



Original Research Article

DETERMINANTS ASSOCIATED WITH PULMONARY CAVITATION IN TUBERCULOSIS

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ABSTRACT

Background: Tuberculosis, which is caused by *Mycobacterium tuberculosis*, remains a major public health threat worldwide. Pulmonary cavitation is one of the frequently observed clinical characteristics in tuberculosis. **Aims & objectives:** To evaluate the association between pulmonary cavitation in tuberculosis with clinical aspects, lifestyle habits like smoking, alcohol consumption and haematological parameters including hemoglobin count, white blood cell (WBC) count, Neutrophil to lymphocyte ratio (NLR) and platelet to lymphocyte ratio (PLR).

Materials and Methods: Cross sectional analytical study of 43 patients with sputum CBNAAT positive pulmonary tuberculosis admitted at Government Hospital for Chest and Communicable Diseases/ Andhra Medical College, Visakhapatnam between July to November 2019.

Results: For this research, we considered a sample of 43 TB patients. 19 (44.2%) had pulmonary cavitation on chest X-ray, while 24 (55.8%) had no cavitation. In the analysis, statistically significant associations were observed between pulmonary cavitation and history of smoking (p value 1.531×10^{-9}), non alcohol consumption (p value 1.651×10^{-8}), low haemoglobin (p value 0.017), and high NLR (p value 0.0007). There was no significant association between pulmonary cavitation and other parameters studied.

Conclusion: Pulmonary cavitation in tuberculosis was significantly associated with smoking, higher NLR, and lower haemoglobin, with smoking showing the strongest association. These factors may help identify patients at higher risk of cavitary disease. The findings are associative and require confirmation in larger prospective studies.

Keywords: Pulmonary tuberculosis, pulmonary cavitation, Haemoglobin, NLR.

INTRODUCTION

Tuberculosis (TB), which is caused by the *Mycobacterium tuberculosis* (MTB) complex, remains a major public health threat worldwide. According to the Global tuberculosis report 2025, tuberculosis affected 10.7 million people globally in the year 2024.^[1] Incidence of tuberculosis in India during the same year was 196 cases per lakh

population. It can affect either pulmonary or extra pulmonary sites in the body, of which pulmonary tuberculosis is the most common.

After a person is exposed to the tubercle bacilli via inhalation, the innate and acquired immune responses of the individual tries to eliminate the pathogen. In some people, the bacilli may not be totally eliminated and lead to tuberculosis infection. A wide spectrum of clinical presentations are seen in those with

tuberculosis infection. In a vast majority of people, it manifests as latent TB infection, which is clinically asymptomatic and non contagious. In some others, it can manifest as clinically symptomatic active tuberculosis disease. The classic innate immune response to tuberculosis lead to caseating granuloma formation in the lung, which consists of central necrotic core, macrophages, epitheloid cells, multinucleated giant cells, neutrophils, surrounded by lymphocytes and fibroblasts. Adaptive immunity to tuberculosis bacilli is mediated by mainly T lymphocytes, which releases cytokines that mediate inflammation.

Lung cavitation in tuberculosis occurs due to erosion of necrotic core of granuloma to an airway. The cavities serve as a place for rapid bacillary growth and increased environmental transmission of tuberculosis. Penetration of immune cells into the necrotic core is limited, leading to increased bacterial replication within. As the oxygen level inside the cavity is high, a nutrient rich niche is provided for bacillary growth. Poor vascularity of the center of cavity hampers the influx of anti tubercular drugs. Faster replication of the mycobacterium can lead to mutations that drive drug resistance. With successful treatment, the cavities which are eradicated of active bacilli can either heal with fibrosis or can remain as open cavity. This can further lead on to chronic lung damage and high chance of opportunistic infections.^[2] Lung cavitation in tuberculosis a result of complex process involving immunologic responses and microbiological properties of Mycobacterium tuberculosis.^[3]

The current study aims to evaluate the determinants of pulmonary cavitation in tuberculosis with special emphasis on the clinical aspects, lifestyle habits like

smoking, alcohol consumption and haematological parameters.

MATERIALS AND METHODS

Type of Study: Cross sectional analytical study

Place of Study: Government Hospital for Chest and Communicable Diseases/ Andhra Medical College, Visakhapatnam.

