

To Investigate the Prevalence of Pulmonary TB in Individuals with Chronic Asthma: An Observational Study

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Abstract

Aim: To investigate the prevalence of pulmonary TB in individuals with chronic asthma.

Material and Methods: This study was conducted in the Department of Microbiology, Nalanda Medical College and Hospital, Patna, Bihar, India from February 2018 to January 2019. Study population was 150 asthmatic patients who were taking treatment for more than 2 years. Data was collected with pre tested questionnaire. Data included demographic data like age, sex and occupation. Detailed clinical history was taken from the patients. Clinical history related to duration of asthma, treatment taking for asthma, frequency of asthmatic attacks, past history of tuberculosis, family history of asthma was noted. A thorough clinical examination of the patients was done. All these patients were treated with anti-histaminics steroids, antibiotics bronchodilators meter dose inhalerpumps on and off frequently. Routine investigations like complete blood count, renal and liver function tests were done.

Results: In our study out of 150 patients 130patients were outside workers and 20 females were housewife. In our study, we enquired about the smoking habit in the patients. Out of 150 patients, 80(53.33%) were nonsmokers. Current smokers were 40(26.67%). 20% of the patients have stopped smoking. Duration of smoking incurrent smoker ranges from 1-23 years. All patients were treated with anti-histaminics, steroids, antibiotics bronchodilators meter dose inhaler pumps on and off frequently. Majority of the patients used inhaled steroids 60(40%) followed by bronchodilators 48 (32%). Pulmonary function test were done in 150 patients and results were in favour of obstructive lung pathology. All the patients were subjected to chest x-ray, sputum examination for acid fast bacilli by Zeil nelson stain. Out of total 150 patients 6 patients were found positive for acid-fast bacilli on ZN stain. Only TWO X-ray were suspecting koch's out of 150 patients.

Conclusion: All chronic asthmatics patients should be screened for AFBby Zn stain.

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Introduction

Pulmonary tuberculosis (TB) and asthma are two prevalent respiratory conditions that pose significant health burdens globally. While each condition presents distinct pathophysiological features and management challenges, the coexistence of TB in patients with chronic asthma represents a complex clinical scenario. This intersection not only complicates diagnosis and treatment but also impacts disease outcomes and patient quality of life. TB remains a major global health issue, with an estimated 10 million new cases reported worldwide in 2020 alone. [1-3] Concurrently, asthma affects approximately 334 million individuals globally, with prevalence rates varying across regions and age groups. The geographical overlap of TB and asthma prevalence underscores the importance of

understanding their interplay and the implications for public health interventions. Asthma is characterized by chronic airway inflammation, bronchial hyperresponsiveness, and reversible airflow obstruction. These underlying pathophysiological mechanisms not only predispose individuals to respiratory symptoms but also influence immune responses. [4-6] In asthmatic patients, alterations in innate and adaptive immunity, such as impaired macrophage function and Th2-mediated inflammation, may potentially increase susceptibility to TB infection and alter disease progression. TB, caused by *Mycobacterium tuberculosis*, primarily affects the lungs and can lead to a spectrum of disease manifestations from latent infection to active pulmonary disease. The intricate

interactions between TB bacilli and the host immune system are critical in determining the course and outcome of TB infection. In asthmatic individuals, compromised airway defenses and chronic inflammation may provide a conducive environment for TB bacilli to establish infection and evade host immune responses. [7-10] Diagnosing TB in chronic asthmatic patients presents several challenges due to overlapping clinical features such as chronic cough, dyspnea, and chest discomfort. Moreover, the immunosuppressive effects of corticosteroid therapy commonly used in asthma management can obscure TB symptoms and delay diagnosis. Differential diagnostic approaches, including advanced imaging modalities and molecular diagnostics, are often required to distinguish TB from asthma exacerbations or other respiratory comorbidities. The management of TB in chronic asthmatic patients requires a multidisciplinary approach tailored to address both conditions effectively. Therapeutic decisions must carefully balance the need for anti-TB medications with asthma control strategies, considering potential drug interactions and adverse effects. Concurrent management involves optimizing asthma therapy to reduce airway inflammation and improve lung function while ensuring adherence to anti-TB treatment to achieve cure and prevent disease transmission. [11,12]

Material and Methods

This study was conducted in the Department of Microbiology, Nalanda Medical College and Hospital Patna, Bihar, India, from February 2018 to January 2019. Study population was 150 asthmatic patients who were taking treatment for more than 2 years. Patients with history of asthma for more than five years and taking treatment were included in this study. Patients with history of asthma less than 2 years and Patients not willing to participate in the study were excluded from the study. Study was approved by ethical committee of the institute. A valid written consent was taken from the patients after explaining study to them.

