

Monaldi Archives for Chest Disease

eISSN 2532-5264 https://www.monaldi-archives.org/

Publisher's Disclaimer. E-publishing ahead of print is increasingly important for the rapid dissemination of science. The ***Early Access*** service lets users access peer-reviewed articles well before print / regular issue publication, significantly reducing the time it takes for critical findings to reach the research community.
These articles are searchable and citable by their DOI (Digital Object Identifier).

The **Monaldi Archives for Chest Disease** is, therefore, e-publishing PDF files of an early version of manuscripts that have undergone a regular peer review and have been accepted for publication, but have not been through the typesetting, pagination and proofreading processes, which may lead to differences between this version and the final one.
The final version of the manuscript will then appear in a regular issue of the journal.

E-publishing of this PDF file has been approved by the authors.

All legal disclaimers applicable to the journal apply to this production process as well.

Monaldi Arch Chest Dis 2026 [Online ahead of print]

To cite this Article:
Bhushan Gupta V, Kumar Rai D, Karmakar S, et al. **Prevalence and clinical correlates of chronic pulmonary aspergillosis among symptomatic post-tuberculosis patients at a tertiary hospital in Eastern India**. *Monaldi Arch Chest Dis* doi: 10.4081/monaldi.2026.3812

Submitted: 14-11-2025
Accepted: 22-10-2026

©The Author(s), 2026
Licensee PAGEPress, Italy

Note: The publisher is not responsible for the content or functionality of any supporting information supplied by the authors. Any queries should be directed to the corresponding author for the article.
All claims expressed in this article are solely those of the authors and do not necessarily represent those of their affiliated organizations, or those of the publisher, the editors and the reviewers. Any product that may be evaluated in this article or claim that may be made by its manufacturer is not guaranteed or endorsed by the publisher.

Prevalence and clinical correlates of chronic pulmonary aspergillosis among symptomatic post-tuberculosis patients at a tertiary hospital in Eastern India

Vatsal Bhushan Gupta,[1,2] Deependra Kumar Rai,[2] Saurabh Karmakar,[2]
Somesh Thakur,[2] Priya Sharma,[2,3] Subhash Kumar[4]

[1]City Super speciality Hospital Pvt Ltd, Gorakhpur, UP; [2]Department of Pulmonary Medicine, All India Institute of Medical Sciences, Patna; [3]Sarvodaya Hospital and Research Centre, Faridabad; [4]Department of Radiodiagnosis, All India Institute of Medical Sciences, Patna, India

Correspondence: Priya Sharma, Sarvodaya Hospital and Research Centre, Faridabad, India. Tel.: 9971934929. E-mail: priyasharma25292@gmail.com

Contributions: all the authors have equally contributed in conceptualization, data collection, analysis and manuscript editing.

Conflict of interest: the authors declare no conflict of interest.

Ethics approval and consent to participate: the study was approved by the Institutional Ethics Committee (AIIMS/Pat/IEC/PGTh/July 21/05).

Informed consent: informed consent taken from all participants.

Patient consent for publication: obtained.

Availability of data and materials: all data underlying the findings are available upon request.

Declaration of generative artificial intelligence and artificial intelligence-assisted technologies in the writing process: AI technologies were not used.

Abstract

Chronic pulmonary aspergillosis (CPA) is an underrecognized but significant sequela of pulmonary tuberculosis (TB), and data from India remain limited. In this single-center, cross-sectional study from eastern India, we evaluated 280 post-TB patients with residual radiological abnormalities to estimate CPA prevalence and identify associated clinical, radiological, and biochemical features. After excluding patients with active TB, interstitial lung disease, malignancy, or prior antifungal therapy, all participants underwent clinical assessment, spirometry, high-resolution computed tomography, Aspergillus-specific serology, and bronchoalveolar lavage when indicated. CPA was diagnosed in 27.5% of patients, with chronic cavitary pulmonary aspergillosis being the most common subtype. Multivariable analysis showed that low body mass index, the presence of aspergilloma, and elevated C-reactive protein were independently associated with CPA, while Aspergillus-specific IgG was positive in 92.2% of cases. These findings highlight the high burden of CPA among post-TB patients in India and underscore the need for greater clinician awareness and standardized screening to ensure timely diagnosis and management.

Key words: post-tuberculosis lung disease, aspergillus, chronic pulmonary aspergillosis, prevalence, clinical correlates.

Introduction

Chronic pulmonary aspergillosis (CPA) is the most deadly chronic sequelae of pulmonary tuberculosis (PTB) with diverse manifestations. [1]. According to various estimates, in India, annual incidence of CPA varies between 27000-1,70,000. Overall, an estimated 1.2 million individuals have CPA that develop after PTB, of whom approximately 300,000 reside in India [2]. CPA is commonly confused with entities like smear negative PTB due to similarities in clinical and radiological presentation. This is more often seen in countries with lack of appropriate diagnostics and having a high prevalence of TB. This leads to delayed diagnosis, wastage of healthcare resources ultimately leading to increased morbidity and mortality. Hence, reaching a diagnosis is of utmost importance to initiate treatment at an appropriate time [3].

