9 million new cases of TB in 2013 [2]. The risk factors for TB included age, male gender, low socioeconomic status, malnutrition, substance abuse, silicosis, human immunodeficiency virus infection, malignancy, diabetes, renal disease, celiac disease, gastrectomy, transplant, and receiving corticosteroids and tumor necrosis factor inhibitors. In addition, an association between obstructive pulmonary disorders (i.e. chronic obstructive pulmonary disease [COPD] and asthma) and active TB has been described, mainly due to smoking and corticosteroid use. Keeping a high suspicion and regularly monitoring for the development of pulmonary TB in COPD patients are necessary, especially for those receiving higher doses of oral corticosteroids and other COPD medications. Although ICS therapy has been shown to predispose COPD patients to pneumonia in large randomized clinical trials, it does not increase the risk of TB in real world practice.

COPD patients are at high risk of developing pulmonary TB, especially those frequently receiving oral corticosteroids and oral β-agonists. Although ICS therapy has been shown to predispose COPD patients to pneumonia in large randomized clinical trials, it does not increase the risk of TB in real world practice.

The study done by chih-Hsin Lee et al showed that COPD patients were more likely to develop pulmonary TB than non-COPD subjects under a wide variety of diagnostic scenarios for COPD. ICS was not a risk factor for developing active pulmonary TB among COPD patients after considering important clinical characteristics and other prescriptions. COPD patients who received higher doses of oral corticosteroids and oral β-agonists were more likely to develop active pulmonary TB[1.] Age, male gender, diabetes, and receiving oral corticosteroids were risk factors for TB[3]. Use of Inhalation corticosteroid ICS does not lead to increased risk of developing TB.[1] COPD patients are at risk of serious bacterial infections including tuberculosis[12]

In the executivesummary of the 2006 update of the Global initiative forchronic obstructive lung disease (GOLD) guidelines,the role of tuberculosis in the development of chronic airways obstruction has been recognized. According to the GOLD Workshop summary, chronic bronchitis or bronchiolitis and emphysema can occur as complications of pulmonary tuberculosis.[4,5]

A Pakistani study found that 55.3% of treated pulmonary tuberculosis patients presenting with dyspnoea, had an obstructive ventilatory defect.[6] Previous studies had also revealed that an obstructive pattern of pulmonary functional impairment following treated pulmonary tuberculosis was more common. Post tubercular impairment can manifest as reversible or irreversible obstructive airway disease, mixed defect or as pure restrictive defects. Immunological mechanisms have been postulated as a cause of Post tubercular asthma. 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. However, the exact abnormality that

results from tuberculosis 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.[8]

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 butclinically we see a post tubercular disease which is more or less similar to COPD.

The overall prevalence of airflow obstruction (forced expiratory volume in one second/forced vital capacity post-bronchodilator <0.7) was 30.7% among those with a history of tuberculosis, compared with 13.9% among those without a history. Males with a medical history of tuberculosis were 4.1 times more likely to present airflow obstruction than those without such a diagnosis. This remained unchanged after adjustment for confounding by age, sex, schooling, ethnicity, smoking, exposure to dust and smoke, respiratory morbidity in childhood and current morbidity. Among females, the unadjusted and adjusted odds ratios were 2.3 and 1.7, respectively.[14]

Mycobacterium Tuberculosis and Opportunist Mycobacteria

Bronchiectasis may result from pulmonary Mycobacterium tuberculosis infection, with the incidence reflecting the prevalence of tuberculosis in the population. It is also increasingly recognised that opportunist mycobacteria are associated with localised or widespread bronchiectasis. Bronchiectasis, like other forms of lung damage, makes patients prone to picking up environmental mycobacterial species and bronchial damage may occur as a result of opportunist mycobacterial infection. Opportunist mycobacteria have been isolated in 2% and 10% of random sputum specimens from patients with bronchiectasis, but the clinical significance is unclear. Patients with Mycobacterium avium complex infection may develop bronchiectasis over years.[10]

The scenario can be different in a populous country like India where tuberculosis is rampant. Tuberculosis can be a secondary complication in a case of post tuberculosis bronchiectasis patient because of endogenous reactivation secondary to poor nutrition and destroyed lung or exogenous reinfection.

Airway diseases, bronchiectasis and bronchial asthma present with similar symptoms. The differentiation between asthma, chronic obstructive pulmonary disease and bronchiectasis in the early stage of disease is extremely important for the adoption of appropriate therapeutic measures. Because of the high prevalence of these diseases and the common pathophysiological pathways, some patients with different diseases may present with similar symptoms.[11]

Bronchiectsis is often accompanied by airway disease. In our study of post tubercular bronchiectsiswas seen 28% of patient showed symptoms of COPD. Bronchiectsis is often accompanied by obstructive disease

CONCLUSION

Tuberculosis can cause bronchiectasis and airway disease. Smoking is an aetiological factor for both Tuberculosis and COPD. Bronchiectasis patients have susceptibility to bacterial infections including Mycobacterium tuberculosis and M.avium-intracellulare. Bronchiectasis is associated with airway disease and COPD patients have high prevalence of Bronchiectasis in nearly 60% of of our patients. COPD patients can have secondary infections and mycobacterial disease.

Tuberculosis, Bronchiectasis and COPD is a deadly triad responsible for increase in hospital admissions, increased health care expenditure and financial burden for the individual families.

The problem becomes much complicated in the event of coexistent Diabetes mellitus and or HIV disease both of which can be facilitating factors for development of Pulmonary Tuberculosis and complications thereon.

High degree of awareness on the part of the physician, early treatment of Tuberculosis with proper drugs, implementation of RNTCP programme, prevention of smoking and explanation of the hazards of smoking at an early age by means of awareness programmes can decrease this problem of third world countries

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