Section: Microbiology

Original Research Article

RAPID MOLECULAR DETECTION OF RIFAMPICIN-RESISTANT TUBERCULOSIS IN PULMONARY TUBERCULOSIS AND HIV-TB CO-INFECTED PATIENTS: A CB-NAAT BASED CROSS-SECTIONAL ANALYSIS

V. Saiprabha[1], M. Sathiyalakshmi[2], S. Vanathi[3]

[1]Assistant Professor, Department of Microbiology, Government Medical College, Pudukottai, Tamil Nadu, India.
[2]Senior Resident, Sri Ramachandra Institute of Higher Education and Research, Chennai, Tamil Nadu, India.
[3]Associate Professor, Department of Microbiology, Government Medical College Pudukottai, Tamil Nadu, India.

Received : 11/02/2026
Received in revised form : 30/03/2026
Accepted : 16/04/2026

Keywords:
Tuberculosis, Rifampicin resistance, CB-NAAT, HIV-TB co-infection, CD4 count, MDR-TB.

Corresponding Author:
Dr. S. Vanathi,
Email: sv-vanathi@gmail.com

DOI: 10.47009/jamp.2026.8.2.207

Source of Support: Nil,
Conflict of Interest: None declared

Int J Acad Med Pharm
2026; 8 (2); 1137-1142

ABSTRACT

Background: Tuberculosis (TB) remains a major global health problem, particularly in developing countries like India. The emergence of drug-resistant TB, especially rifampicin-resistant TB (RR-TB), poses a serious threat to TB control programs. Rapid molecular diagnostics such as Cartridge-Based Nucleic Acid Amplification Test (CB-NAAT) have significantly improved early detection. The aim is to detect Mycobacterium tuberculosis and rifampicin resistance using CB-NAAT among pulmonary TB patients and to evaluate its association with HIV co-infection and CD4 cell counts. **Materials and Methods:** This analytical cross-sectional study included 190 suspected pulmonary TB patients attending a tertiary care hospital. Sputum samples were tested using CB-NAAT. HIV status was determined using rapid assays, and CD4 counts were analyzed by flow cytometry. Statistical analysis was performed using SPSS version 16. **Result:** Out of 188 confirmed TB cases, rifampicin resistance was observed in 4.3% (8 cases). Among HIV co-infected patients, RR-TB was 9.1%, compared to 4% in non-HIV patients (p=0.389). Female gender showed a significant association with rifampicin resistance (p=0.038). A significant reduction in CD4 count was observed in HIV-TB patients with rifampicin resistance (p<0.0001). **Conclusion:** CB-NAAT is an effective tool for rapid detection of TB and rifampicin resistance. HIV co-infection influences disease severity and immune status. Early diagnosis and integrated TB-HIV management are essential to control drug-resistant TB.

INTRODUCTION

Tuberculosis (TB) remains one of the most significant infectious diseases worldwide and continues to be a major cause of morbidity and mortality, particularly in developing countries such as India.[1] It is caused primarily by Mycobacterium tuberculosis, an intracellular, acid-fast bacillus belonging to the Mycobacterium tuberculosis complex, which includes M. bovis, M. africanum, M. microti, M. pinnipedii, and M. caprae.[2] The disease is transmitted through inhalation of aerosolized droplets containing the bacilli, expelled by individuals with active pulmonary tuberculosis.[3] Once inhaled, the organism primarily targets the lungs due to its obligate aerobic nature, although it can disseminate to other organs.[4] The unique lipid-rich cell wall of M. tuberculosis, composed of complex structures such as mycolic acids, arabinogalactan, and peptidoglycan, contributes to its acid-fastness, virulence, and resistance to environmental stress and host immune responses.[5,6] The pathogenesis of tuberculosis is closely linked to the host immune response, particularly cell-mediated immunity. Following infection, M. tuberculosis survives and multiplies within macrophages, evading host defenses.[7] Activation of CD4+ T lymphocytes plays a crucial role in containing the infection by stimulating macrophage activation and granuloma formation.[8] Clinically, pulmonary tuberculosis often presents with persistent cough, hemoptysis, fever, night sweats, weight loss, and chest pain.[9] Early and accurate diagnosis is essential not only for effective patient management but also for preventing transmission within the community.[10] A variety of diagnostic methods are available for the detection of tuberculosis, including conventional techniques such as Ziehl–Neelsen staining and culture methods using