Data was collected with pre tested questionnaire. Data included demographic data like age, sex and occupation. Detailed clinical history was taken from the patients. Clinical history related to duration of asthma, treatment taking for asthma, frequency of asthmatic attacks, past history of tuberculosis, family history of asthma was noted. A thorough clinical examination of the patients was done. All these patients were treated with antihistaminics, steroids, antibiotics bronchodilators meter dose inhaler pumps on and off frequently. Routine investigations like complete blood count, renal and liver function tests were done. ESR is the main haematological investigation done but is found to be inconclusive as the patients are treated with steroids. All the patients

were subjected to chest x-ray, sputum examination for acid-fast bacilli by Zeil nelson stain. Data was subjected to statistical analysis using.

Statistical package for social sciences (SPSS v 21.0, IBM). Descriptive statistics like frequencies and percentage for categorical data, Mean and SD for numerical data has been depicted.

Results

Table 1 shows distribution of patients according to age group. Majority of the patients were from the age group of 31-40 years (30%) followed by 41-50 years (20.67%). Patients in the age group of 51-60 were 18.67%. 18-30 years age group patients contributed 17.33%. Patients above 60 years were 13.33%. Mean age of the patients was 38.5 ± 3.1 years. Majority of the patients in our study were males 95 (63.33%). Female population in our study was 55 (36.67%). Male to female ratio was 1.73:1. Socioeconomic status of the patient was determined by using Kuppaswamy scale of socioeconomic status. Majority of the patients (40.67%) belonged to lower middle class followed by upper lower class (28%). Patients in upper middle class were 14%. Patients in extremes of the class i.e upper class and lower class were 2% and 15.33% respectively. (table 2) In our study out of 150 patients 130 patients were outside workers and 20 females were housewife. In our study, we enquired about the smoking habit in the patients. Out of 150 patients, 80 (53.33%) were non smokers. Current smokers were 40 (26.67%). 20% of the patients have stopped smoking. Duration of smoking in current smoker ranges from 1-23 years. (table 3) All patients were treated with antihistaminics, steroids, antibiotics bronchodilators meter dose inhaler pumps on and off frequently. Majority of the patients used inhaled steroids 60 (40%) followed by bronchodilators 48 (32%). Pulmonary function test were done in 150 patients and results were in favour of obstructive lung pathology. All the patients were subjected to chest x-ray, sputum examination for acid-fast bacilli by Zeil nelson stain. Out of total 150 patients 6 patients were found positive for acid-fast bacilli on ZN stain. Only TWO X-ray were suspecting koch's out of 150 patients. Thus the incidence of pulmonary tuberculosis in our study was 4%. Among the positive patients one was female and 5 were male. Regarding the smoking habits, the female was non smoker. Among the remaining 5 patients 4 were current smokers with duration of smoking ranging from 2-17 years. One patient was past smoker, he left smoking 3 years back. Among the positive patients all were using inhaled steroids. Mean duration of use of inhaled steroid was 9.41 ± 2.7 years. Due to unavailability of AFB culture facility and Gene expert study we were not able to talk about MDR cases in this study.

Table 1: Distribution of Patients According to Age Group

Age Group	Percentage (%)
18-30 years	17.33
31-40 years	30.00
41-50 years	20.67
51-60 years	18.67
>60 years	13.33
Mean Age	38.5 ± 3.1

Table 2: Distribution of Patients According to Gender and Socioeconomic Status

Gender	Number of Patients	Percentage (%)
Male	95	63.33
Female	55	36.67
Socioeconomic Status		
Lower Class I (Lower)	23	15.33
Lower Class II (Upper Lower)	42	28.00
Lower Class III (Lower Middle)	61	40.67
Upper Class I (Upper)	3	2.00
Upper Class II (Upper Middle)	21	14.00

Table 3: Smoking Habits among Patients

Smoking Habit	Number of Patients	Percentage (%)
Non-smokers	80	53.33
Current Smokers	40	26.67
Past Smokers	30	20.00
Duration of Smoking (Current Smokers)		
1-5 years	15	
6-10 years	10	
11-15 years	8	
16-20 years	5	
>20 years	2	

