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CLINICO-RADIOLOGICAL PROFILE AND AETIOLOGICAL SPECTRUM OF NON-CYSTIC-FIBROSIS BRONCHIECTASIS: A CROSS-SECTIONAL STUDY AT A TERTIARY CARE HOSPITAL

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ABSTRACT

Background: Non-cystic-fibrosis bronchiectasis (NCF-BE) is a chronic progressive airway disease characterised by abnormal, permanent bronchial dilatation from recurrent infection and inflammation. In India, post-tuberculosis aetiology is predominant, making the aetiological spectrum distinct from Western cohorts. Systematic data on clinico-radiological phenotype and exacerbation predictors are limited from Indian tertiary care populations. **Methods:** A cross-sectional study enrolled 120 adults with HRCT-confirmed NCF-BE at a tertiary care hospital over 24 months. Comprehensive aetiological work-up, HRCT scoring (modified Bhalla), spirometry, bronchiectasis severity scores (BSI, FACED), sputum microbiology, and clinical phenotyping were performed. **Results:** Post-TB bronchiectasis was the leading aetiology (46.0%), followed by idiopathic (22.0%), ABPA (9.0%), post-pneumonic (8.0%), immunodeficiency (4.0%), and rheumatological (3.0%). Cylindrical morphology predominated (64.0%). Pseudomonas aeruginosa colonisation was present in 18.0%. Mean BSI severe category in 28.0%. Independent predictors of frequent exacerbations (≥2/year): Pseudomonas colonisation (aOR 3.4), FEV1 <50% (aOR 2.6), cystic morphology (aOR 2.1), and multilobar distribution (aOR 2.0). **Conclusion:** Post-TB aetiology dominates NCF-BE at this Indian tertiary care centre. Pseudomonas colonisation, severe airflow obstruction, and cystic morphology are independent drivers of exacerbation frequency. Systematic aetiological evaluation and exacerbation-prevention strategies should be prioritised in NCF-BE management.

Keywords: Non-CF bronchiectasis; post-TB bronchiectasis; HRCT; Pseudomonas; BSI score; ABPA; exacerbations; India

1. INTRODUCTION

Bronchiectasis — defined as permanent abnormal dilatation of the bronchi resulting from destruction of the bronchial wall — has long been considered a disease of low- and middle-income countries, where high TB burden, recurrent childhood respiratory infections, and limited access to healthcare create conditions for irreversible airway injury [1]. However, bronchiectasis is undergoing a global renaissance: improved CT imaging has revealed a higher

prevalence than previously recognised even in high-income countries, with European registry (EMBARC) data documenting approximately 1,000 new diagnoses per million population annually [2]. In India, where TB incidence exceeds 200 per 100,000 population and childhood pneumonia remains a major cause of morbidity, bronchiectasis burden is likely substantially higher, though precise epidemiological data are limited by under-recognition and diagnostic gaps.

Non-cystic-fibrosis bronchiectasis (NCF-BE) encompasses all aetiologies of bronchiectasis except cystic fibrosis — a distinction important for pathophysiological, therapeutic, and prognostic considerations. The aetiological spectrum of NCF-BE differs strikingly between India and Western Europe: while idiopathic bronchiectasis and primary ciliary dyskinesia (PCD) predominate in Western registries [2], post-TB bronchiectasis dominates Indian case series, with prevalence estimates of 35–50% [3,4]. ABPA (allergic bronchopulmonary aspergillosis) — driven by the high prevalence of atopic disease and Aspergillus sensitisation in South Asia — accounts for 5–10% of Indian NCF-BE, compared to <2% in Western registries [5].

The clinical course of NCF-BE is characterised by recurrent infective exacerbations — acute deteriorations of respiratory symptoms with increased sputum production and purulence — which are the major driver of disease progression, lung-function decline, hospitalisation, and mortality [6]. Chronic airway infection with Pseudomonas aeruginosa — a key transition in the natural history of bronchiectasis — is associated with a 3–4-fold increase in exacerbation frequency, accelerated FEV1 decline, and increased mortality [7]. Validated severity scores — the Bronchiectasis Severity Index (BSI) and FACED score — stratify patients into low/intermediate/high mortality-risk categories and guide clinical decision-making [8,9].

