"EVALUATION OF PREVALENCE AND PREDISPOSING FACTORS FOR BRONCHIECTASIS IN COPD PATIENTS – A CROSS SECTIONAL STUDY."

By

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With reference to the above, we wish to inform you that your proposed research project titled **"EVALUATION OF PREVALENCE AND PREDISPOSING FACTORS FOR BRONCHIECTASIS IN COPD PATIENTS- A CROSS SECTIONAL STUDY",** is ethical and justifiable. The proposed research project has been cleared by the JNMC Institutional Ethics Committee.

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LIST OF ABBREVIATIONS

- AATD: Alpha 1 Antitrypsin Deficiency
- ABG: Arterial Blood Gas
- ABPA: Allergic Bronchopulmonary Aspergillosis
- ACO: Asthma COPD Overlap
- AECOPD: Acute Exacerbations of Chronic Obstructive Pulmonary Disease
- BCO: Bronchiectasis-COPD overlap
- BHQ: Bronchiectasis Health Questionnaire
- BMI: Body Mass Index
- BSI: Bronchiectasis Severity Index
- CAT: COPD Assessment Test
- CF: Cystic fibrosis
- CKD: Chronic Kidney Disease
- CLD: Chronic Liver Disease
- COPD: Chronic Obstructive Pulmonary Disease
- CPET: Cardiopulmonary Exercise Testing
- EMBARC: European Multicentre Bronchiectasis Audit and Research Collaboration
- FVC: Forced Vital Capacity
- GBD: Global Burden of Disease
- GERD: Gastroesophageal Reflux Disease
- GOLD: Global Initiative for Chronic Obstructive Lung Disease
- HRCT: High Resolution Computed Tomography

- HTN: Hypertension
- ICS: Inhaled Corticosteroids
- IHD: Ischemic Heart Disease
- JVP: Jugular Venous Pressure
- LCQ: Leicester Cough Questionnaire
- MMRC: Modified Medical Research Council
- PEFR: Peak Expiratory Flow Rate
- PEP: Positive Expiratory Pressure
- PFT: Pulmonary Function Test
- PLB: Lip Breathing
- SGRQ: Respiratory Questionnaire
- WBC: White Blood Cells

ABSTRACT

Introduction:

Chronic Obstructive Pulmonary Disease (COPD) is a progressive respiratory disorder marked by persistent airflow limitation and chronic inflammation. Bronchiectasis—characterized by irreversible bronchial dilatation—is increasingly recognized as a common comorbidity in COPD, forming the Bronchiectasis-COPD Overlap (BCO) phenotype. This overlap is associated with more severe symptoms, frequent exacerbations, greater bacterial colonization, and poorer outcomes. However, bronchiectasis remains underdiagnosed, especially in resource-limited settings like India, where routine HRCT is not always feasible. Identifying its prevalence and risk factors in COPD is essential for early diagnosis, appropriate management, and improved prognosis.

Objectives:

To determine the prevalence of bronchiectasis in COPD patients and identify associated predisposing risk factors.

Methods:

This hospital-based cross-sectional study included 146 COPD patients diagnosed using GOLD 2024 criteria at a tertiary care center. All underwent clinical evaluation, spirometry, HRCT thorax, 6-minute walk test, sputum culture, and relevant labs. Bronchiectasis was diagnosed by HRCT. Patients were grouped as COPD with or without bronchiectasis and compared for clinical, functional, and radiological parameters. Statistical analysis used t-tests and Chi-square tests, with $p < 0.05$ considered significant.

Results:

