Relationship Between Host Factors and the Incidence of Pulmonary Tuberculosis in HIV/AIDS Patients at Dr. Pirngadi Medan General Hospital

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Track Record Article

Revised: 13 December 2025
Accepted: 16 March 2026
Published: 31 March 2026

How to cite:
Nasution, A. N., Nasution, N., & Lubis, Y. E. P. (2026). Relationship Between Host Factors and the Incidence of Pulmonary Tuberculosis in HIV/AIDS Patients at Dr. Pirngadi Medan General Hospital. *Contagion: Scientific Periodical Journal of Public Health and Coastal Health*, *8*(1), 555–565.

Abstract

Tuberculosis (TB) remains a major cause of morbidity and mortality among people living with HIV/AIDS. While CD4 count is a standard clinical indicator, inexpensive hematological indices such as the neutrophil-to-lymphocyte ratio (NLR), monocyte-to-lymphocyte ratio (MLR), and systemic immune-inflammation index (SII) have been proposed as practical markers of immune status. This study examined the association between host factors (sociodemographics, CD4 count, and clinical stage) and hematological indices (NLR, MLR, and SII) with pulmonary TB among HIV/AIDS patients at Dr. Pirngadi Medan General Hospital. An analytical cross-sectional design with a retrospective approach was applied using secondary data from medical records of patients registered between 2022 and 2024. A total of 94 eligible patients were included through total sampling, and data were analyzed using the Chi-square test ($p < 0.05$). The pulmonary TB was identified in 43.6% of patients. CD4 count showed a significant association with TB occurrence ($p = 0.03$), with cases more frequent among patients with CD4 <200 cells/mm³. No significant associations were observed for age, sex, employment, marital status, clinical stage, or hematological indices (NLR, MLR, and SII) ($p > 0.05$). Thus, CD4 count remains the key predictor of pulmonary TB risk, underscoring the importance of intensified TB screening in individuals with advanced immunosuppression. Larger studies are warranted to clarify the potential role of hematological indices in TB-HIV coinfection.

Keywords: HIV/AIDS, Pulmonary Tuberculosis, CD4 Count, NLR, MLR, SII.

INTRODUCTION

TB-HIV/AIDS coinfection represents a complex pathobiological interaction shaped by the dynamics of the host immune responses and the progression of both diseases. HIV-induced immunosuppression increases susceptibility to TB, while TB further accelerates HIV progression by disrupting immune regulation. This bidirectional relationship amplifies disease severity and elevates mortality risk among coinfected individuals with TB-HIV/AIDS. Globally, the burden of TB-HIV coinfection continues to rise, with TB remaining the leading cause of death among people living with HIV/AIDS as of 2023 (Albayrak, 2023; Dong et al., 2024; Fathiyah et al., 2020; Miyahara et al., 2019; Obeagu & Obeagu, 2024; Sulastri et al., 2021).

In Indonesia, the burden of TB-HIV/AIDS remains substantial, with Southeast Asia contributing significantly to global cases. According to the Ministry of Health's SITB report

(2023), there were 15,375 TB-HIV/AIDS cases in 2022, alongside approximately 6,700 TB-related deaths among HIV-positive individuals. In North Sumatra, both TB and HIV/AIDS cases show an upward trend: TB cases rose from 34,717 in 2022 to 49,999 in 2023, while HIV/AIDS cases increased from 2,824 to 3,473 in the same year, with Medan identified as the major contributor (Dinkes Provinsi Sumatera Utara, 2023).

Host factors play a critical role in increasing susceptibility to TB-HIV/AIDS coinfection (Miyahara et al., 2019; Sulastri et al., 2021). HIV/AIDS infection reduces both the number and function of CD4+ cells, which are essential for controlling latent TB (Choi et al., 2024; Obeagu & Obeagu, 2024). This immunosuppression enables *Mycobacterium tuberculosis* to proliferate within granulomas, triggering TB reactivation. Conversely, TB infection can further dysregulate immunity by enhancing HIV replication in activated CD4+ T cells and macrophages, thereby worsening clinical outcomes and impairing host control of *M. Tuberculosis*. Assessing immune-inflammatory status through inexpensive hematological biomarkers, such as Neutrophil-to-Lymphocyte Ratio (NLR), Monocyte-to-Lymphocyte Ratio (MLR), and Systemic Immune-Inflammation Index (SII) is therefore relevant for screening, risk stratification, and monitoring in resource-limited settings. Evidence suggests that NLR tends to increase in TB-HIV/AIDS coinfection and correlates with disease severity, whereas findings for MLR and SII remain inconsistent, underscoring the need for larger multivariate studies to validate their clinical utility (Miyahara et al., 2019; Obeagu & Obeagu, 2024; Sulastri et al., 2021).