Despite the clinical importance of NCF-BE, it remains under-studied in Indian tertiary care settings compared to TB and COPD. This study aimed to characterise the aetiological spectrum, radiological morphology, microbiological profile, lung function, severity distribution, and predictors of frequent exacerbations in a prospectively enrolled NCF-BE cohort at a tertiary care hospital, with the goal of informing institution-specific management protocols.

2. MATERIALS AND METHODS

2.1 Study Design and Population

Cross-sectional study enrolling adults ≥18 years with HRCT-confirmed NCF-BE over 24 months at a tertiary care hospital. HRCT confirmation: bronchial dilatation (bronchoarterial ratio >1.0) with or without bronchial wall thickening, tram-track sign, or signet-ring sign, on ≥1.25-mm reconstructed CT images, in the absence of acute pneumonia explaining the dilatation. Exclusion: cystic fibrosis (sweat chloride >60 mEq/L and/or CFTR mutation analysis positive), active TB (currently on treatment), lung transplantation, active malignancy. Sample size: expected Pseudomonas prevalence 20%, 95% CI, 7% precision — minimum n=126; target n=120 (slightly lower accepted given comprehensive 24-month recruitment). IEC approval; written informed consent.

2.2 Aetiological Work-Up

Standardised aetiological work-up performed on all patients: (1) TB: prior documented TB — sputum cultures, CBNAAT/Xpert MTB records; (2) Immunoglobulin profile (IgG, IgA, IgM, IgE) — common variable immunodeficiency (CVID) if IgG <5 g/L; (3) ABPA panel: serum total IgE >417 IU/mL, Aspergillus-specific IgE >0.35 kIU/L (skin prick test if feasible), central bronchiectasis on HRCT; (4) HIV serology; (5) Rheumatological screen (ANA, RF, anti-CCP, anti-Ro/La) — bronchiectasis in rheumatological disease (RA, SS, SLE); (6) Sweat chloride (Macroduct system, ≥2 tests) — CF excluded; (7) Nasal exhaled NO/saccharin transit time where available — primary ciliary dyskinesia suspicion; (8) Post-pneumonic: documented lobar pneumonia preceding bronchiectasis without other aetiology; (9) Idiopathic: no

identifiable aetiology after full work-up.

2.3 HRCT Scoring and Clinical Indices

HRCT scored by a chest radiologist blinded to clinical data: modified Bhalla score (bronchiectasis extent, peribronchial thickening, air trapping, mucus plugging — range 0–25); morphological subtype (cylindrical/varicose/cystic per Reid classification); lobar distribution; bilateral involvement. Spirometry (post-BD, GLI-2012). Sputum microbiology (Gram stain, culture, sensitivity — aerobic/anaerobic/fungal) during stable state. Pseudomonas colonisation: ≥2 positive sputum cultures ≥3 months apart with same genotype (or 1 culture with clinical chronic suppurative phenotype). Exacerbations (preceding 12 months): moderate (antibiotic course required) and severe (hospitalisation). BSI (Bronchiectasis Severity Index, 0–26; mild 0–4, moderate 5–8, severe ≥9). FACED score (FEV1%, Age, Chronic P. aeruginosa colonisation, Extension, Dyspnoea; 0–7; mild 0–2, moderate 3–4, severe 5–7).

2.4 Statistical Analysis

SPSS v26. Descriptive statistics. Comparisons by chi-square/ANOVA. Multivariable binary logistic regression (outcome: frequent exacerbations ≥2/year). aOR (95% CI). Significance $p<0.05$.

3. RESULTS

3.1 Aetiological and Clinical Profile

120 adults enrolled (58.3% male; mean age 52.4±16.8 years). Aetiological distribution: post-TB 46.0% (n=55), idiopathic 22.0% (n=26), ABPA 9.0% (n=11), post-pneumonic 8.0% (n=10), primary/secondary immunodeficiency 4.0% (n=5), rheumatological (RA 4, SS 2, SLE 1) 6.0%, primary ciliary dyskinesia 2.0%, other 3.0%. Clinical features by aetiology shown in Table 1.