Study Duration: From July 2019 to November 2019

Sample Size: The study included 43 patients with active pulmonary tuberculosis who were sputum CBNAAT positive

Inclusion Criteria

- Newly diagnosed Sputum CBNAAT positive Pulmonary Tuberculosis

Exclusion Criteria

- Patients with associated bacterial pneumonia
- Extra pulmonary tuberculosis

Study Variables

Age, Gender, Body Mass Index(BMI), Duration of symptoms, previous pulmonary tuberculosis history, Hypertension, Diabetes Mellitus, Smoking , alcohol consumption, Haemoglobin, Total WBC count, Neutrophil to lymphocyte ratio (NLR), platelet to lymphocyte ratio (PLR)

Statistical Analysis

Data were entered in Excel spreadsheet and subsequently analysed using R statistical software. Pulmonary cavitation was considered as the response variable. The association with other variables were statistically modelled by using a penalised maximum likelihood logistic regression model. A p-value of ≤ 0.05 was considered statistically significant.

RESULTS

Table 1: The distribution of variables between patients with pulmonary cavitation and those without cavitation

Variable	Category	Cavitation present	Cavitation absent
Age (years)	Mean	36.74	43.04
Sex	Male	14	8
	Female	5	16
BMI (kg/m ²)	Mean	14.74	17.52
Duration of symptoms (days)	Mean	33.84	34.11
Previous history of Tb	Present	10	7
	Absent	9	17
Diabetes Mellitus	Present	2	6
	Absent	17	18
Hypertension	Present	2	3
	Absent	17	21
Other comorbidities	Present	1	3
	Absent	18	21
Smoking	Present	7	5
	Absent	12	19
Alcohol consumption	Present	4	6
	Absent	15	18
HIV	Present	1	2
	Absent	18	22
Haemoglobin (gm/dl)	Mean	7.77	8
WBC Count (cells/mm ³)	Mean	9221.05	8826.09
NLR	Mean	3.82	3.02
PLR	Mean	161.70	125.52

Table 2: Results of the logistic regression model

Variable	β Coefficient	Standard error	p-value	
Age	-0.526	1.017		
Sex	1.38	1.93		
BMI	-1.065	0.946		
Duration of symptoms	0.029	0.698		
Previous history of Tb	-0.309	1.809		
Diabetes Mellitus	-0.611	1.79		
Hypertension	0.996	2.272		
Other comorbidities	-0.766	2.020		
Smoking	2.309	2.70	1.531×10^{-9}	***
Alcohol consumption	-2.799	2.522	1.651×10^{-8}	***
Haemoglobin	-0.255	0.695	0.017	*
WBC Count	-0.126	1.26	0.106	
NLR	0.696	2.009	0.0007	***
PLR	-0.072	1.138	0.065	

Table 3: Odds ratio of variables with significant association with cavity formation

Variable	Odd's ratio
Smoking	10.07
Alcohol consumption	0.06
Haemoglobin	0.77
NLR	2.006