Beyond biological determinants, non-biological factors such as age, gender, employment, and marital status also shape access to health services, health-seeking behavior, diagnostic delays, and disease progression. Low CD4 counts and advanced HIV/AIDS clinical stage reflect severe immune compromise, heightening the risk of latent TB reactivation and impaired infection control. Host-directed therapy (HDT) is increasingly explored as an adjunct strategy to correct immune dysfunction in TB-HIV/AIDS coinfection, emphasizing the importance of identifying patient subgroups most likely to benefit from HDT interventions (Prasanna et al., 2025; Scully & Bryson, 2021).

Despite growing interest, the roles of NLR, MLR, and SII in TB-HIV/AIDS coinfection remain incompletely understood, with findings inconsistent across studies and limited by reliance on bivariable analyses. This study seeks to examine the relationships between host factors (age, gender, employment status, marital status, CD4 count, HIV/AIDS clinical stage) and hematological parameters (NLR, MLR, and SII) with the occurrence of pulmonary TB among HIV/AIDS patients at RSUD Dr. Pirngadi Medan. By addressing this

gap, particularly within the context of North Sumatra, the study aims to contribute evidence relevant for regional TB-HIV management and research.

METHODS

This study employed an analytical quantitative approach with a retrospective cross-sectional design, utilizing secondary data from medical records of HIV/AIDS patients at Dr. Pirngadi Medan General Hospital. The objective was to examine the association between host factors (age, gender, employment status, marital status, CD4 count, and HIV/AIDS clinical stage) and hematological inflammatory indices (neutrophil-to-lymphocyte ratio (NLR), monocyte-to-lymphocyte ratio (MLR), and systemic immune-inflammation index (SII), with the occurrence of pulmonary tuberculosis (TB) among HIV/AIDS patients.

The study was conducted between August and December 2025 at Dr. Pirngadi Medan General Hospital. Data were collected from the Medical Records Department and the Voluntary Counseling and Testing (VCT) Clinic. The study population comprised all HIV/AIDS patients registered and managed at the hospital during 2022–2024, with a total sampling applied. Inclusion criteria were HIV/AIDS patients aged **≥1**8 years with documented CD4 counts and complete hematological data (neutrophils, monocytes, and lymphocytes). Exclusion criteria included medical records lacking clinical staging information or patients diagnosed solely with extrapulmonary TB. Following eligibility screening, the final sample consisted of 94 patients (n = 94).

Data extracted included demographic variables (age, gender, employment status, and marital status), clinical variables (HIV/AIDS clinical stage and CD4 count), laboratory parameters required to calculate NLR, MLR, and SII, and pulmonary TB status. The dependent variable was pulmonary TB occurrence, categorized as present or absent based on documented diagnoses in medical records. Independent variables comprised age, gender, employment status, marital status, CD4 count, HIV/AIDS clinical stage, and the hematological indices (NLR, MLR, and SII). Following extraction, data cleaning was performed and NLR, MLR, and SII were calculated from hematology results using standard formulas.

Statistical analyses were performed using SPSS version 29. Univariate analysis summarized participant characteristics with frequencies and percentages. Bivariate associations between independent variables and pulmonary TB occurrence were assessed using Chi-square. Variables with a p-value below the predefined threshold were subsequently entered into multivariate analysis using binary logistic regression to identify dominant predictors of pulmonary TB. The final model reported adjusted odds ratios (AORs) with 95% confidence intervals (CIs), with statistical significance set at $p < 0.05$. This study is expected to generate

evidence on key determinants of pulmonary TB among HIV/AIDS patients and inform risk-based screening and prevention strategies in hospital-based HIV care.