Lowenstein–Jensen medium or liquid culture systems like MGIT. However, these methods have limitations in terms of sensitivity and turnaround time.[11,12] Recent advances in molecular diagnostics have revolutionized TB detection, with nucleic acid amplification tests (NAATs) offering rapid and accurate results. Among these, the Cartridge-Based Nucleic Acid Amplification Test (CB-NAAT), also known as the Xpert MTB/RIF assay, has gained widespread acceptance due to its ability to simultaneously detect M. tuberculosis and rifampicin resistance within a short time frame. This assay targets mutations in the rpoB gene, which are responsible for rifampicin resistance, a key marker for multidrug-resistant tuberculosis (MDR-TB).[13,14] The emergence of drug-resistant tuberculosis poses a significant challenge to global TB control efforts. Rifampicin-resistant TB, often associated with resistance to other first-line drugs, requires prolonged treatment with second-line drugs that are more toxic, expensive, and less effective. India bears a substantial burden of MDR-TB, contributing significantly to global estimates. Therefore, early detection of drug resistance is crucial for initiating appropriate therapy and reducing disease transmission.[15-17] Another critical aspect in the epidemiology of tuberculosis is its strong association with Human Immunodeficiency Virus (HIV) infection. HIV compromises the immune system, particularly by reducing CD4+ T cell counts, thereby increasing susceptibility to opportunistic infections such as TB.[18,19] TB is one of the leading causes of death among HIV-infected individuals. The coexistence of TB and HIV not only complicates diagnosis and treatment but also accelerates disease progression. In such patients, atypical clinical presentations and lower bacterial loads may further hinder diagnosis using conventional methods.[20] Given these challenges, rapid molecular diagnostic tools like CB-NAAT play a pivotal role in the early detection of TB and drug resistance, especially in high-risk groups such as HIV-infected individuals. Additionally, assessment of immune status through CD4 cell count provides valuable insights into disease severity and prognosis in HIV-TB co-infected patients.[21] In this context, the present study aims to evaluate the utility of CB-NAAT in detecting rifampicin resistance among pulmonary tuberculosis patients and to analyze its association with HIV co-infection and CD4 cell levels, thereby contributing to improved diagnostic and management strategies.

MATERIALS AND METHODS

Study Design: This study was designed as an analytical cross-sectional study to evaluate the detection of Mycobacterium tuberculosis and rifampicin resistance using CB-NAAT and to assess its association with HIV co-infection and CD4 cell counts.

Study Setting: The study was conducted in the Department of Microbiology, Central Microbiology Laboratory, Government Thanjavur Medical College and Hospital, Thanjavur, Tamil Nadu.

Study Duration: The study was carried out over a period of one year.

Ethical Approval: Ethical clearance for the study was obtained from the Institutional Ethics Committee prior to the commencement of the study. Written informed consent was obtained from all participants.

Study Population: The study population included patients attending the Outpatient Department of Chest Medicine with clinical features suggestive of pulmonary tuberculosis, such as cough for more than two weeks, fever, weight loss, and loss of appetite.

Sample Size: The sample size was calculated using OpenEpi software by assuming a population size of 1,000,000, anticipated frequency of 50%, confidence limits of 7%, and a confidence level of 95%. The calculated sample size was 186, which was rounded to 190 to account for potential dropouts. A total of 188 patients were included in the final analysis.

Inclusion Criteria

- Patients aged above 18 years
- Patients presenting with symptoms suggestive of pulmonary tuberculosis (cough >2 weeks, fever, weight loss, loss of appetite)
- Newly diagnosed pulmonary tuberculosis patients as per National Tuberculosis Elimination Programme (NTEP) guidelines
- Newly diagnosed HIV-TB co-infected patients

Exclusion Criteria

- Patients with extra-pulmonary tuberculosis
- Patients receiving immunosuppressive therapy, chemotherapy, or radiotherapy
- Patients with lung malignancies
- Patients with chronic lung diseases such as interstitial lung disease, bronchiectasis, and cystic fibrosis
- Patients below 18 years of age

Sample Collection: Sputum samples were collected under aseptic precautions. Two samples were obtained from each patient: a spot sample collected under supervision and an early morning sample collected the next day. Patients were instructed to produce deep cough sputum, and samples of 2–5 mL volume, preferably mucopurulent, were collected in sterile containers in well-ventilated areas. Blood samples were collected by venipuncture. Approximately 2 mL of blood was collected for HIV testing, and an additional 2 mL was collected in EDTA vacutainers for CD4 count analysis.