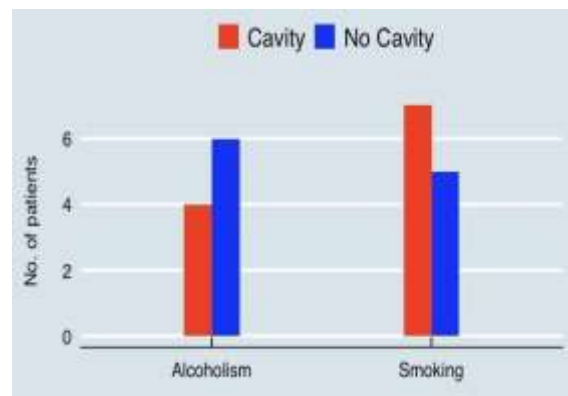


Figure 1: Alcohol and Smoking characteristics among the study group

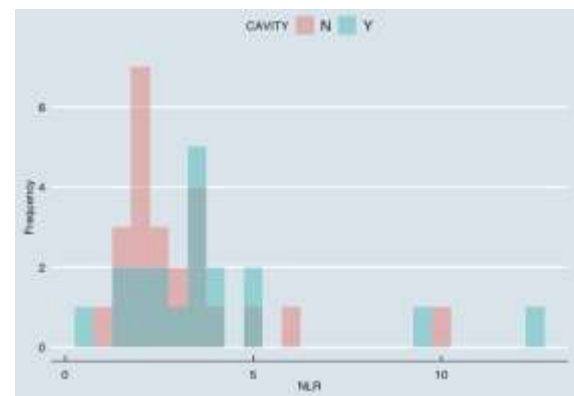


Figure 3: Category-wise distribution of Neutrophil to lymphocyte ratio

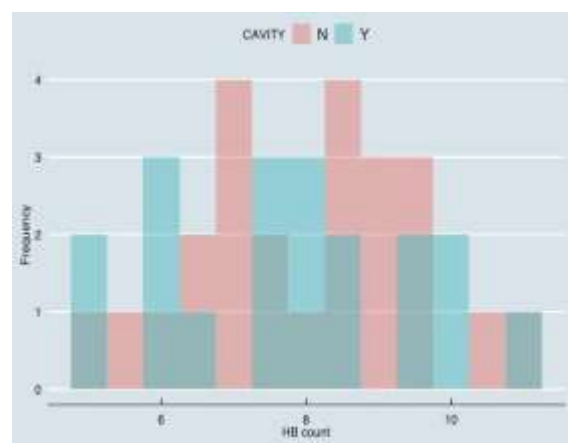


Figure 2: Category-wise distribution of Haemoglobin(gm/dl)

For this research, we considered a sample of 43 TB patients. 19 patients (44.18%) had cavitation on chest X-ray and 24(55.81%) did not have cavities. The age of the study population varied between 10 to 75 years with a mean age of 41 years. Of the total number of patients, 22 (51.16%) were males and 21(48.83%) were females. The BMI of the study population ranged from 9.98 kg/m² to 21.33 kg/m², with a mean of 15.91 kg/m². The sample included 12(27.9%) smokers and 10(23.25%) patients with history of alcohol consumption. The distribution of smoking and alcohol consumption among the study participants is shown in Figure 1. Duration of symptoms at the time of presentation varied from 4 days to 120 days with a mean of 35 days. 17 patients (39.53%) had a previous history of pulmonary tuberculosis. The data included 8(18.6%) diabetics, 5(11.6%) hypertensives and 4 patients had other comorbidities. 3 patients in the sample(6.9%) were diagnosed with HIV. The haemoglobin of patients ranged between 4.8 gm/dl to 11.100 gm/dl with a mean of 7.916 gm/dl. The category-wise distribution of haemoglobin levels is shown in Figure 2. The total leukocyte count varied between 3600 cells/mm³ to 23,000 cells/mm³, with a mean of 9191.59 cells/

mm3. The NLR of the sample ranged from 0.7288 to 12.5714, with a mean of 3.3834. The category-wise distribution of NLR is shown in Figure 3. The PLR of the sample ranged from 2.15 to 566.16, with a mean of 137.96. The distribution of variables between patients with cavitation and those without cavitation is illustrated in table 1.

Pulmonary cavitation was considered the binary response variable, with age, sex, body mass index (BMI), duration of symptoms, previous history of pulmonary tuberculosis, diabetes mellitus, hypertension, other comorbidities, smoking, alcohol consumption, haemoglobin, total WBC count, NLR, and PLR included as explanatory variables. A penalised maximum likelihood logistic regression model was fitted to identify variables significantly associated with pulmonary cavitation. The regression coefficients (β) and corresponding p-values are presented in Table 2. Statistically significant associations with pulmonary cavitation were observed for alcohol consumption ($p = 1.651 \times 10^{-8}$), smoking ($p = 1.531 \times 10^{-9}$), haemoglobin ($p = 0.017$), and NLR ($p = 0.0007$). No statistically significant association was observed with age, sex, BMI, duration of symptoms, previous history of pulmonary tuberculosis, diabetes mellitus, hypertension, other comorbidities, total WBC count, or PLR.

Odds ratios (ORs), obtained by exponentiating the regression coefficients, were subsequently calculated to quantify the magnitude and direction of these associations (Table 3). Smoking was positively associated with pulmonary cavitation (OR = 10.07), with smokers having approximately 10-fold higher odds of cavitation compared with non-smokers. NLR was also positively associated with cavitation (OR = 2.01). Conversely, alcohol consumption (OR = 0.06) and haemoglobin level (OR = 0.77) were inversely associated with pulmonary cavitation.