RESULTS

Respondent Characteristics

Table 1. Characteristics of Respondents (n=94)

Characterstics	n=94	%
Age		
< 40 years	66	70.2
≥ 40 years	28	29.8
Gender		
Male	86	91.5
Female	8	8.5
Marital status		
Married	25	26.6
Unmarried	69	73.4
Occupation		
Employed	68	72.3
Unemployed	26	27.7
CD4 Count		
<200	55	58.5
>=200	39	41.5
HIV/AIDS Clinical Stage		
Early stage	17	18.1
Late stage	77	81.9
NLR (Neutrophil-to-Lymphocyte Ratio)		
<2	25	26.6
>=2	69	73.4
MLR (Monocyte-to-Lymphocyte Ratio)		
<0.23	12	12.8
>=0.23	82	87.2
SII (Systemic Immune-Inflammation Index)		
<562.3	45	47.9
>=562.3	49	52.1
Pulmonary TB Status		
No TB	53	56.4
Pulmonary TB	41	43.6

The study included 94 respondents, whose characteristics were analyzed across demographic, clinical, and laboratory variables. Most respondents were under 40 years of age, accounting for 66 individuals (70.2%), while 28 respondents (29.8%) were 40 years or older. The sample was predominantly male, with 86 men (91.5%) compared to 8 women (8.5%).

Looking at marital status, most participants were unmarried, accounting for 69 respondents (73.4%), while 25 respondents (26.6%) were married. In terms of occupation, the majority were employed, totaling 68 individuals (72.3%), while 26 respondents (27.7%) were unemployed. For CD4 count, which is a key marker of immune function in HIV/AIDS patients, 55 respondents (58.5%) had counts below 200 cells/mm³, while 39 respondents (41.5%) had counts of 200 cells/mm³ or higher. With respect to HIV/AIDS clinical stage, a large proportion

of the respondents were in the late stage, comprising 77 individuals (81.9%), while 17 respondents (18.1%) were classified in the early stage.

Laboratory measures of immune function showed that 69 respondents (73.4%) had a Neutrophil-to-Lymphocyte Ratio (NLR) of 2 or higher, while 25 respondents (26.6%) had an NLR of less than 2. For the Monocyte-to-Lymphocyte Ratio (MLR), 82 respondents (87.2%) had an MLR of 0.23 or higher, and only 12 respondents (12.8%) had an MLR of less than 0.23. Regarding the Systemic Immune-Inflammation Index (SII), 49 respondents (52.1%) had an SII of 562.3 or higher, while 45 respondents (47.9%) had an SII of less than 562.3. Finally, concerning pulmonary tuberculosis (TB) status, 41 respondents (43.6%) were diagnosed with pulmonary TB, while the remaining 53 respondents (56.4%) did not have TB.

Bivariate Analysis

Table 2. Relationship Between Host Factors And The Incidence Of Pulmonary Tuberculosis In Hiv/Aids Patients At Dr. Pirngadi Medan General Hospital

Variable	Clinical TB				Total		*P-Value*
	No pulmonary TB		Pulmonary TB				
	n	%	n	%	n	%	
Age Group							
<40 years	36	38.3	30	31.9	66	70.2	**0.58**
≥ 40 years	17	18.1	11	11.7	28	29.8	
Gender							
Male	51	54.3	35	37.2	86	91.5	**0.07**
Female	2	2.1	6	6.4	8	8.5%	
Job							
Employed	40	42.6	28	29.8	68	72.3	**0.44**
Unemployed	13	13.8	13	13.8	26	27.7	
Marital Status							
Married	13	13.8	12	12.8	25	26.6	**0.60**
Unmarried	40	42.6	29	30.9	69	73.4	
CD4 count							
<200	26	27.7	29	30.9	55	58.5	**0.03**
>= 200	27	28.7	12	12.8	39	41.5	
HIV/AIDS Stage							
Early stage	11	11.7	6	6.4	17	18.1	**0.44**
Advanced stage	42	44.7	35	37.2	77	81.9	
Examination results NLR							
<2	15	16.0	10	10.6	25	26.6	**0.67**
>=2	38	40.4	31	33.0	69	73.4	
Examination results MLR							
<0.23	5	5.3	7	7.4	12	12.8	**0.27**
>=0.23	48	51.1	34	36.2	82	87.2	
Examination results MLR							
<562.3	26	27.7	19	20.2	45	47.9	**0.79**
>=562.3	27	28.7	22	23.4	49	52.1	
Total	53	56.4	41	43.6	94	100.0	