Very few studies have looked into the prevalence of CPA in India. These have been performed in patients with small sample size [4,5]. None have looked into its various subtypes. This research was performed with the aim of studying the profile of CPA among post TB patients. Its primary objective was to estimate the proportion of CPA amongst post TB patients visiting a tertiary care institute. The secondary objective was to determine independent factors associated with CPA through univariable regression analysis, and then perform a multivariable regression analysis on the factors which show a significant difference between the CPA and non CPA group.

Materials and Methods

Study design

We conducted a hospital-based, cross-sectional study at the Department of Pulmonary Medicine, All India Institute of Medical Sciences (AIIMS), Patna over a period of 2 years from January 2022 to January 2024. The study was approved by the Institutional Ethics Committee (AIIMS/Pat/IEC/PGTh/July 21/05).

All consecutive eligible patients aged ≥ 12 years with a prior history of anti-tubercular therapy (ATT) and residual radiological abnormalities suggestive of post-TB sequelae, who presented to the outpatient or inpatient services during the study period, were enrolled after obtaining informed written consent. The treating physician suspected the radiological changes which was further confirmed by Radiologist. All patients suffering from other respiratory illness like interstitial lung disease (ILD), malignancy, active TB, non-tubercular mycobacteria (NTM), any known immunocompromised states (HIV infection, chronic steroid use, organ transplantation,

or other systemic immunosuppressive disorders), chronic infections, history of intake of antifungal therapy in the past, pregnant and lactating females and those not giving voluntary informed consent were excluded from the study. Active TB and NTM was ruled out on basis of sputum or bronchoalveolar lavage for acid fast bacilli (AFB) smear/culture and GeneXpert for mycobacterium tuberculosis.

The required sample size was calculated as 253 using the formula $n = Z^2pq/d^2$ (with expected CPA prevalence of 10% as per study done by Bongomin et al. [6], 95% confidence level, and 3.7% absolute precision), and 280 patients were ultimately enrolled over two years.

Definitions

CPA was diagnosed based on European Respiratory Society (ERS) guidelines [1], when all the following criteria were met:

1. Compatible clinical presentation lasting ≥3 months — including chronic cough, hemoptysis, weight loss, dyspnea, or fatigue.
2. Characteristic radiological abnormalities on chest HRCT — such as one or more cavities with or without aspergilloma, pleural thickening, fibrotic changes, or progressive paracavitary infiltrates.
3. Microbiological or immunological evidence of Aspergillus infection, demonstrated by at least one of the following:
    - Positive Aspergillus-specific IgG by ImmunoCAP™ or ELISA (using validated cut-offs ≥27 mgA/L for ImmunoCAP™, or >12 IU/mL for ELISA);
    - Growth of Aspergillus species from respiratory samples (sputum or BAL culture);
    - Positive BAL galactomannan (GM) index ≥2.5, performed selectively in patients with equivocal imaging or serology.
4. Exclusion of alternative diagnoses, including active pulmonary tuberculosis, non-tuberculous mycobacterial infection, lung malignancy, or other chronic infections, through microbiological, cytological, and radiological assessment.

Patients meeting all these criteria were classified as having CPA, and further subtyped (CCPA, SIA, CFPA, aspergilloma, or Aspergillus nodule) based on HRCT and clinical evolution. This was a cross-sectional study, and patients were not followed prospectively for outcomes.

Aspergillus Sensitization (AS) was defined as *A. fumigatus*–specific IgE ≥0.35 kUA/L or positive skin prick test in absence of radiological or serological evidence of tissue invasion or bronchopulmonary aspergillosis.

Allergic Bronchopulmonary Aspergillosis (ABPA) was diagnosed per revised ISHAM guidelines [7] requiring compatible host factors, Aspergillus sensitization, elevated total IgE (≥500 IU/mL), and at least two additional criteria (raised *A. fumigatus*-specific IgG, eosinophilia, or characteristic radiologic findings).

Data collection

Clinical history, physical examination, spirometry, High resolution CT chest, laboratory tests (Blood test, sputum test, viral markers and inflammatory markers including CRP and ESR) and/or Bronchoscopy with bronchoalveolar lavage (BAL) were performed. Lung functions were also assessed using spirometry (Quark PFT-COSMED) and interpreted as per American Thoracic Society (ATS) guidelines. [8] Dyspnoea was graded using the Modified Medical Research Council (mMRC) scale. Weight loss was defined as >5% reduction in body weight over 6–12 months. [9] Aspergillus-specific IgG and IgE were measured using ImmunoCAP™ (182 patients) and ELISA (98 patients), with validated cut-offs i.e., 27mgA/L while using ImmunoCAP™ [10] and graded as >12 IU/L (positive), 8-12 (equivocal) and <8 (negative) for ELISA. [11]