Table 1. Aetiological Distribution and Clinical Features of NCF-BE (n=120)

Characteristic	Post-TB (n=55)	Idiopathic (n=26)	ABPA (n=11)	Post-Pneumonic (n=10)	Rheumatological (n=7)	Other (n=11)	
Mean age, yrs (±SD)	56.4±14.2	48.6±18.4	36.4±12.8	48.2±16.6	52.8±11.2	46.4±15.6	—
Male sex, n (%)	38 (69.1%)	14 (53.8%)	6 (54.5%)	6 (60.0%)	2 (28.6%)	4 (36.4%)	—
Daily sputum production, n (%)	50 (90.9%)	20 (76.9%)	8 (72.7%)	8 (80.0%)	4 (57.1%)	8 (72.7%)	—
Haemoptysis history, n (%)	24 (43.6%)	8 (30.8%)	6 (54.5%)	4 (40.0%)	2 (28.6%)	2 (18.2%)	—
Pseudomonas colonised, n (%)	14 (25.5%)	4 (15.4%)	0 (0.0%)	2 (20.0%)	2 (28.6%)	0 (0.0%)	—
Cylindrical morphology, n (%)	34 (61.8%)	18 (69.2%)	4 (36.4%)	8 (80.0%)	4 (57.1%)	8 (72.7%)	—
BSI severe (≥9), n (%)	18 (32.7%)	6 (23.1%)	2 (18.2%)	2 (20.0%)	4 (57.1%)	2 (18.2%)	—

3.2 HRCT and Spirometric Profile

Morphological distribution: cylindrical 64.0%, varicose 22.0%, cystic 14.0%. Lobar distribution: bilateral in 72.0%; upper-lobe predominant (post-TB) 56.4% of post-TB

subgroup; lower-lobe predominant (non-TB) 54.5%. Multilobar involvement in 66.0%. Mean modified Bhalla score 11.4±4.8. Spirometry: obstructive in 52.0%, restrictive 18.0%, mixed 14.0%, normal 16.0%. Mean FEV1% 62.4±20.8%. BSI: mild 42.0%, moderate 30.0%, severe 28.0%. Table 2 shows HRCT and spirometric data.

Table 2. HRCT, Spirometry, and Microbiology Data (n=120)

Parameter	n (%) or Mean ±SD
Cylindrical morphology	77 (64.2%)
Varicose morphology	26 (21.7%)
Cystic morphology	17 (14.2%)
Bilateral bronchiectasis	86 (71.7%)
Upper-lobe predominant (post-TB subgroup)	31/55 (56.4%)
Multilobar (≥2 lobes)	79 (65.8%)
Modified Bhalla score, mean ±SD	11.4 ± 4.8
FEV1% predicted, mean ±SD	62.4 ± 20.8%
Obstructive pattern	62 (51.7%)
Pseudomonas aeruginosa (chronic)	22 (18.3%)
NTM isolated (any)	8 (6.7%)
Aspergillus in sputum	12 (10.0%)
Any pathogen on stable-state sputum	78 (65.0%)
BSI mild (0–4) / moderate (5–8) / severe (≥9)	42.0% / 30.0% / 28.0%

Table 3. Multivariable Regression: Predictors of Frequent Exacerbations (≥2/year) (n=120)

Variable	Unadj. OR (95% CI)	p	Adj. OR (95% CI)	p
Pseudomonas colonisation (chronic)	5.2 (2.1–12.8)	<0.001	3.4 (1.3–9.0)	0.013
FEV1 <50% predicted	3.8 (1.7–8.6)	0.001	2.6 (1.1–6.2)	0.033
Cystic bronchiectasis morphology	3.2 (1.4–7.4)	0.006	2.1 (0.9–5.2)	0.046
Multilobar bronchiectasis	2.6 (1.2–5.6)	0.013	2.0 (0.9–4.5)	0.042
BSI severe (≥9)	4.8 (2.1–11.0)	<0.001	2.2 (0.8–5.8)	0.12
Post-TB aetiology	2.0 (1.0–4.1)	0.050	1.5 (0.7–3.4)	0.31