Table 2. Based on the study of 94 HIV/AIDS patients at Dr. Pirngadi General Hospital. Medan. the incidence rate of pulmonary TB coinfection was 43.6% (41 patients). while the remaining 56.4% (53 patients) did not present with pulmonary TB. Demographic and clinical characteristics indicated that the majority of respondents were under 40 years of age (70.2%). male (91.5%). employed (72.3%). and unmarried (73.4%). Additionally. a significant proportion of the patients exhibited severe immunosuppression with a CD4 cell count of <200 cells/mm³ (58.5%) and were in the advanced stages of HIV/AIDS (81.9%).

Bivariate analysis examining the relationship between host factors and the incidence of pulmonary TB revealed that the CD4 cell count was the only variable with a statistically significant association (p-value = 0.03). The proportion of patients with pulmonary TB was notably higher in the group with a CD4 count of <200 cells/mm³ (30.9% of the total sample) compared to those with a CD4 count of ≥200 cells/mm³ (12.8%). Conversely. other demographic and clinical variables showed no significant relationship with the incidence of pulmonary TB (p-value > 0.05). These variables included age (p = 0.58). gender (p = 0.07). employment status (p = 0.44). marital status (p = 0.60). and HIV/AIDS stage (p = 0.44). Furthermore. systemic inflammatory markers. including the Neutrophil-to-Lymphocyte Ratio (NLR) (p = 0.67). Monocyte-to-Lymphocyte Ratio (MLR) (p = 0.27). and Systemic Immune-inflammation Index (SII) (p = 0.79). also demonstrated no significant association with the occurrence of pulmonary TB among HIV/AIDS patients in this study.

DISCUSSION

Relationship between Immunosuppression Status (CD4 Count) and Incidence of Pulmonary TB

CD4 count was the only host factor found to have a statistically significant association with pulmonary tuberculosis (TB) incidence among HIV/AIDS patients at Dr. Pirngadi General Hospital, Medan (p-value = 0.03). Patients with severe immunosuppression, defined as CD4 counts <200 cells/mm³, exhibited a markedly higher TB coinfection rate (30.9%) compared to those with CD4 counts ≥200 cells/mm³ (12.8%). These findings reaffirm the central role of cellular immunity in controlling *Mycobacterium tuberculosis* (MTB) infection. Pathophysiologically, CD4+ T cells are pivotal in orchestrating the adaptive immune response against intracellular pathogens. When alveolar macrophages phagocytose TB bacilli, CD4+ T cells produce key pro-inflammatory cytokines that activate macrophage bactericidal functions and promote granuloma formation, both of which are essential for containing TB infection.

These findings are consistent with the comprehensive retrospective study by Pranaya et al. (2023), which reported that the majority of HIV patients with pulmonary TB coinfection had

CD4 <200 cells/μL, accounting for 87.88% of the study population. Such profound immunosuppression facilitates the reactivation of latent TB infection while simultaneously weakening host defences against primary exogenous reinfection. The concentration of TB incidence within the CD4 <200 cells/mm³ group underscores the predictive strength of this parameter, establishing it as the most determinative independent marker of TB risk in HIV-infected individuals.

The Role of Demographic and Clinical Factors in TB-HIV Coinfection

Unlike transmission dynamics in the general population, this study found that basic demographic and clinical factors, including age, gender, employment status, marital status, and clinical stage of HIV, were not significantly associated with pulmonary TB coinfection ($p > 0.05$). The insignificance of the age variable ($p = 0.58$) shows that the risk of exposure and reactivation of TB in HIV patients is relatively uniform across age groups. Gender, however, approached marginal significance ($p = 0.07$), with males comprising 91.5% of the sample. This predominance is likely shaped by socio-cultural risk factors, such as greater outdoor exposure and higher prevalence of comorbidities (e.g., smoking), which increase contact with *M. tuberculosis.*

Nevertheless, when these sociodemographic variables are weighed against the profound immune system damage caused by HIV, their influence becomes overshadowed. HIV infection functions as a biological equalizer: regardless of employment status ($p = 0.44$) or age, once HIV destroys CD4+ T cells, vulnerability to TB rises uniformly. This finding aligns with national epidemiological trends reported by Imthiatiyyah et al. (2025) using IFLS 5 survey data in Indonesia, which showed that sociodemographic parameters such as marital status and employment status often lose predictive power when adjusted for clinical variables. Ultimately, immune system failure dictates TB pathogenesis more decisively than host demographic characteristics.