Laboratory Procedures

CB-NAAT (Xpert MTB/RIF Assay): Sputum samples were processed using the CB-NAAT technique according to standard operating procedures. A sample reagent containing sodium hydroxide and isopropanol was added to the sputum sample in a 2:1 ratio and incubated for 15 minutes at room temperature. Following this, 3 mL of the treated sample was transferred into the test cartridge and loaded into the GeneXpert system. The assay performs automated sample processing, DNA extraction, amplification, and detection of

ISSN (O): 2687-5365; ISSN (P): 2753-6556

Mycobacterium tuberculosis and mutations in the rpoB gene associated with rifampicin resistance. Results were generated within approximately 100 minutes and reported as MTB detected or not detected, along with rifampicin resistance status categorized as detected, not detected, or indeterminate.

HIV Testing: HIV testing was carried out among confirmed TB patients using a sequential testing algorithm with three rapid diagnostic methods:

- HIV 1 & 2 antibody detection by dot immunoassay
- HIV 1–2 antibody detection by flow-through immunoassay
- HIV 1–2 tri-line card test based on lateral flow immunochromatography

The tests were performed according to standard protocols, and results were interpreted based on the presence or absence of control and test bands or dots. A sample was considered positive only if all three tests were reactive.

CD4 Cell Count Estimation: CD4 cell count estimation was performed using flow cytometry. Whole blood samples collected in EDTA vacutainers were incubated with fluorochrome-labeled monoclonal antibodies specific for CD4 receptors. After incubation in the dark at room temperature, samples were processed in a flow cytometer. Cells were aligned in a single stream using hydrodynamic focusing and passed through a laser beam. Forward scatter, side scatter, and fluorescence signals were measured to identify and quantify CD4+ T lymphocytes. The results were expressed as absolute CD4 cell count (cells/µL of blood).

Data Collection: All demographic, clinical, and laboratory data were recorded in a pre-designed data collection form and subsequently entered into Microsoft Excel for analysis.

Statistical Analysis: Statistical analysis was performed using SPSS version 16. Continuous variables were expressed as mean and standard deviation, while categorical variables were presented as frequencies and percentages. The association between categorical variables was assessed using the Chi-square test or Fisher's exact test. Comparison of means between groups was performed using the unpaired t-test, and one-way analysis of variance (ANOVA) was used for multiple group comparisons. A p-value of less than 0.05 was considered statistically significant.

RESULTS

Study Population: A total of 190 patients with clinical suspicion of pulmonary tuberculosis were enrolled in the study. Out of these, 188 patients were confirmed positive for Mycobacterium tuberculosis by CB-NAAT and were included in the final analysis [Figure 1].

Figure 1: CB-NAAT

Distribution of Pulmonary TB and HIV Co-infection: Among the 188 confirmed cases, 177 (94.1%) were diagnosed with pulmonary tuberculosis (PTB) alone, while 11 (5.9%) patients were found to have HIV-TB co-infection. Rifampicin resistance was observed in both groups. Among PTB patients without HIV co-infection, 7 out of 177 cases (4%) showed rifampicin resistance, whereas among PTB patients with HIV co-infection, 1 out of 11 cases (9.1%) showed rifampicin resistance. However, the difference between the two groups was not statistically significant (p = 0.389) [Table 1].

Table 1: Comparison of frequency distribution of rifampicin resistance between PTB and PTB+HIV co-infected patients

S.No	Rifampicin Resistance	PTB alone (N=177) n (%)	PTB+HIV (N=11) n (%)	Chi-square	df	P value
1	Not detected	170 (96%)	10 (90.9%)	0.671	1	0.389 (NS)
2	Detected	7 (4%)	1 (9.1%)			

Gender Distribution and Association with Rifampicin Resistance: The study population showed a male predominance with 136 (72.3%) males and 52 (27.7%) females. However, when analyzed in relation to rifampicin resistance, a statistically significant association was observed (p = 0.038). Among rifampicin-resistant cases, females constituted 62.5% (5/8), whereas males accounted for

ISSN (O): 2687-5365; ISSN (P): 2753-6556

37.5% (3/8). In contrast, among non-resistant cases, males were predominant (73.9%) [Table 2].