Table 4: Treatment Modalities Used in the Study

Treatment Modality	Number of Patients	Percentage (%)
Antihistaminics	150	100.00
Steroids	150	100.00
Antibiotics	150	100.00
Bronchodilators	48	32.00
Meter Dose Inhaler Pumps	60	40.00

Table 5: Incidence of Pulmonary Tuberculosis in Patients with Chronic Asthma

Pulmonary Tuberculosis Status	Number of Patients	Percentage (%)
Positive	6	4.00
Negative	144	96.00

Discussion

In our study, Majority of the patients were from the age group of 31-40 years (30%) followed by 41-50 years(20.67%). Mean age of the patients was 38.5± 3.1 years. Similar to our study, Lee *et al.* observed the mean age of the COPD cases as 54.5 ± 22.9 years. [13-15] Majority of the patients in our study were males 95(63.33%). Female population in our

study was 55 (36.67%). Male to female ratio was 1.73:1. Similar to our study Lee *et al.* observed a male–female ratio of 1.6:1. ⁸ We found that, majority of the patients (40.67%) belonged to lower middle class followed by upper lower class (28%). The relationships between socio-economic status and respiratory diseases like COPD, tuberculosis is proved in previous studies. [16] Out of total 150

patients 6 patients were found positive for acid-fast bacilli on ZN stain. Thus the incidence of pulmonary tuberculosis in our study was 4%. In a study by Popescu *et al.*, where in 90% of the patients, bronchial asthma developed after tuberculosis and 10% patients presented with bronchial asthma and subsequently developed pulmonary TB. [17] In our study, Out of 150 patients, 80(53.33%) were non smokers. Current smokers were 40(26.67%). 20% of the patients have stopped smoking. Duration of smoking in current smoker ranges from 1-23 years. Among the positive patients 4 were current smokers with duration of smoking ranging from 2-17 years. One patient was past smoker. Cigarette smoking also increases the risk of developing TB by 3–5 folds. [18-19] Smoking suppresses the innate and adaptive immune response with decreased levels of pro-inflammatory cytokines and circulating immunoglobulins and reduces the activity of alveolar macrophages, dendritic cells, and natural killer cells. [20-21] Majority of the patients used inhaled steroids 60(40%) followed by bronchodilators 48 (32%). Among the positive patients all were using inhaled steroids. Mean duration of use of inhaled steroid was $9.41 \pm$ years. In a study on patients with inhaled corticosteroids researches observed that Multivariate Cox regression showed ICS use was an independent risk factor for the occurrence of pulmonary TB in patients who had an abnormal chest radiograph. (hazard ratio, 9.079; 95% CI, 1.012-81.431; P = .049) [22] There are many mechanisms by which steroids can increase the risk of tuberculosis. Steroids have profound effects on the cellular immune response. Glucocorticoids inhibit the lymphokine effect and monocyte chemotaxis and also block Fc receptor binding and function. [23-25] Glucocorticoids decrease the number of peripheral blood monocytes as well as monocyte functions it leads to decreased bactericidal activity and production of interleukin-1 and TNF- α . [26] Glucocorticoids also inhibit T cell activation, leading to reduced proliferative responses and cytokine production, and they also induce a redistribution of lymphocytes (predominantly T cells) out of the circulation, leading to peripheral lymphocytopenia. [27] These various effects of glucocorticoids on the cellular immune system may play a significant role in predisposing to tuberculosis infection.

Conclusion

All chronic asthmatics patients should be screened for AFB by Zn stain.

References

1. World Health Organization. Global Tuberculosis Report 2020. Geneva: WHO; 2020.
2. Global Initiative for Asthma (GINA). Global Strategy for Asthma Management and