Among 146 COPD patients, 34 (23.3%) had bronchiectasis on HRCT. These patients

showed significantly worse lung function (FEV_1: 54.5 ± 10.6% vs. 59.4 ± 12.4%, p = 0.026; FVC: 77.7 ± 15.0% vs. 86.3 ± 18.6%, p = 0.008; PEFR: 42.4 ± 14.9 vs. 49.9 ± 19.1, p = 0.018), higher symptom scores (CAT: 18.9 ± 4.4 vs. 14.9 ± 4.1, p < 0.001; mMRC: 2.4 ± 0.7 vs. 1.7 ± 0.8, p < 0.001), and reduced 6-minute walk distance (307.6 ± 70.8 m vs. 382.7 ± 126.9 m, p = 0.001). They also had longer symptom duration (9.4 ± 4.5 vs. 7.1 ± 3.8 years, p = 0.009) and more hospitalizations over two years (3.1 ± 0.8 vs. 1.3 ± 0.9, p < 0.001). Comorbidities such as hypertension (61.8% vs. 39.3%, p = 0.035), diabetes (50.0% vs. 25.9%, p = 0.015), and ischemic heart disease (35.3% vs. 13.4%, p = 0.009) were significantly more common in the bronchiectasis group. Radiologically, cystic bronchiectasis was most frequent (38.2%), followed by traction (32.4%) and cylindrical (23.5%) types, with bilateral (58.8%) and distal (70.6%) involvement predominating. Pseudomonas aeruginosa was isolated in 11.76% of bronchiectasis patients (p = 0.018). Chronic bronchitis phenotype was more common in those with bronchiectasis (67.6% vs. 21.4%, p < 0.001).

Conclusion:

Bronchiectasis was present in 23.3% of COPD patients and was associated with more severe airflow limitation, higher symptom burden, reduced exercise capacity, frequent hospitalizations, and greater comorbidity. The overlap phenotype also showed higher prevalence of chronic bronchitis and Pseudomonas colonization. These findings highlight BCO as a distinct, severe phenotype. Routine HRCT screening in symptomatic or frequently exacerbating COPD patients may enable earlier diagnosis and more tailored management.

Keywords: COPD, bronchiectasis, bronchiectasis-COPD overlap, HRCT thorax, PFT.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

LIST OF PHOTOGRAPHS

INTRODUCTION

Chronic obstructive pulmonary disease (COPD) is a leading cause of morbidity and mortality worldwide, characterized by persistent airflow limitation and chronic inflammation of the airways [1]. While COPD primarily involves emphysema and chronic bronchitis, an increasing body of research highlights the coexistence of bronchiectasis in a subset of COPD patients, a condition termed COPD-bronchiectasis overlap. [2]

Bronchiectasis is a chronic respiratory disease marked by permanent and irreversible dilation of the bronchi, leading to mucus accumulation, recurrent infections, and worsening lung function [3].

The development of bronchiectasis In COPD patients is attributed to multiple predisposing factors, including recurrent airway infections, impaired mucociliary clearance, chronic inflammation, and host immune dysregulation[2], Smoking history, frequent exacerbations, chronic bacterial colonization (especially with Pseudomonas aeruginosa), and underlying genetic predispositions such as alpha-1 antitrypsin deficiency contribute to an increased risk of bronchiectasis in COPD patients [2]. These factors lead to a vicious cycle of inflammation, airway remodeling, and progressive lung function decline, making COPD-bronchiectasis overlap a clinically significant phenotype requiring further investigation [4,5].

Despite its clinical significance, bronchiectasis in COPD remains underdiagnosed, primarily due to reliance on symptoms and spirometry in COPD diagnosis rather than routine HRCT scan, which is the gold standard for detecting bronchiectasis [2]. As a result, many cases remain unidentified until advanced disease

stages, when exacerbations become frequent and difficult to manage. Understanding the true prevalence of bronchiectasis in COPD patients and identifying the key predisposing factors can help in early diagnosis and targeted intervention [5].

Bronchiectasis is increasingly recognized as a significant comorbidity in patients with Chronic Obstructive Pulmonary Disease (COPD), with its prevalence varying widely across different studies and populations. Chalmers et al [4] conducted a meta-analysis reviewing data from six observational studies, reporting that up to 54.3% of COPD patients had radiological evidence of bronchiectasis. The prevalence varied significantly across individual studies, with some reporting as low as 4% and others as high as 50%, reflecting differences in diagnostic criteria and study populations[4].

In North-East India, a cross-sectional study by Sangtam et al [5] found that 17.7% of COPD patients had coexisting bronchiectasis. The study highlighted that the presence of bronchiectasis was associated with lower FEV1 values and an increased frequency of exacerbations. Moreover, a majority of these patients had a high Bronchiectasis Severity Index (BSI) score, indicating a more severe disease phenotype[5].