BAL galactomannan was performed in a subset of patients with indeterminate imaging or equivocal Aspergillus IgG results, or when clinical suspicion of CPA persisted despite negative serology. For BAL fluid Galactomannan, a cut-off value of 2.5 showing sensitivity and specificity of 50% and 100% respectively which was used as a diagnostic criteria for CPA. [12]

Statistical analysis

Data were analyzed using SPSS v23 (IBM Corp). Continuous variables were reported as mean ± SD or median (IQR) depending on distribution. Categorical variables were summarized as frequencies and percentages. Group comparisons were performed using t-test, Mann–Whitney U test, chi-square, or Fisher's exact test, as appropriate. Logistic regression was used to identify factors independently associated with CPA. Results were expressed as odds ratios (OR) with 95% confidence intervals (CI). A p-value <0.05 was considered statistically significant. Univariable logistic regression was used to identify factors associated with CPA, and those with significant associations were considered for inclusion in the multivariable model. The final model comprised clinical, radiological, and biochemical features and results were expressed as adjusted odds ratios (aOR) with 95% confidence intervals.

Results

A total of 280 patients were enrolled. The mean age was 44.0 ± 14.97 years, with 60% male participants. Median duration of symptoms was 2 years. The mean BMI (kg/m²) of the entire study population was 20.16 ± 3.30. Most participants were never-smokers (87.1%). Common residual symptoms included cough (92.1%), sputum (90.1%), dyspnea (73.2%), hemoptysis (59.6%) loss of appetite (37.1%) and loss of weight (33.6%). Of the 280 post-TB participants, 206 (73.6%) had microbiologically confirmed TB based on prior AFB smear/culture or GeneXpert positivity, while 74 (26.4%) had been treated empirically for radiologically suspected TB. Median interval from ATT completion to presentation was shorter in CPA patients (2.6 years) than in non-CPA patients (5.2 years, p = 0.04), with over half of the CPA group reporting no symptom-free period following TB treatment. Table 1 shows the CT findings in the entire study population.

Prevalence of CPA

CPA was diagnosed in 77/280 patients (27.5%). The percentage of subtypes including chronic cavitary pulmonary aspergillosis (CCPA) was 67.5% (n = 52), chronic fibrosing pulmonary aspergillosis (CFPA) was 24.7% (n = 19), aspergilloma was 6.5% (n = 5), and Aspergillus nodule was 1.3% (n = 1). We couldn't find any case of sub-acute invasive aspergillosis (SIA) in our study.

Among the 221 patients who underwent spirometry, restrictive (42.6%) and mixed (35.2%) patterns predominated in the CPA group, while the non-CPA group showed relatively higher proportions of normal (27.5%) and obstructive (10.2%) patterns; however, these differences were not statistically significant.

Associated features

- Clinical: CPA patients had significantly lower BMI (18.99 ± 3.17 vs. 20.60 ± 3.25; $p<0.001$) and higher prevalence of hemoptysis, loss of appetite, and weight loss compared to non-CPA patients (Table 2)
- Radiological: On HRCT, cavities (66.2% vs. 38.4%), aspergilloma (33.8% vs. 5.4%), and pleural thickening (14.3% vs. 3.9%) were significantly more frequent in the CPA group compared to non-CPA patients ($p<0.05$), while other lesions such as fibrosis and bronchiectasis showed no significant difference between groups (Table 2)

- Biochemical: ESR and CRP levels showed higher values in CPA subset (Table 2). BAL galactomannan testing was performed in 42 patients (Table 2). Of these, 17 had values >2.5, supporting a diagnosis of CPA when combined with other criteria. In two patients, CPA was diagnosed despite negative BAL galactomannan (<2.5), while in the remaining 21 cases, results were consistent with the overall diagnostic impression. The mean BAL galactomannan levels were significantly higher in the CPA group compared to the non-CPA group ($p<0.05$), with a strong association (correlation coefficient 0.78). Of the 77 CPA patients, Aspergillus fumigatus-specific IgG was assessed using either the ImmunoCAP™ assay (n = 182 overall) or ELISA (n = 98 overall), based on test availability. Among CPA cases, 60 were tested by ImmunoCAP™ and 17 by ELISA. Using validated cut-offs as mentioned in methodology, 71patients (92.2%) had positive IgG, 5 (6.5%) were negative, and 1 (1.3%) equivocal. The mean IgG values were markedly higher in CPA than in non-CPA patients (87.9 [38.8–137] mgA/L vs 21.4 [0–50.2] mgA/L, $p < 0.001$ for ImmunoCAP™; and 19.3 [10.9–27.8] U/L vs 6.4 [3.7–9.2] U/L, $p < 0.001$ for ELISA) (Table 2). Among those with negative or equivocal serology (n = 6, 7.8%), the diagnosis of CPA was established using a combination of characteristic HRCT findings, compatible chronic symptoms lasting ≥ 3 months, and either positive BAL galactomannan (≥ 2.5 in 4 patients) or isolation of *A. fumigatus* from culture (in 2 patients), after exclusion of alternative diagnoses. A single patient (1.3%) was classified as having an isolated *Aspergillus* nodule, which presented as a solitary, non-cavitating lesion on HRCT and was confirmed histopathologically on CT-guided biopsy demonstrating necrotizing granulomatous inflammation with septate fungal hyphae. Although seronegative, the histopathological and microbiological evidence met ERS/ESCMID diagnostic criteria for the *Aspergillus* nodule subtype of CPA.[1] Aspergillus fumigatus-specific IgG was positive in 92.2% of CPA patients, with significantly higher mean values compared to non-CPA patients ($p<0.001$) (Table 2). In 16 patients, IgG levels were positive but a diagnosis of CPA- (allergic bronchopulmonary aspergillosis) ABPA overlap was made as the clinico-radiological picture and serological tests met both the criteria. Aspergillus sensitisation was seen in 27.3% of CPA patients. Overall, serological diagnosis of ABPA secondary to PTB was identified in 27 patients (9.6%) (Table 2).