Table 2: Comparison of gender distribution with respect to rifampicin resistance status

S. No	Gender	No RR (N=180) n (%)	RR (N=8) n (%)	Chi-square	df	P value
1	Female	47 (26.1%)	5 (62.5%)	5.06	1	0.038*
2	Male	133 (73.9%)	3 (37.5%)			

Age Distribution and Association with Rifampicin Resistance: The most commonly affected age group among TB patients was 51–60 years (26.7%). A statistically significant association was observed between age category and rifampicin resistance (p = 0.017). A higher proportion of rifampicin-resistant cases was noted in the 61–70 years age group (50%), followed by the 31–40 years age group (37.5%) [Table 3].

Table 3: Comparison of age distribution with respect to rifampicin resistance status

S. No	Age Group (years)	No RR (N=180) n (%)	RR (N=8) n (%)	Chi-square	df	P value
1	<20	2 (1.1%)	0 (0%)	15.5	6	0.017*
2	21–30	31 (17.2%)	0 (0%)			
3	31–40	37 (20.6%)	3 (37.5%)			
4	41–50	40 (22.2%)	0 (0%)			
5	51–60	48 (26.7%)	1 (12.5%)			
6	61–70	18 (10%)	4 (50%)			
7	>70	4 (2.2%)	0 (0%)			

CD4 Count Analysis: CD4 cell count analysis revealed a significant difference between groups. Patients with PTB and HIV co-infection with rifampicin resistance had a markedly lower mean CD4 count (452 cells/µL) compared to patients with PTB and rifampicin resistance without HIV co-infection (mean 847 ± 128 cells/µL) [Figure 2]. This difference was found to be highly statistically significant (p < 0.0001) [Table 4].

Table 4: Comparison of CD4 count with respect to rifampicin resistance status

S. No	Category	n	Mean CD4 (cells/µL)	SD	F value	df	P value
1	PTB + HIV + RR	1	452	-	25.6	2,15	<0.0001*
2	PTB + RR	7	847	128			

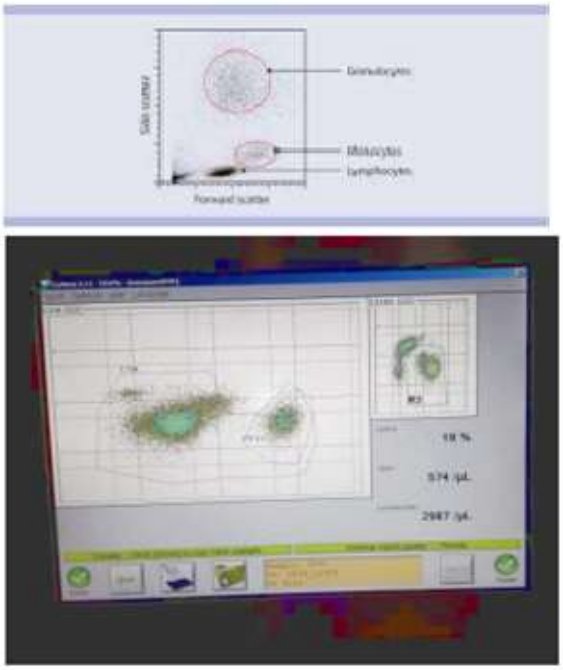

Figure 2: Flow Cytometery

The study demonstrated that the prevalence of rifampicin resistance among pulmonary tuberculosis patients was 4.3%. Although a higher proportion of resistance was observed among HIV co-infected patients, the association was not statistically significant. However, significant associations were found with gender and age. Additionally, CD4 cell count was significantly reduced in HIV-TB co-infected patients with rifampicin resistance, indicating severe immunosuppression.

DISCUSSION

Tuberculosis (TB) continues to be a major global health concern, particularly in developing countries, and the emergence of drug-resistant strains further complicates its control. In the present study, the prevalence of rifampicin-resistant TB (RR-TB) was found to be 4.3%, which lies between the global estimate of approximately 3.4% and the Indian National Drug Resistance Survey (NDRS) estimate of 6.19%. These findings are comparable with reports from the World Health Organization Global TB Report, which highlights India as one of the highest burden countries for MDR/RR-TB. The slightly lower prevalence observed in this study may be attributed to inclusion of predominantly newly diagnosed cases with minimal prior exposure to anti-tubercular therapy.[22,23] The present study demonstrated a clear male predominance (72.3%) among pulmonary TB patients. This observation is