Dou et al [6] conducted a study involving 1,739 COPD patients and found that 8.1% had bronchiectasis. Notably, the prevalence was significantly higher in patients with emphysema-predominant COPD (16.5%) compared to those without emphysema predominance (10.3%). This finding suggests a strong association between emphysema and bronchiectasis, highlighting the potential role of lung structure and disease phenotype in bronchiectasis development[6].

A study by Gatheral et al [7] reported a particularly high prevalence of bronchiectasis, identifying its presence in 69% of 406 COPD patients. The severity of bronchiectasis varied, with 40% of cases classified as minor and 8% as severe. This study also found a significant correlation between bronchiectasis and increased respiratory infections and hospitalizations, reinforcing the impact of bronchiectasis on COPD progression and patient outcomes[7].

In Taiwan, Lu et al[8] utilized a nationwide database from 2005 to 2018 to examine the incidence of bronchiectasis in COPD patients. Their findings indicated an incidence rate of 87.83 per 10,000 person-years among COPD patients, compared to 69.80 per 10,000 person-years in the non-COPD group. The adjusted hazard ratio showed that the risk of bronchiectasis was 1.9 times higher in COPD patients, with incidence increasing significantly with age[8].

Overall, the prevalence of bronchiectasis in COPD patients varies considerably, ranging from 8% to as high as 69%, depending on the study population and diagnostic criteria used. Factors such as emphysema predominance, advanced COPD stages, and recurrent infections are strongly associated with a higher prevalence of bronchiectasis. These findings underscore the need for early detection and targeted management to improve outcomes for COPD patients with bronchiectasis[4–8].

Gap in Literature and Need for the Study [4-8]

Despite the recognition of Bronchiectasis-COPD Overlap (BCO) as a distinct phenotype associated with worse clinical outcomes, several research gaps persist, particularly in the Indian context[4–8]. One major challenge is the wide variability in prevalence estimates, with studies reporting rates ranging from 4% to 72%,

highlighting the need for region-specific data to determine the true burden of this overlap condition. Differences in diagnostic criteria, study populations, and imaging availability contribute to these inconsistencies[4–8]. Additionally, most research on BCO has been conducted in Western populations, where factors such as genetic predisposition, environmental exposures, and healthcare access differ significantly from those in India. The impact of biomass fuel exposure and tuberculosis-related lung damage, which are more prevalent in the Indian population, remains largely unexplored in the context of BCO[4–8].

Another critical gap is the lack of clarity regarding predisposing factors for bronchiectasis in COPD patients in India. While previous studies have identified low FEV1, chronic Pseudomonas aeruginosa colonization, frequent exacerbations, and emphysema predominance as key risk factors, data specific to Indian patients are insufficient. Identifying these risk factors could help in early risk stratification, targeted interventions, and personalized treatment approaches[4–8]. Furthermore, the long-term prognosis of COPD patients with coexisting bronchiectasis remains poorly understood, particularly in terms of disease progression, hospitalization rates, and quality of life[4–8].

A major limitation in current practice is the underutilization of high-resolution computed tomography (HRCT) in COPD diagnosis. HRCT is the gold standard for detecting bronchiectasis, yet it is not routinely performed in COPD patients in India, leading to underdiagnosis and delayed treatment. Without systematic screening, many cases of BCO go unrecognized until frequent exacerbations and infections make management more difficult. Implementing systematic screening protocols could facilitate early identification, timely interventions, and improved clinical outcomes[4–8].

Addressing these gaps is essential for optimizing diagnosis, treatment, and patient care in India, ensuring better outcomes for COPD patients at risk of bronchiectasis[4–8].

AIMS AND OBJECTIVES

Primary objective :

To study the prevalence of bronchiectasis in chronic obstructive pulmonary disease patients.

Secondary objective :

To evaluate the risk factors associated with development of bronchiectasis in COPD patients.