On multivariable logistic regression analysis (Table 3) three variables remained independently associated with CPA. Lower BMI (aOR 0.86; 95% CI 0.77–0.96; p=0.007), elevated serum

CRP (aOR 1.02; 95% CI 1.002–1.028; p=0.027) and aspergilloma (aOR 6.536; 95%CI 2.46-17.34; p=0.000).

Discussion

This study demonstrates a significant burden of chronic pulmonary aspergillosis (CPA) among post-tuberculosis (post-TB) patients, with a prevalence of 27.5% at a tertiary care hospital in eastern India. The findings emphasize the clinical relevance of CPA in TB-endemic settings where it often remains underdiagnosed. While our estimate is higher than community-based data from North India (~10%) [5], it is consistent with hospital-based studies showing prevalence between 20% and 57% [4,13]. This variation likely reflects differences in patient selection, diagnostic facilities, and disease awareness. Recent systematic reviews [14,15] further contextualize our results: Sehgal et al. reported a pooled CPA prevalence of ~18% in post-mycobacterial lung abnormality cohorts, whereas Madden et al. observed an overall prevalence of 9% among all TB patients, increasing to 13% post-treatment and up to 48% among those with persistent symptoms. These findings highlight that symptomatic, hospital-based populations, similar to ours, represent the higher end of the CPA spectrum. Mathematical modelling studies estimate over 1.5 million prevalent cases and 360,000 incident cases annually in India [16-18], underscoring the magnitude of under-recognition in routine practice.

The DEMOGRAPHIC PROFILE of CPA patients in this study was comparable to prior Indian and international data. The mean age of 44 years mirrors reports from India (42.2 years) [4] and neighbouring Asian countries where mean age ranged from 39.8 to 59 years [11,16,19-21]. Male predominance was observed (60%), consistent with earlier studies reporting 53–96% male representation [4,11,16,21]. Low body mass index (BMI) emerged as a key correlate of CPA, aligning with previous studies that identified malnutrition as a risk factor for aspergillus colonization and disease progression [4]. Undernutrition compromises immune function and impairs lung defence mechanisms, possibly promoting fungal persistence. Diabetes mellitus was observed in 12% of our CPA cases, comparable to other studies (5–28%) [4,11,16,21], supporting its recognized role in predisposing to fungal infections through immune dysregulation and structural lung damage [21].

CLINICAL FEATURES were largely consistent with the established symptom profile of CPA. Persistent cough, hemoptysis, anorexia, and weight loss were significantly more common in CPA patients compared to non-CPA counterparts, in agreement with studies from Vietnam,

Iran, and Pakistan [11,16,21]. These chronic symptoms are often misattributed to post-TB scarring, delaying diagnosis and treatment. Importantly, our finding of a shorter asymptomatic interval following anti-TB therapy among CPA patients (2.6 vs. 5.2 years) suggests that CPA can develop soon after TB treatment completion, echoing observations by Page et al. [21]. Early post-treatment surveillance, therefore, becomes critical to detect CPA before irreversible lung damage occurs. The mean BMI difference (18.99 vs. 20.6 kg/m^2) between CPA and non-CPA groups further reinforces the need to address nutritional status during post-TB follow-up.

LUNG FUNCTION analysis showed predominantly restrictive and mixed ventilatory patterns in CPA patients, paralleling earlier findings [4]. Reduced FVC and FEV1 values reflect parenchymal fibrosis and volume loss due to chronic infection. Although spirometry is not a diagnostic criterion [1], its use may help in functional characterization and monitoring of disease progression in CPA, particularly when structural changes are extensive.