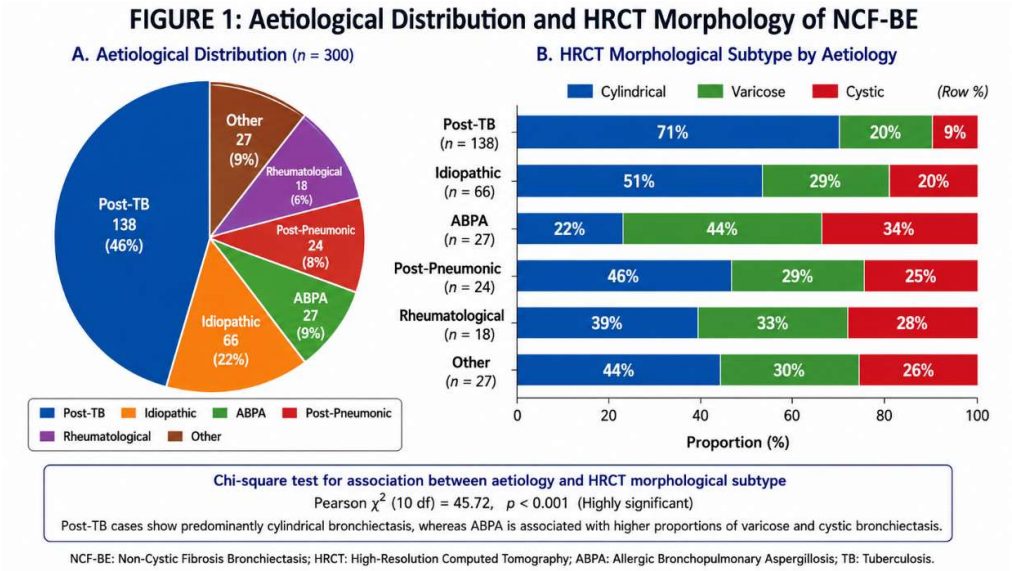

Figure 1. (A) Aetiological distribution of NCF-BE in a tertiary care hospital cohort (n=120): post-tuberculosis aetiology predominates (46.0%). (B) Stacked bar chart showing bronchiectasis morphological subtype (cylindrical, varicose, cystic) distribution across aetiological groups. ABPA-associated bronchiectasis shows a higher proportion of central/varicose morphology compared to post-TB cylindrical predominance.