REVIEW OF LITERATURE

Understanding COPD :

Global Initiative for Chronic Obstructive Lung Disease (GOLD) 2024 now defines COPD as "a heterogeneous lung condition characterized by chronic respiratory symptoms (dyspnea, cough, sputum production, and/or exacerbations) due to abnormalities of the airways (bronchitis, bronchiolitis) and/or alveoli (emphysema) that cause persistent, often progressive, airflow obstruction." [1]

Burden of COPD

COPD is a leading cause of morbidity and mortality worldwide, with a significant and growing economic and social impact. The prevalence, morbidity, and mortality of COPD vary across different countries. Generally, the prevalence of COPD correlates with tobacco smoking rates, but in many regions, outdoor, occupational, and household air pollution (such as the burning of wood and biomass fuels) also contribute significantly to the risk of developing COPD.The prevalence and burden of COPD are expected to rise in the coming decades due to ongoing exposure to COPD risk factors and the aging global population.

According to the systematic review and meta-analysis conducted by AL Wachami et al [9], the global prevalence of Chronic Obstructive Pulmonary Disease (COPD) in individuals aged 40 years and older is estimated to be 12.64% (95% CI: 10.75%–14.65%) when using the fixed ratio (FR) criteria for diagnosis. The study revealed that the prevalence is significantly higher in men (15.47%) compared to women (8.79%). Regionally, the highest prevalence was recorded in the Americas (22.93%), followed by the Southeast Asia region (19.48%), while the lowest

prevalence was observed in the Eastern Mediterranean region (7.95%). The findings highlight significant regional and gender disparities in the burden of COPD, emphasizing the need for targeted prevention and management strategies to reduce the global impact of this chronic respiratory disease.

According to Daniel et al [10], the prevalence of Chronic Obstructive Pulmonary Disease (COPD) in India is 7.4% (95% CI: 5.0%–9.8%) among adults, based on pooled data from eight studies involving 8,569 participants. The prevalence of COPD varies significantly across different regions of India, with the highest prevalence in northern India (10.4%), followed by the eastern region (6.8%) and the southern region (3.7%). Gender-wise, COPD is more common among males (11.4%) than females (7.4%), and it is notably higher in urban areas (11.4%) compared to rural areas (5.6%). This disparity highlights the role of environmental exposure, smoking, and occupational hazards in urban regions as contributing factors. The study emphasizes the importance of implementing nationwide community-based surveys to better assess the true burden of COPD in India and the need for adequate training and resources at the primary healthcare level for early diagnosis and management of the disease.

A study conducted by the India State-Level Disease Burden Initiative CRD Collaborators[11] analyzed the burden of chronic respiratory diseases (CRDs), particularly COPD and asthma, in India from 1990 to 2016 using Global Burden of Disease (GBD) data. India accounted for 32% of global CRD-related disability-adjusted life years (DALYs), with COPD contributing 75.6% and asthma 20.0%. The prevalence of COPD rose from 3.3% in 1990 to 4.2% in 2016. Major risk factors included air pollution (53.7%), tobacco use (25.4%), and occupational hazards (16.5%). The study highlights the need for targeted interventions to reduce air pollution, smoking, and improve CRD management in India.

Pathophysiology :

Chronic obstructive pulmonary disease (COPD) is marked by persistent airflow obstruction that is not easily reversible, along with an abnormal inflammatory response in the lungs. This inflammation involves both the innate and adaptive immune systems reacting to long-term exposure to harmful particles and gases, particularly from cigarette smoke.

While all smokers experience some degree of lung inflammation, those who develop COPD have an exaggerated or abnormal response to these toxic agents. This heightened response can lead to excessive mucus production (chronic bronchitis), tissue damage (emphysema), and a disruption of normal repair and defence processes, causing inflammation and fibrosis in the small airways (bronchiolitis) [1].

These pathological changes lead to increased resistance in the small conducting airways, greater lung compliance, air trapping, and progressively worsening airflow obstruction—all hallmarks of COPD. We have a strong understanding of the cellular and molecular mechanisms that underlie the pathological changes seen in COPD.

Mucous Hyper secretion and Ciliary Dysfunction

Mucous hyper secretion leads to a chronic productive cough, which is a hallmark of chronic bronchitis, though it is not always associated with airflow obstruction, and not all COPD patients experience symptomatic mucous hypersecretion. This increased mucus production occurs due to squamous metaplasia, an increase in goblet cells, and the enlargement of bronchial submucosal glands in response to chronic exposure to irritating particles and gases. Ciliary dysfunction

results from squamous metaplasia of the epithelial cells, impairing the mucociliary escalator and making it harder to expectorate mucus.[1]

Airflow Obstruction and Hyperinflation or Air Trapping

Airflow obstruction primarily occurs in the small conducting airways, which are less than 2 mm in diameter, due to inflammation, airway narrowing (airway remodeling), and inflammatory exudates in these small airways. Other contributing factors include a loss of lung elastic recoil from the destruction of alveolar walls and the breakdown of alveolar support structures.