RADIOLOGICAL ABNORMALITIES remain the cornerstone of CPA diagnosis. In our cohort, cavities, aspergilloma, and pleural thickening were significantly more frequent among CPA patients. These findings are consistent with previous reports by Singla et al. [4] and Page et al. [22], who identified cavitation and residual cavity walls as important risk factors. The strong association between aspergilloma and CPA (OR 8.89, 95% CI 4.12–19.21) reaffirms its diagnostic value. The presence of pleural thickening also correlated significantly with CPA (p=0.002), a feature often associated with chronic inflammation and prior pleural involvement. On multivariable logistic regression, low BMI (aOR 0.864, 95% CI 0.77–0.96) and elevated CRP (aOR 1.015, 95% CI 1.002–1.028) and aspergilloma (aOR6.536, CI 2.46-17.34) remained independent predictors of CPA. These findings highlight the synergistic contribution of systemic inflammation, nutritional compromise, and structural damage in CPA pathogenesis.

SEROLOGICAL TESTING played a pivotal role in diagnosis. Aspergillus-specific IgG was positive in 92.2% of CPA patients, corroborating global data reporting >90% sensitivity [1,22-24]. The high seropositivity rate across both ELISA and ImmunoCAP assays validates its diagnostic reliability, although inter-assay variability remains a concern. A subset of patients exhibited positive IgG but were classified as ABPA, consistent with overlapping immunological phenotypes described in earlier studies [23,13]. BAL galactomannan was positive in 17 of 42 tested patients, aligning with prior reports of moderate sensitivity and high specificity in CPA [12]. Despite its limited incremental yield, BAL galactomannan can aid diagnosis when imaging or serology are inconclusive. The low fungal culture yield (2.6%) parallels global trends [11,16,21,25-27], reflecting poor sensitivity of conventional methods and the need for

advanced fungal diagnostics. Aspergillus fumigatus remained the predominant species, consistent with prior Indian and international series [11,23].

The strengths of this study include its relatively large sample size, use of standardized ERS diagnostic criteria [1], and comprehensive evaluation integrating clinical, radiological, serological, and spirometric data. To our knowledge, this is one of the few studies from eastern India providing an in-depth analysis of CPA prevalence and associated factors among post-TB patients. However, certain limitations merit mention. Being a single-centre, hospital-based study, referral bias may have influenced prevalence estimates. The use of two different serological assays may have introduced variability, and long-term follow-up data on treatment outcomes were not available. The multivariable analysis may be susceptible to overfitting given the number of observed CPA events relative to the candidate predictors; therefore, the adjusted associations should be interpreted cautiously, and future studies with larger cohorts should incorporate formal assessment of model stability, collinearity, and calibration.

In conclusion, CPA is a common sequela of TB in India, with a prevalence of nearly 27.5% in our post-TB cohort. Clinical symptoms such as cough, hemoptysis, and weight loss, along with radiological findings of cavities and aspergilloma, and elevated inflammatory markers, should raise suspicion of CPA. Low BMI, aspergilloma, and elevated CRP were the strongest independent associations in our study. These findings highlight the need for greater clinician awareness, early screening strategies, and integration of CPA diagnosis into post-TB care pathways to prevent misdiagnosis and improve patient outcomes. Based on our findings, we propose a structured approach for early diagnosis of CPA in post-TB patients, which is illustrated in Figure 1.

Conclusions

CPA is highly prevalent amongst post TB population. Proportion of CPA amongst post TB patients was 27.5% (77/280) in this cohort at a tertiary care institute. Most common subtype of CPA was CCPA followed by CFPA and aspergilloma. Low BMI, aspergilloma, and elevated CRP are key associated features. These findings underscore the importance of routine screening for CPA in patients with post-TB sequelae to enable timely diagnosis and management. There is need to sensitize the clinicians about this entity and to formulate a screening plan for early diagnosis of the disease.