ISSN (O): 2687-5365; ISSN (P): 2753-6556

consistent with studies such as that by R. Março et al., who reported a higher incidence of TB among males across different age groups.[24] The increased prevalence among males is often attributed to higher exposure to risk factors such as smoking, alcohol consumption, occupational hazards, and delayed healthcare-seeking behavior. However, when rifampicin resistance was analyzed, a statistically significant association with female gender ($p = 0.038$) was observed, with females constituting 62.5% of resistant cases. Similar findings were reported in a study by Ukwamedua et al., and Liu et al., where female gender was identified as a risk factor for MDR-TB.[25,26] This variation may be due to biological differences in immune response, hormonal influences, or differences in treatment adherence and access to healthcare. Age-wise distribution in the present study revealed a significant association between older age groups and rifampicin resistance ($p = 0.017$), with the highest proportion seen in the 61–70 years age group. This finding is in agreement with the study conducted by Henry Victor Omote et al., which reported a higher prevalence of rifampicin resistance among elderly patients.[27] The increased susceptibility in older individuals may be due to declining immunity, presence of comorbidities, and previous incomplete or irregular TB treatment. The relationship between HIV infection and rifampicin resistance was also explored in this study. The prevalence of RR-TB among HIV-TB co-infected patients was 9.1%, compared to 4% among non-HIV TB patients. Although this difference was not statistically significant ($p = 0.389$), it is consistent with trends observed in studies from high HIV burden settings such as Brazil, Ethiopia, and the United States, where higher rates of drug-resistant TB have been reported among HIV-infected individuals. A study by Okonkwo et al,[28] reported a similar RR-TB prevalence of 4.8% among HIV co-infected patients, which closely aligns with the findings of the present study. Factors such as malabsorption of anti-TB drugs, increased pill burden, drug-drug interactions, and poor adherence to therapy in HIV patients may contribute to the development of drug resistance. A key finding of this study was the significant reduction in CD4 cell count among HIV-TB co-infected patients with rifampicin resistance ($p < 0.0001$). Patients with HIV and RR-TB had markedly lower CD4 counts compared to those without HIV co-infection. This observation is supported by studies demonstrating that severe immunosuppression is associated with increased susceptibility to TB and poor clinical outcomes. Reduced CD4 counts impair the host's ability to mount an effective cell-mediated immune response against Mycobacterium tuberculosis, leading to increased bacillary load and disease progression. This emphasizes the importance of immunological monitoring in HIV-TB co-infected patients.[29,30] The use of CB-NAAT (Xpert MTB/RIF assay) in this study proved to be highly effective for rapid diagnosis of TB and detection of rifampicin resistance. The assay significantly reduces diagnostic delay compared to conventional culture methods, enabling early initiation of appropriate therapy. Similar observations have been reported in multiple studies and endorsed by the World Health Organization, which recommends CB-NAAT as an initial diagnostic test for suspected TB and drug-resistant TB cases. However, CB-NAAT detects only rifampicin resistance and does not provide comprehensive drug susceptibility data, necessitating further testing with line probe assays or culture-based drug susceptibility testing. Overall, the findings of the present study are in concordance with several national and international studies, reinforcing the importance of early detection of drug resistance and integrated TB-HIV management. While the prevalence of rifampicin resistance remains relatively low, the significant associations with gender, age, and immune status highlight the need for targeted interventions. Strengthening diagnostic infrastructure, improving treatment adherence, and integrating TB and HIV control programs are essential steps in curbing the spread of drug-resistant tuberculosis.

Limitations of the Study: The present study has certain limitations that should be considered while interpreting the results. Firstly, the sample size was relatively small, particularly in the HIV-TB co-infected group, which may have affected the statistical significance of some associations. Secondly, the study duration was limited to one year, which may not fully reflect long-term trends in rifampicin resistance. Thirdly, the diagnosis of tuberculosis and rifampicin resistance was based solely on CB-NAAT, and confirmation using gold standard culture methods or line probe assays was not performed. Additionally, resistance to drugs other than rifampicin was not evaluated, limiting the assessment of multidrug-resistant TB. Finally, CD4 count comparison was restricted and not assessed across all patient categories, which may have influenced the interpretation of immune status in relation to disease severity.