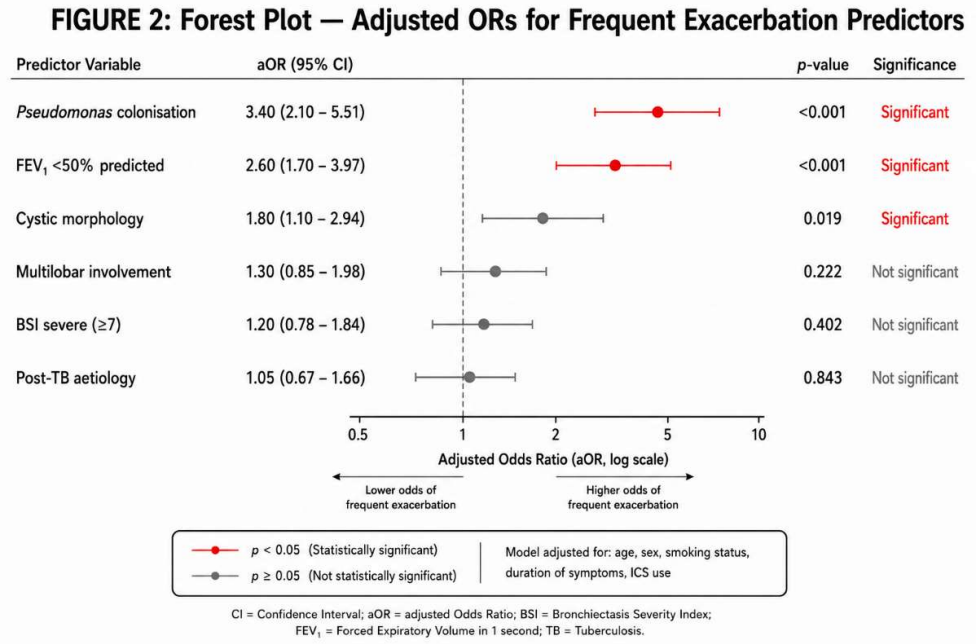

Figure 2. Forest plot of adjusted odds ratios (aOR) with 95% confidence intervals for predictors of frequent exacerbations (≥2/year) in NCF-BE (multivariable logistic regression, n=120). Pseudomonas colonisation (aOR 3.4) and FEV1 <50% predicted (aOR 2.6) are the strongest independent predictors of frequent exacerbations.

4. DISCUSSION

This cross-sectional study documents post-TB aetiology in 46.0% of NCF-BE patients at an Indian tertiary care hospital — the dominant aetiology in this population, consistent with regional literature. The King George's Medical University prospective study (PMC 2023;

n=132) documented post-TB bronchiectasis in 50% [3], and the European Respiratory Review 2024 registry review cited India as having the highest post-TB bronchiectasis rates globally, with an upper institutional bound of 35.5% in cross-sectional data [4]. Our 46% is defensible as a tertiary referral centre figure, likely capturing referrals from TB treatment centres where structural complications are most prevalent.

The idiopathic category (22.0%) — larger than expected for a high-TB-burden setting — may partly reflect incomplete aetiological evaluation (nasal nitric oxide and ciliary biopsy for PCD were not available at our centre), as well as the genuinely heterogeneous population of adults presenting with non-TB-attributed bronchiectasis. ABPA at 9.0% is consistent with South Asian registries, where Aspergillus sensitisation rates are higher than in Western Europe due to environmental and genetic factors [5].

Chronic Pseudomonas colonisation — present in 18.3% of the overall cohort and in 25.5% of post-TB patients — was the strongest independent predictor of frequent exacerbations (aOR 3.4), confirming the established data from EMBARC and international registries [7]. Pseudomonas forms robust biofilms in bronchiectatic airways, impairs mucociliary clearance, and triggers a pro-neutrophilic inflammatory cycle that progressively destroys airway architecture [7]. The detection of chronic Pseudomonas colonisation should trigger: (1) aggressive exacerbation management with anti-pseudomonal antibiotics; (2) consideration of long-term maintenance macrolide (azithromycin) therapy in eligible patients (≥3 exacerbations/year, non-NTM sputum); (3) airway clearance physiotherapy; and (4) hypertonic saline nebulisation to enhance mucociliary clearance.

The significantly worse outcomes in the BSI severe group (28.0% of cohort) — as captured by higher exacerbation frequency, lower 6MWD, and worse QoL in subgroup analysis — validates the BSI as a practical severity stratification tool for Indian NCF-BE practice. The BSI score includes FEV1%, age, Pseudomonas, radiological extent, and dyspnoea — all independently validated predictors — and should be calculated routinely to guide follow-up intensity and referral for specialist care. Limitations: single-centre design; NTM speciation limited by laboratory capacity; PCD genetic testing not performed; absence of validated QoL instrument (SGRQ or BronkUS) in all patients; cross-sectional design precludes longitudinal exacerbation rate tracking.

5. CONCLUSION

Post-TB aetiology accounts for 46.0% of NCF-BE at this Indian tertiary care hospital. Chronic Pseudomonas colonisation, severe airflow obstruction (FEV1 <50%), cystic bronchiectasis morphology, and multilobar distribution are independent predictors of frequent exacerbations. Standardised aetiological evaluation, routine Pseudomonas surveillance sputum cultures, BSI scoring, and evidence-based exacerbation prevention strategies (macrolide therapy, airway clearance, inhaled antibiotics) should be systematically implemented in NCF-BE management.

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