References

1. Denning DW, Cadranel J, Beigelman-Aubry C, et al. Chronic pulmonary aspergillosis: rationale and clinical guidelines for diagnosis and management. Eur Respir J 2016;47:45-68.
2. Agarwal R, Denning DW, Chakrabarti A. Estimation of the burden of chronic and allergic pulmonary aspergillosis in India. PLoS One 2014;9:e114745.
3. Beardsley J, Denning DW, Chau NV, et al. Estimating the burden of fungal disease in Vietnam. Mycoses 2015;58:101-6.
4. Singla R, Singhal R, Rathore R, et al. Risk factors for chronic pulmonary aspergillosis in post-TB patients. Int j Tuberc Lung Dis 2021;25:324-6.
5. Soundappan K, Sehgal IS, Prabhakar N, et al. Incidence and prevalence of chronic pulmonary aspergillosis in patients with post-tuberculosis lung abnormality: Results from a community survey in North India. Mycoses 2024;67:e13711.
6. Bongomin F. Post-tuberculosis chronic pulmonary aspergillosis: An emerging public health concern. PLoS Pathog 2020;16:e1008742.
7. Agarwal R, Sehgal IS, Muthu V, et al. Revised ISHAM-ABPA working group clinical practice guidelines for diagnosing, classifying and treating allergic bronchopulmonary aspergillosis/mycoses. Eur Respir J 2024;63:2400061.
8. Chhabra SK, Kumar R, Gupta U, et al. Prediction equations for spirometry in adults from northern India. Indian J Chest Dis Allied Sci 2014;56:221-9.
9. Gaddey HL, Holder K. Unintentional weight loss in older adults. Am Fam Physician 2014;89:718-22.
10. Sehgal IS, Choudhary H, Dhooria S, et al. Diagnostic cut-off of Aspergillus fumigatus-specific IgG in the diagnosis of chronic pulmonary aspergillosis. Mycoses 2018;61:770-6.
11. Hedayati MT, Azimi Y, Droudinia A, et al. Prevalence of chronic pulmonary aspergillosis in patients with tuberculosis from Iran. Eur J Clin Microbiol Infect Dis 2015;34:1759-65.
12. Sehgal IS, Dhooria S, Choudhary H, et al. Utility of serum and bronchoalveolar lavage fluid galactomannan in diagnosis of chronic pulmonary aspergillosis. J Clin Microbiol 2019;57:e01821-18.
13. Ajayababu A, Meena VP, Sethi P, et al. Allergic bronchopulmonary aspergillosis in patients with prior pulmonary tuberculosis: a study on the burden, clinic-radiological features, treatment outcomes and comparison with chronic pulmonary aspergillosis and post-tubercular lung disease patients. Mycoses 2025;68:e70034.

14. Sehgal IS, Soundappan K, Agarwal R, et al. Prevalence of chronic pulmonary aspergillosis in patients with mycobacterial and non-mycobacterial tuberculosis infection of the lung: a systematic review and meta-analysis. Mycoses 2025;68:e70060.
15. Madden A, Ofori SK, Budu M, et al. A systematic review of chronic pulmonary aspergillosis among patients treated for pulmonary tuberculosis. Clin Infect Dis 2025;81:e163-71.
16. Nguyen NTB, Le Ngoc H, Nguyen NV, et al. Chronic pulmonary aspergillosis situation among post tuberculosis patients in Vietnam: an observational study. J Fungi 2021;7:532.
17. Denning DW, Pleuvry A, Cole DC. Global burden of chronic pulmonary aspergillosis as a sequel to pulmonary tuberculosis. Bull World Health Organ 2011;89:864-72.
18. Denning DW, Cole DC, Ray A. New estimation of the prevalence of chronic pulmonary aspergillosis (CPA) related to pulmonary TB – a revised burden for India. IJID Regions 2023;6:7-14.
19. Denning DW, Riniotis K, Dobrashian R, Sambatakou H. Chronic cavitary and fibrosing pulmonary and pleural aspergillosis: case series, proposed nomenclature change, and review. Clin Infect Dis 2003;37:S265-80.
20. Oladele RO, Irurhe NK, Foden P, et al. Chronic pulmonary aspergillosis as a cause of smear-negative TB and/or TB treatment failure in Nigerians. Int J Tuberc Lung Dis 2017;21:1056-61.
21. Iqbal N, Irfan M, Mushtaq A, Jabeen K. Underlying conditions and clinical spectrum of chronic pulmonary aspergillosis (CPA): an experience from a tertiary care hospital in Karachi, Pakistan. J Fungi 2020;6:41.
22. Page ID, Byanyima R, Hosmane S, et al. Chronic pulmonary aspergillosis commonly complicates treated pulmonary tuberculosis with residual cavitation. Eur Respir J 2019;53:1801184.
23. Kousha M, Tadi R, Soubani AO. Pulmonary aspergillosis: a clinical review. Eur Respir Rev 2011;20:156-74.
24. Page ID, Richardson M, Denning DW. Antibody testing in aspergillosis--quo vadis? Med Mycol 2015;53:417-39.
25. Schweer KE, Bangard C, Hekmat K, Cornely OA. Chronic pulmonary aspergillosis. Mycoses 2014;57:257-70.
26. Kurhade AM, Deshmukh JM, Fule RP, et al. Mycological and serological study of pulmonary aspergillosis in central India. Indian J Med Microbiol 2002;20:141-4.

27. Shahid M, Malik A, Bhargava R. Prevalence of aspergillosis in chronic lung diseases. Indian J Med Microbiol 2001;19:201-5.

Table 1. Findings on computed tomography of chest.

TYPE OF LESION	n (%)
Fibrosis	242 (86.4%)
Bronchiectasis	226 (80.7%)
Cavity	129 (46.1%)
Nodules	129 (46.1%)
Aspergilloma	37 (13.2%)
Consolidation	31 (11.1%)
Ground Glass Opacity	25 (8.9%)
Bronchial Artery Hypertrophy	24 (8.6%)
Pleural Thickening	19 (6.8%)
Mucus Plug	18 (6.4%)
Others	7 (2.5%)
TYPE OF BRONCHIECTASIS	
Cystic	67 (30.6%)
Varicose	18 (8.2%)
Cylindrical	7 (3.2%)
Traction	176 (80.4%)
NUMBER OF CAVITIES	
1	74 (57.3%)
2	9 (6.9%)
>2	46 (35.65%)
SIDE OF LESION	
Right	73 (26.1%)
Left	65 (23.2%)
Bilateral	142 (50.7%)

Overlap was found in types of bronchiectasis in many patients therefore sum of individual types of bronchiectasis is more.