DISCUSSION

The significant variables from the logistic regression can be expected to show the same type of association in the population as they are exhibiting in the sample. The model only establishes associations and not causality. The significant variables must be interpreted clinically and based on existing medical theory to figure out causality.

A history of smoking showed a particularly strong association, with smokers having approximately 10-fold higher odds of cavitation compared with non-smokers. A review article by Silva DR et al. mentions data from existing literature establishing increased risk of pulmonary tuberculosis in those with history of smoking.^[4] Susceptibility to tuberculosis is increased due to ciliary dysfunction and reduced immune responses, thus preventing elimination of the bacilli. Smoking reduces production of TNF alpha by lung macrophages, thus preventing effective granuloma formation and containment of infection. Previous studies by Song M et al. and Altet-Gomez

MN et al. have demonstrated an increased likelihood of cavitation among pulmonary tuberculosis patients with a history of smoking, which is similar to the findings of the present study.^[5,6]

In the present study, alcohol consumption was significantly associated with lower odds of pulmonary cavitation among tuberculosis patients. This finding is unexpected, as previous studies including those by Silva DR et al. and Kan CK et al. have reported either no significant association or an increased frequency of cavitation among patients with alcohol use.^[4,7] A recent study by Tripathi D et al. on a mice model for the effect of alcohol on tuberculosis demonstrated enhanced production of interferon alpha, leading to impaired macrophage immune response and increased mortality.^[8] The observed inverse association should therefore not be interpreted as a protective effect of alcoholism. Differences in patient characteristics, definition and severity of alcohol use, radiological assessment, and residual confounding by factors such as smoking, diabetes, nutritional status, or disease severity may partly explain this finding. A small number of patients in relevant subgroups may also have resulted in an unstable estimate. Further studies with larger sample sizes and standardized assessment of alcohol use are needed to clarify this association.

Lower haemoglobin levels were associated with increased odds of cavitation, with each unit increase in haemoglobin corresponding to a 23% reduction in the odds of cavity formation. This finding suggests that anaemia or lower haemoglobin levels may be an important marker associated with pulmonary cavitation. According to Gil-Santana L et al. anemia is seen in around 32% to 86% patients with tuberculosis.^[9] The reason behind it can be anemia of chronic disease due to inflammation, nutritional causes like iron deficiency anemia or due to suppression of bone marrow by tuberculosis. Study Song M et al. report anemia as an independent risk factor for tubercular cavity formation.^[5] Study by Luo M et al. showed association of anemia with higher bacillary load in sputum, extensive lung damage and delayed closure of pulmonary cavitation with anti tubercular treatment when compared to those without anemia.^[10]

Higher NLR was significantly associated with cavity formation, with each unit increase in NLR approximately doubling the odds of cavitation. This indicates greater systemic inflammatory activity among patients with lung cavitation. The finding in the current study was consistent with study by Nakao M et al,^[11] and He x et al.^[12] An increased neutrophil-to-lymphocyte ratio (NLR) may reflect a reduction in lymphocyte levels, potentially indicating a diminished lymphocyte-mediated immune response. Macrophages and T lymphocytes are essential in the development of pulmonary cavitation in tuberculosis. Consequently, changes in the relative distribution of circulating lymphocytes may affect the process of cavity formation within the lungs. In this context, the NLR may serve as a simple, readily accessible

indicator of underlying immunological alterations and could potentially be used as a marker for pulmonary cavitation.

The limitations of the present study were as follows: small sample size, cross-sectional design, and single facility study.

CONCLUSION

Pulmonary cavitation in tuberculosis was significantly associated with smoking history, higher NLR, lower haemoglobin levels, and non alcohol consumption. Smoking showed the strongest positive association, with smokers having approximately ten-fold higher odds of cavitation. Higher NLR and lower haemoglobin were also associated with increased odds of cavity formation, suggesting that systemic inflammation and anaemia may be important markers of cavitary disease. The inverse association observed with alcohol consumption was unexpected and should not be interpreted as protective, as it may reflect confounding or differences in patient characteristics.

No significant association was found with age, sex, BMI, symptom duration, previous tuberculosis, diabetes, hypertension, other comorbidities, total WBC count, or PLR. Overall, smoking, NLR, and haemoglobin may help identify patients at higher risk of pulmonary cavitation. However, these findings represent associations rather than causation and require confirmation in larger prospective studies.

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