CONCLUSION

This study demonstrates that CB-NAAT is an effective and rapid diagnostic tool for detecting Mycobacterium tuberculosis and rifampicin resistance in pulmonary tuberculosis patients. The prevalence of rifampicin resistance was found to be 4.3%, which is comparable with global estimates. Although a higher proportion of resistance was observed among HIV-TB co-infected patients, the association was not statistically significant. A significant association was noted between rifampicin resistance and female gender, as well as with increasing age. Importantly, HIV co-infected patients with rifampicin resistance showed markedly reduced CD4 cell counts, indicating severe immunosuppression. These findings highlight the

ISSN (O): 2687-5365; ISSN (P): 2753-6556

importance of early detection of drug resistance and the role of molecular diagnostics in improving patient outcomes. Integrated management of TB and HIV, along with routine monitoring of immune status, is essential. Strengthening diagnostic strategies and ensuring timely initiation of appropriate therapy are crucial to control the spread of drug-resistant tuberculosis.

REFERENCES

1. Chakaya J, Khan M, Ntoumi F, Aklillu E, Fatima R, Mwaba P, Kapata N, Mfinanga S, Hasnain SE, Katoto PD, Bulabula AN. Global Tuberculosis Report 2020–Reflections on the Global TB burden, treatment and prevention efforts. International journal of infectious diseases. 2021 Dec 1;113:S7-12.
2. Sulis G, Roggi A, Matteelli A, Raviglione MC. Tuberculosis: epidemiology and control. Mediterranean journal of hematology and infectious diseases. 2014 Nov 1;6(1):e2014070.
3. Javalkar SR, Jukaku R. A study on the role of Cartridge Based Nucleic Acid Amplification Test (CBNAAT) in the Diagnosis of Sputum Negative Pulmonary Tuberculosis. Medica. 2025 Jan;14(1):43.
4. Devi NP, Singh TN, Singh HR, Singh WS, Joyshankar Y. CONTINUING MENACE OF RIFAMPICIN RESISTANT TUBERCULOSIS IN RIMS HOSPITAL, IMPHAL, MANIPUR. JK Practitioner. 2021 Jan 1;26.
5. Mathew P. A Comparative Study on Microscopy, Culture and Xpert MTB/RIF Assay in the Diagnosis of Pulmonary Tuberculosis in Hiv-Infected Patients in a Tertiary Care Hospital (Doctoral dissertation, Rajiv Gandhi University of Health Sciences (India)).
6. Sharma AK, Gupta N, Verma S, Chandran A, Dixit R. A study on procedural delay in diagnosis and start of treatment in drug resistant tuberculosis under RNTCP. Indian Journal of Tuberculosis. 2019 Jul 1;66(3):394-401.
7. Cooper AM. Cell-mediated immune responses in tuberculosis. Annual review of immunology. 2009 Apr 23;27(1):393-422.
8. Dannenberg AM. Delayed-type hypersensitivity and cell-mediated immunity in the pathogenesis of tuberculosis. Immunology today. 1991 Jul 1;12(7):228-33.
9. Flynn JL, Chan J. Immune cell interactions in tuberculosis. Cell. 2022 Dec 8;185(25):4682-702.
10. Achkar JM, Casadevall A. Antibody-mediated immunity against tuberculosis: implications for vaccine development. Cell host & microbe. 2013 Mar 13;13(3):250-62.
11. THANGAVELU K, JAMIR I, ELLAPPAN K, KRISHNAKUMARIAMMA K, GOPICHAND P, DAS S, JOSEPH N. Comparison of MGIT 960 with Lowenstein Jensen Media for Recovery of Mycobacteria from Extrapulmonary Specimens in Southern India. Journal of Clinical & Diagnostic Research. 2021 Mar 1;15(3).
12. Kaur KP, Arora B. Enhanced diagnostic yield of Auramine-O staining and mycobacteria growth indicator tube over Ziehl-Neelsen staining and Lowenstein Jensen media in extra-pulmonary tuberculosis. Journal of evolution of Medical and Dental Sciences. 2014 Jul 14;3(28):7787-92.
13. Opota O, Mazza-Stalder J, Greub G, Jaton K. The rapid molecular test Xpert MTB/RIF ultra: towards improved tuberculosis diagnosis and rifampicin resistance detection. Clinical microbiology and Infection. 2019 Nov 1;25(11):1370-6.