The airway obstruction progressively leads to air trapping during exhalation, causing hyperinflation at rest and dynamic hyperinflation during physical activity. This hyperinflation reduces the inspiratory capacity and functional residual capacity during exercise. As a result, individuals experience breathlessness and reduced exercise capacity, which are typical in COPD. Spirometry is the most effective method to measure airflow obstruction and is necessary for diagnosing COPD. [1]

Gas Exchange Abnormalities

In advanced COPD, gas exchange abnormalities occur, characterized by arterial hypoxaemia, sometimes accompanied by hypercapnia. The main mechanism behind these abnormalities is an abnormal distribution of ventilation and perfusion ratios due to the structural changes in the lungs caused by COPD. The degree of impairment in the diffusing capacity for carbon monoxide is strongly linked to the severity of emphysema. [1]

Pulmonary Hypertension

Pulmonary hypertension typically develops in the later stages of COPD, often when significant gas exchange abnormalities are present. Contributing factors include pulmonary arterial constriction caused by hypoxia, endothelial dysfunction, and remodelling of the pulmonary arteries (such as smooth muscle hypertrophy and hyperplasia).

The destruction of the pulmonary capillary bed also plays a role. These structural changes in the pulmonary arterioles lead to persistent pulmonary hypertension, as well as right ventricular hypertrophy, enlargement, and dysfunction, which can result in cor pulmonale. [1]

Causes and Risk Factors

COPD results from interactions between genetic (G) and environmental (E) factors throughout an individual's lifetime (T), a concept known as GETomics. These factors can damage the lungs or interfere with their normal development and aging processes. [1]

The primary environmental causes of COPD are tobacco smoking and exposure to toxic particles and gases from both household and outdoor air pollution. However, other environmental and host factors—such as abnormal lung development and accelerated lung aging—can also play a role. [1]

The most well-known (though epidemiologically rare) genetic risk factor for COPD is mutations in the SERPINA1 gene, which leads to α1-antitrypsin deficiency. Additionally, other genetic variations, each with a smaller effect, are associated with reduced lung function and an increased risk of developing COPD. [1]

Taxonomic Classification of COPD (GOLD 2024)[1]

Traditionally viewed as a smoking-related disease, COPD is now recognized as a heterogeneous condition with diverse etiologies. GOLD 2024 introduces a broader classification incorporating non-smoking-related COPD subtypes to enhance diagnosis and treatment.

1. Etiological Classification (Etiotypes of COPD)[1]

Smoking-Related COPD: The most common form, driven by tobacco exposure, leading to inflammation, mucus hypersecretion, and emphysema.

Environmental COPD: Resulting from exposure to biomass fuel, industrial pollutants, and occupational hazards, prevalent in lower-income regions.

Genetic COPD: A rare form, including α1-antitrypsin deficiency, linked to early-onset emphysema.

Asthma-COPD Overlap (ACO): Features of both diseases, with reversible airflow limitation.

Infection-Related COPD: Linked to prior severe respiratory infections, tuberculosis, or chronic bronchiectasis, causing persistent inflammation and lung damage.

2. Phenotypic Classification[1]

Emphysema-Predominant COPD: Characterized by alveolar destruction, air trapping, and progressive dyspnea, common in smokers.

Chronic Bronchitis-Predominant COPD: Defined by chronic cough and sputum production, increasing exacerbation risk.

Frequent Exacerbator Phenotype: Marked by recurrent exacerbations, leading to rapid lung function decline.

3. Severity-Based Classification (GOLD Staging System)[1]

COPD severity is determined via spirometry (FEV1/FVC ratio & FEV1 % predicted):

GOLD 1 (Mild): FEV1 ≥ 80% predicted

GOLD 2 (Moderate): FEV1 50–79% predicted

GOLD 3 (Severe): FEV1 30–49% predicted

GOLD 4 (Very Severe): FEV1 < 30% predicted

4. Emerging Concepts: Pre-COPD, Young COPD, and PRISm[1]

Pre-COPD: Early lung abnormalities (mild emphysema, airway remodeling) without airflow obstruction.