Table 2. Association between CPA and parameters.

Parameters	CPA		p-value
	Present (n=77)	Absent (n=203)	
Age (Years)	45.23 ± 14.26	43.56 ± 15.25	0.392[1]
Gender			0.743[2]
Male	45 (58.4%)	123 (60.6%)	
Female	32 (41.6%)	80 (39.4%)	
BMI (kg/m²)* **	**18.99 ± 3.17**	**20.60 ± 3.25**	**<0.001[3]**
Smoking Status			0.453[4]
Never Smoker	65 (84.4%)	179 (88.2%)	
Ex-Smoker	11 (14.3%)	23 (11.3%)	
Current Smoker	1 (1.3%)	1 (0.5%)	
Comorbidities: Diabetes Mellitus (Yes)	9 (12.0%)	11 (5.5%)	0.063[2]
Any History of MDR-TB (Yes)	5 (6.5%)	4 (2.0%)	0.120[4]
Any Residual Symptoms (Yes)	51 (66.2%)	110 (54.2%)	0.069[2]
Asymptomatic period after ATT completion (years)* **	**2.6 [0-6.2]**	**5.2 [0-10.8]**	**0.041[3]**
Duration of Symptoms (Years)	4.8 [0.9-8.8]	3.6 [0.6-6.6]	0.097[3]
Dyspnoea (Yes)	57 (74.0%)	148 (72.9%)	0.850[2]
Cough (Yes)* **	**75 (97.4%)**	**183 (90.1%)**	**0.044[2]**
Sputum (Yes)	73 (94.8%)	179 (88.2%)	0.099[2]
Hemoptysis (Yes)* **	**57 (74.0%)**	**110 (54.2%)**	**0.003[2]**
Loss of Appetite (Yes)* **	**47 (61.0%)**	**57 (28.1%)**	**<0.001[2]**
Loss of Weight (Yes)* **	**46 (59.7%)**	**48 (23.6%)**	**<0.001[2]**
FVC in litres (Absolute)* **	**1.9 ± 0.9**	**2.1 ± 0.9**	**0.025[3]**
FVC (%)* **	**60.1 ± 20.0**	**67.6 ± 20.2**	**0.019[1]**
FEV1 in litres (Absolute)	1.4 ± 0.7	1.6 ± 0.8	0.082[3]
FEV1 (%)	57.3 ± 22.6	63.3 ± 24.4	0.101[1]
Type of Impairment			0.120[2]
Normal	11 (20.4%)	46 (27.5%)	
Obstructive	1 (1.9%)	17 (10.2%)	
Restrictive	23 (42.6%)	59 (35.3%)	
Mixed	19 (35.2%)	45 (26.9%)	
Type of Lesion:			
Fibrosis	65 (84.4%)	177 (87.2%)	0.545[2]
Bronchiectasis	66 (85.7%)	160 (78.8%)	0.192[2]
Cavity * **	**51 (66.2%)**	**78 (38.4%)**	**<0.001[2]**
Nodules	42 (54.5%)	87 (42.9%)	0.080[2]
Aspergilloma * **	**26 (33.8%)**	**11 (5.4%)**	**<0.001[2]**
Pleural Thickening * **	**11 (14.3%)**	**8 (3.9%)**	**0.002[2]**
Segment Involved:			
Left Upper Lobe * **	**58 (75.3%)**	**121 (59.6%)**	**0.014[2]**
Left Lower Lobe * **	**37 (48.1%)**	**69 (34.0%)**	**0.030[2]**
Haemoglobin (g/dL)	11.84 ± 2.08	12.40 ± 1.69	0.096[3]
ESR (mm/Hr)* **	**61 [39-83]**	**48 [28-67]**	**0.002[3]**
CRP (mg/L)* **	**39 [0-79]**	**15 [28-67]**	**<0.001[3]**
Serum Total IgE (FEIA)	1586 [0-3932]	1777 [0-3776]	0.667[3]
Values of Specific IgG Against Aspergillus (mgA/L)* **	**87.9 [38.8 – 137]**	**21.4 [0-50.2]**	**<0.001[3]**
Values of Specific IgG Against Aspergillus (U/L)* **	**19.3 [10.9-27.8]**	**6.4 [3.7-9.2]**	**<0.001[3]**
Result of Specific IgG Against Aspergillus* **			**<0.001[4]**
Positive	**71 (92.2%)**	**16 (7.9%)**	
Negative	**5 (6.5%)**	**183 (90.1%)**	
Equivocal	**1 (1.3%)**	**4 (2.0%)**	
Fungal Culture (Positive)	2 (2.6%)	0 (0.0%)	0.075[4]
Bal Galactomannan* *(n=42)**	**3.5 [2.6-4.4]**	**1.0 [0.6-1.5]**	**<0.001[1]**

***Significant at p<0.05, 1: t-test, 2: Chi-Squared Test, 3: Wilcoxon-Mann-Whitney U Test, 4: Fisher's Exact Test

Table 3. Univariable and multivariable logistic regression analysis for risk factors associated with development of CPA.