- Prevention, 2022. Available from: <http://ginathma.org/>.
3. Holgate ST. Pathogenesis of asthma. *Clin Exp Allergy*. 2008;38(6):872-897.
4. Wenzel SE. Asthma: defining of the persistent adult phenotypes. *Lancet*. 2006;368(9537):804-813.
5. Lönnroth K, Castro KG, Chakaya JM, et al. Tuberculosis control and elimination 2010-50: cure, care, and social development. *Lancet*. 2010;375(9728):1814-1829.
6. Nahid P, Dorman SE, Alipanah N, et al. Official American Thoracic Society/Centers for Disease Control and Prevention/Infectious Diseases Society of America Clinical Practice Guidelines: Treatment of Drug-Susceptible Tuberculosis. *Clin Infect Dis*. 2016;63(7).
7. Aditi A, Sushil K. Tuberculosis and asthma: an overview. *Tuberculosis Research and Treatment*. 2012;1(1):1-5.
8. Lee CH, Lee MC, Star CC, Lim CS, Wang JY, Lee LN, *et al.*. Risk factors for pulmonary tuberculosis in patients with chronic obstructive airway disease in Taiwan: A nationwide cohort study. *BMC Infect Dis* 2013;13:194-6.
9. Aktogu S, Yorgancioglu A, Cirak K, Köse T, Dereli SM. Clinical spectrum of pulmonary and pleural tuberculosis: A report of 5,480 cases. *Eur Respir J* 1996;9:2031-5.
10. Didilescu C, Ibraim E, Ploeanu D. A study of the risk factors for relapse in pulmonary tuberculosis patients and the results of the re-treatment. *Pneumologia* 2000;49:247-52.
11. Wang JY, Lee LN, Hsueh PR. Factors changing the manifestation of pulmonary tuberculosis. *Int J Tuberc Lung Dis* 2005;9: 777-83.
12. Rizvi N, Shah RH, Inayat N, Hussain N. Differences in clinical presentation of pulmonary tuberculosis in association with age. *J Pak Med Assoc* 2003;53:321-4.
13. Yakar HI, Gunen H, Pehlivan E, Aydogan S. The role of tuberculosis in COPD. *Int J Chron Obstruct Pulmon Dis* 2017;12:323-9.
14. Shprykov AS, Zhadnov VZ. Effects of tobacco smoking on the course of infiltrative pulmonary tuberculosis and effectiveness of its treatment. *Probl Tuberk* 1994;5:26-7.
15. Leung CC, Yew WW, Chan CK, Tam CM, Lam CW, Chang KC, *et al.*. Smoking and tuberculosis in HongKong. *Int J Tuberc Lung Dis* 2003;7:980-6
16. Smit KR. National Burden of disease in India from indoor air pollution. 2000. *Proc Natl Acad Sci USA*. Nov 21, 97(24). 13286-93.
17. Popescu C, Gheorghiu T, Russu R, Sepeanu S. Asthma and tuberculosis (considerations based on hospital case studies). *Rev Ig Bacteriol Virusol Parazitol Epidemiol Pneumoftiziol*. 1978;27:23-8.
18. Davies PD, Yew WW, Ganguly D, Davidow

- AL, Reichman LB, Dheda K, *et al.*. Smoking and tuberculosis: The epidemiological association and immunopathogenesis. *Trans R Soc Trop Med Hyg* 2006;100:291-8.
19. Bates MN, Khalakdina A, Pai M, Chang L, Lessa F, Smith KR. Risk of tuberculosis from exposure to tobacco smoke: A systematic review and meta-analysis. *Arch Intern Med* 2007;167:335-42.
20. Arcavi L, Benowitz NL. Cigarette smoking and infection. *Arch Intern Med* 2004;164: 2206-16.
21. Pai M, Mohan A, Dheda K, Leung CC, Yew WW, Christopher DJ, *et al.*. Lethal interaction: The colliding epidemics of tobacco and tuberculosis. *Expert Rev Anti Infect Ther* 2007;5:385-91.
22. Jung-Hyun Kim MD, Soo Park MD Kyung-Ho Kim MD Hye-Cheon Jeong MD Eun-Kyung Kim MD Hyun Lee MD. Inhaled Corticosteroid Is Associated With an Increased Risk of TB in Patients With COPD. *CHEST*. Volume 143, Issue 4, April 2013, Pages 1018-1024
23. Orme IM, Andersen P, Boom WH. T cell response to *Mycobacterium tuberculosis*. *J Infect Dis* 1993;167:1481-97.
24. Balow JE, Rosenthal AS. Glucocorticoid suppression of macrophage migration inhibitory factor. *J Exp Med* 1973;137: 1031-41.
25. Rinehart JJ, Sagone AL, Balcerzak SP, Ackerman GA, LoBuglio AF. Effects of corticosteroid therapy on human monocyte function. *N Engl J Med* 1975;292:236 - 41.
26. Segal BH, Sneller MC. Infectious complications of immunosuppressive therapy in patients with rheumatic diseases. *Rheum Dis Clin North Am* 1997;23:219 -37. Fauci AS, Dale DC, Balow JE. Glucocorticosteroid therapy: mechanisms of action and clinical considerations. *Ann Intern Med* 1976;84:304.