14. Zifodya JS, Schiller I, Dendukuri N, Tollefson D, Schumacher SG, Ochodo EA, Pai M, Steingart KR. Xpert MTB/RIF and Xpert MTB/RIF Ultra for pulmonary tuberculosis and rifampicin resistance in adults. Cochrane Database of Systematic Reviews. 2019(6).
15. Kohli M, Schiller I, Dendukuri N, Dheda K, Denkinger CM, Schumacher SG, Steingart KR. Xpert® MTB/RIF assay for extrapulmonary tuberculosis and rifampicin resistance. Cochrane Database of Systematic Reviews. 2018(8).
16. Kay AW, Fernández LG, Takwoingi Y, Eisenhut M, Detjen AK. Xpert MTB/RIF and Xpert MTB/RIF Ultra assays for active tuberculosis and rifampicin resistance in children. Cochrane Database of Systematic Reviews. 2020(8).
17. Cochrane Central Editorial Service, Kay AW, Madison M, Scandrett K, Ness T, Amuge P, Inbaraj LR, Sathya Narayanan MK, González Fernández L, Eisenhut M, Ismail N. Xpert MTB/RIF Ultra assay for tuberculosis disease and rifampicin resistance in children. Cochrane Database of Systematic Reviews. 1996 Sep 1;2025(10).
18. Okoye AA, Picker LJ. CD 4+ T-cell depletion in HIV infection: mechanisms of immunological failure. Immunological reviews. 2013 Jul;254(1):54-64.
19. Ronacher K, Joosten SA, van Crevel R, Dockrell HM, Walzl G, Ottenhoff TH. Acquired immunodeficiencies and tuberculosis: focus on HIV/AIDS and diabetes mellitus. Immunological reviews. 2015 Mar;264(1):121-37.
20. Abrahem R, Chiang E, Haquang J, Nham A, Ting YS, Venketaraman V. The role of dendritic cells in TB and HIV infection. Journal of Clinical Medicine. 2020 Aug 17;9(8):2661.
21. Khatawkar AV, Talikoti N, Malkiwadeyar PK, Juned M, Hubballi VB. The study of utility of CBNAAT in diagnosing pulmonary tuberculosis in HIV positive patients in North Karnataka. J. Evid. Based Med. Healthc. 2019;6(19):1464-9.
22. World Health Organization. World Health Organization Global TB Report 2014. Geneva, Switzerland [Internet].
23. Behera D, Pannu VP, Behera RK. National TB elimination programme—Can it end TB in India by 2025: an appraisal. The Indian Journal of Chest Diseases and Allied Sciences. 2020 Dec 7;62:203-27.
24. Marçôa R, Ribeiro AI, Zão I, Duarte R. Tuberculosis and gender–Factors influencing the risk of tuberculosis among men and women by age group. Pulmonology. 2018 May 30;24(3):199-202.
25. Ukwamedua H, Omote V, Etaghene J, Oseji ME, Agwai IC, Agbroko H. Rifampicin resistance among notified pulmonary tuberculosis (PTB) cases in South-Southern Nigeria. Heliyon. 2019 Jul 1;5(7).
26. Liu Q, Lu P, Martinez L, Yang H, Lu W, Ding X, Zhu L. Factors affecting time to sputum culture conversion and treatment outcome of patients with multidrug-resistant tuberculosis in China. BMC infectious diseases. 2018 Mar 6;18(1):114.
27. Omote V, Ukwamedua HA, Bini N, Etaghene J, Omoviye EO, Iloka MO. Awareness, Risk Factors and Prevalence of Viral Hepatitis B and C among Antenatal Attendees in South-southern Nigeria: A Cross-sectional and Hospital-based Study. American Journal of Public Health. 2020 Dec 15;8(6):202-8.
28. Okonko IO, Ejike IU, Innocent-Adiele C, Cookey TI. HIV coinfections with tuberculosis among HIV-1 infected individuals in old Cross River State, Nigeria. Journal of Immunoassay and Immunochemistry. 2020 May 3;41(3):245-56.
29. Lawn SD, Churchyard G. Epidemiology of HIV-associated tuberculosis. Current Opinion in HIV and AIDS. 2009 Jul 1;4(4):325-33.
30. Steingart KR, Sohn H, Schiller I, Kloda LA, Boehme CC, Pai M, Dendukuri N. Xpert® MTB/RIF assay for pulmonary tuberculosis and rifampicin resistance in adults. Cochrane database of systematic reviews. 2013(1).

ISSN (O): 2687-5365; ISSN (P): 2753-6556