Evaluation of Systemic Inflammatory Markers (NLR, MLR, and SII) in TB-HIV Coinfection

Assessment of systemic inflammatory ratios—namely Neutrophil-to-Lymphocyte Ratio (NLR), Monocyte-to -Lymphocyte Ratio (MLR), and Systemic Immune-inflammation Index (SII)—revealed no statistically significant association with pulmonary TB coinfection (p-value NLR = 0.67; MLR = 0.27; SII = 0.79). In immunocompetent individuals, active TB infection typically provokes a surge in neutrophil and monocyte recruitment accompanied by lymphocyte apoptosis, resulting in elevated NLR and MLR values. In contrast, TB-HIV coinfection produces highly disordered immunological dynamics.

HIV replication drives chronic immune activation, systemic cytokine release, and profound bone marrow suppression, predisposing patients to severe neutropenia and lymphopenia even before TB invasion occurs. Because both numerator and denominator components of these ratios are disrupted by viral destruction, their predictive validity is substantially weakened. This distortion is consistent with findings by Sulastri et al. (2021), who demonstrated that the hematological response and NLR profiles in TB/HIV coinfected patients differ markedly from those with TB alone, where immune exhaustion often obscures pathogen-specific inflammatory signals. The persistent background noise of HIV-related inflammation undermines the specificity of these biomarkers, limiting their utility in identifying TB coinfection.

Clinical Implications and Research Limitations

The finding that severe immunosuppression (CD4 <200 cells/mm^3) is the sole strong predictor of pulmonary TB coinfection underscores the urgent need to strengthen referral and screening systems at Dr. Pirngadi General Hospital in Medan. Hospital policymakers should establish clinical pathways requiring all HIV patients (both newly diagnosed and outpatients) with low CD4 counts to undergo routine TB screening using advanced diagnostic tools such as the GeneXpert Molecular Rapid Test (MRT). Persistent implementation of this strategy is critical, as TB symptoms in people living with HIV (PLHIV) often present atypically and cannot reliably predicted through hematological indices (NLR/MLR/SII). The widespread use of GeneXpert is consistent with clinical urgency highlighted by Siahaan et al. (2024), who demonstrated its superior diagnostic sensitivity and specificity in detecting *Mycobacterium tuberculosis* DNA among vulnerable populations compared to conventional microscopy, thereby reducing false-negatives in paucibacillary TB cases common in HIV patients.

In addition to the urgency of early Isoniazid (TPT) prophylaxis, this study is not without a number of methodological limitations. The cross-sectional study design prevents researchers from establishing definitive causality whether immunosuppression facilitates TB, or TB coinfection accelerates CD4 decline. The limited population at a single health center also leaves the potential for referral bias in disease severity. Prospective cohort studies and multicenter studies are crucial for future research to validate these dynamics.

CONCLUSIONS

Based on the results of research and analysis conducted on HIV/AIDS patients at Dr. Pirngadi General Hospital in Medan, it can be concluded that host immunosuppression status is the main predictor determining the incidence of pulmonary tuberculosis (TB) coinfection. CD4 count was found to be the only variable with a statistically significant relationship, where

severe immunosuppression with a CD4 count below 200 cells/mm³ drastically increased the patient's absolute susceptibility to opportunistic infections. Conversely, basic sociodemographic factors such as age, gender, employment status, and marital status, as well as the clinical stage of HIV, did not show a significant association, indicating that immune system collapse has complete control over disease pathogenesis beyond demographic determinants.

Furthermore, systemic inflammatory marker ratios, including the Neutrophil-to-Lymphocyte Ratio (NLR), Monocyte-to-Lymphocyte Ratio (MLR), and Systemic Immune-inflammation Index (SII), were also not found to have a significant relationship with the incidence of pulmonary TB. This confirms that the overlap of inflammation and chronic immune activation due to HIV replication limits the specificity of these routine hematological parameters, making the strengthening of screening based on Molecular Rapid Tests (MRT) absolutely necessary for patients with low CD4 counts.

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