PRISm : FEV1/FVC ≥ 0.7, but reduced FEV1 (<80% predicted), indicating high COPD risk.

Young COPD: Found in 20–50-year-olds, often due to impaired lung growth or early decline in lung function, requiring early intervention.

5. Multidimensional Classification (ABE-GOLD 2024)[1]

Group A: Low symptoms, low exacerbation risk.

Group B: High symptoms, low exacerbation risk.

Group E: High exacerbation risk, irrespective of symptoms.

The expanded taxonomic classification in GOLD 2024 enables personalized COPD management by recognizing diverse causes and phenotypes.

Clinical Presentation of COPD [12,13]

COPD manifests with progressive symptoms, significantly affecting quality of life.

1. Dyspnea: Initially exertional but progresses to breathlessness at rest, caused by airflow obstruction and lung hyperinflation.
2. Chronic Cough: Persistent, worsening over time, often triggered by tobacco and pollutants.
3. Sputum Production: Excessive mucus production, often worsening during exacerbations.
4. Exacerbations: Acute worsening of symptoms, commonly triggered by infections or pollutants, leading to hospitalizations.
5. Wheezing: Occurs due to airway narrowing, often worsening during exacerbations.
6. Chest Tightness: Discomfort aggravated by exertion or exacerbations.
7. Fatigue: Affects daily activities due to increased breathing effort and systemic inflammation.
8. Weight Loss & Muscle Wasting: Common in advanced COPD, leading to reduced exercise capacity and functional decline.

Early detection and personalized management are crucial to improve outcomes and slow disease progression.

Physical Findings in COPD [12,13]

A thorough clinical examination helps assess COPD severity and complications.

1. Wheeze Absence in Early Stages: Becomes more evident as airflow limitation progresses.
2. Hyperinflation Signs: Barrel chest, diminished breath sounds, and tripod posture suggest advanced disease.
3. Pursed-Lip Breathing (PLB): A compensatory mechanism to improve ventilation and airway patency.
4. Systemic Manifestations – Cor Pulmonale: Right ventricular hypertrophy and loud P2 heart sound indicate pulmonary hypertension.
5. Clubbing: Rare in COPD but may indicate lung cancer or bronchiectasis.
6. COPD Subtypes:

Pink Puffers (Emphysematous Type): Severe weight loss, muscle wasting, pursed-lip breathing.

Blue Bloaters (Chronic Bronchitis Type): Cyanosis, peripheral edema, ventilation-perfusion mismatch. (Table A)

Table A. Comparison of Pink Puffers and Blue Bloaters [13]

Features	Pink Puffers (Emphysema)	Blue Bloaters (Chronic Bronchitis)
Body Type	Thin due to increased energy expenditure	Overweight and cyanotic due to chronic hypoxemia
Posture	Tend to lean forward while sitting	No characteristic posture
Chest Appearance	Barrel chest (increased AP diameter)	No characteristic chest deformity
Breathing Pattern	Tachypnea with prolonged expiration through pursed lips	Normal or slightly increased respiratory rate
Use of Accessory Muscles	Uses accessory muscles (especially strap muscles in neck)	No apparent use of accessory muscles
Cough & Sputum	Minimal cough and sputum production	Chronic cough with sputum production
Gas Exchange	Normal or mild hypoxemia (early stages)	Severe hypoxemia and hypercapnia
Complications	Less likely to develop cor pulmonale	Cor pulmonale may be present in severe or long-standing disease
Distress Level	Patient appears distressed	No apparent distress

Diagnostic Criteria [1-5, 12-14]

Diagnostic Tests for COPD (Based on GOLD 2024) [1]

Chronic Obstructive Pulmonary Disease (COPD) is diagnosed based on a combination of clinical symptoms, spirometry, imaging, and laboratory tests. According to GOLD 2024, a comprehensive approach is required for accurate diagnosis, phenotyping, and disease severity assessment.

Spirometry (Primary Diagnostic Test)

Gold Standard for Diagnosis, Confirms persistent, non-reversible airflow obstruction.Requires measurement of Forced Expiratory Volume in 1 second (FEV_1) and Forced Vital Capacity (FVC).