Predictor/Risk Factor	Odds Ratio (95% CI)	p-value	Adjusted odds ratio (95% CI)	p-value
Low BMI	**0.84 (0.76-0.92)**	**<0.001**	**0.864 (0.77-0.96)**	**0.007**
Presence of cough	4.09 (0.93-17.97)	0.06	1.874 (0.33-10.48)	.475
Haemoptysis	2.41 (1.35-4.30)	0.003	1.831 (0.88-3.78)	.102
Loss of Appetite	4.01 (2.31-6.96)	<0.001	1.020 (0.11-9.25)	.986
Loss of Weight	4.79 (2.74-8.37)	<0.001	3.085 (0.33-28.39)	.320
Cavity	3.21 (1.85-5.56)	<0.001	1.062 (0.52-2.15)	.868
Presence of Aspergilloma	**8.89 (4.12-19.21)**	**<0.001**	**6.536 (2.46-17.34)**	**.000**
Pleural Thickening	3.63 (1.37-9.59)	0.009	2.199 (0.61-7.84)	.224
Elevated CRP	**1.02 (1.01-1.03)**	**<0.001**	**1.015 (1.002-1.028)**	**.027**

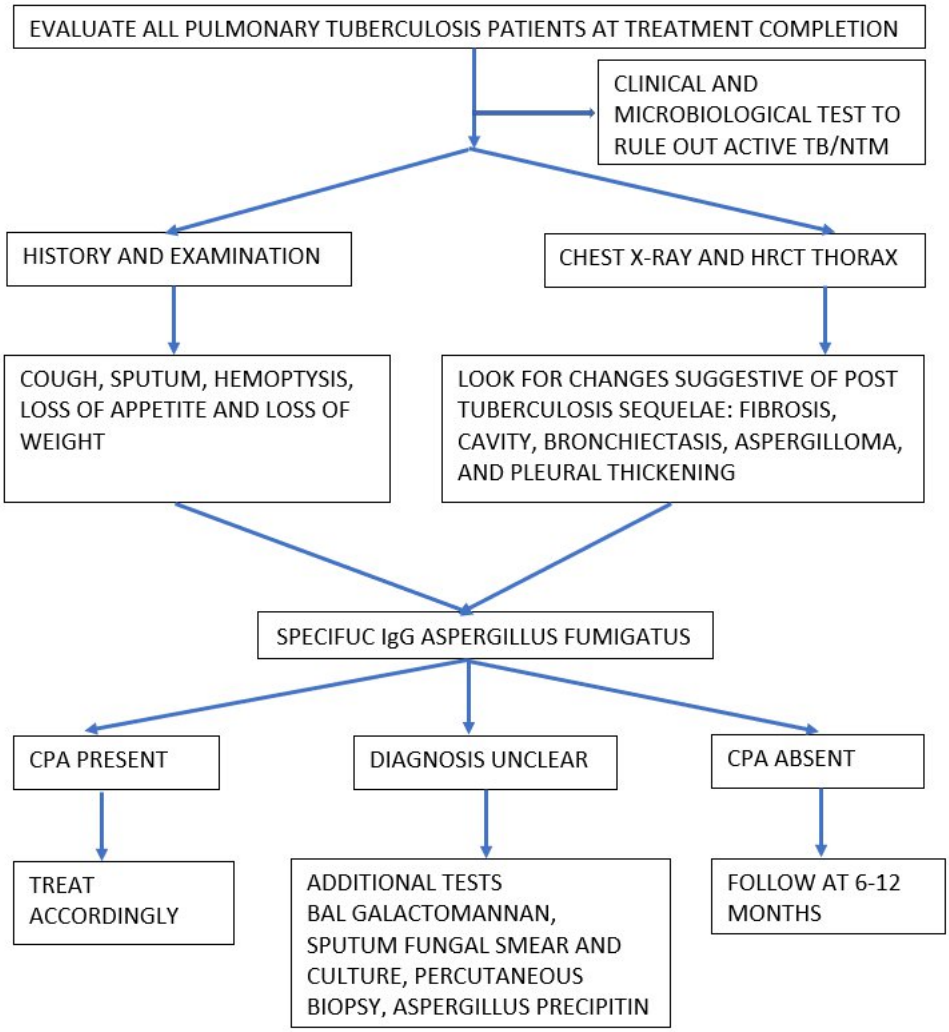


Figure 1. Structured approach for early diagnosis of CPA in post-TB patients.