Diagnostic Criteria for COPD

FEV_1/FVC ratio < 0.7 post-bronchodilator confirms COPD.

Pre-bronchodilator spirometry may indicate obstruction, but post-bronchodilator values are required for confirmation.

Severity Grading (GOLD 2024 Classification)

Mild (GOLD 1): $FEV_1 \geq 80\%$ predicted

Moderate (GOLD 2): FEV_1 50–79% predicted

Severe (GOLD 3): FEV_1 30–49% predicted

Very Severe (GOLD 4): $FEV_1 < 30\%$ predicted

Additional Pulmonary Function Tests [1,14-17]

These tests help in further characterization of COPD, particularly in borderline or complex cases.

Lung Volumes Measurement[1,14-17]

Measures residual volume, total lung capacity, and functional residual capacity.

Assists in evaluating gas trapping and hyperinflation (common in emphysema).

Methods:

Body Plethysmography: Most accurate for lung volume measurement.

Helium Dilution/Nitrogen Washout: Alternative techniques for lung volume estimation.

Carbon Monoxide Diffusing Capacity (DLco)

Assesses gas exchange efficiency in the alveoli.

Decreased DLco: Suggests emphysema due to alveolar destruction.

Differentiates pure airway disease (chronic bronchitis) from emphysema-predominant COPD.

Oximetry & Arterial Blood Gas (ABG)

Pulse Oximetry:Measures oxygen saturation (SpO_2) non-invasively.

If $SpO_2 \leq 92\%$, ABG testing is required.

Arterial Blood Gas (ABG) Analysis:

Identifies hypoxemia (PaO_2 < 60 mmHg) and hypercapnia ($PaCO_2$ > 45 mmHg).Essential in severe COPD and acute exacerbations.

Imaging Studies

While not diagnostic, imaging plays a crucial role in ruling out other lung diseases and assessing COPD severity.

Chest X-ray (Findings suggestive of COPD)

Hyperinflation: Flattened diaphragm, increased retrosternal airspace.

Emphysematous Changes: Hyperlucency, reduced vascular markings.

Used to exclude: Lung cancer, tuberculosis, interstitial lung disease, and heart failure.

High-Resolution Computed Tomography (HRCT)

More sensitive than CXR for detecting emphysema, bronchiectasis, and bullae. Helps guide surgical or bronchoscopic lung volume reduction therapy (LVRS, BLVR).

Exercise Testing & Functional Assessments

These tests evaluate the impact of COPD on physical activity and prognosis.

6-Minute Walk Test (6MWT)

Assesses exercise tolerance and oxygen desaturation during exertion. Drop in SpO_2 > 4% suggests the need for long-term oxygen therapy (LTOT).

Cardiopulmonary Exercise Testing (CPET)

Differentiates pulmonary from cardiac causes of dyspnea.

Determines exercise-induced ventilatory limitations.

Blood Biomarkers & Genetic Testing

Blood Eosinophil Count, Helps guide inhaled corticosteroid (ICS) therapy.

Eosinophils > 300 cells/μL → Greater benefit from ICS.

Eosinophils < 100 cells/μL → ICS may not be effective.

Alpha-1 Antitrypsin Deficiency (AATD) Screening

Indicated in: COPD onset before age 45, Family history of COPD, Lower lobe emphysema pattern, Genetic Testing (SERPINA1 gene mutations) confirms AATD-related COPD.

Screening & Case Finding in High-Risk Groups

COPD should be suspected in individuals with:

Age ≥ 40 years.

Chronic symptoms: dyspnea, cough, sputum production.

Risk factor exposure: Smoking, biomass fuels, occupational dust/fumes.

Screening Tests:

COPD Assessment Test (CAT): Evaluates symptom burden.

Modified Medical Research Council (mMRC) Dyspnea Scale: Assesses breathlessness severity.

Pulmonary Function Testing and Diagnosis of COPD :

Spirometry is a fundamental tool for diagnosing, staging, and monitoring chronic obstructive pulmonary disease (COPD). It plays a crucial role in distinguishing COPD from other respiratory disorders with similar clinical features. However, despite its significance, spirometry remains underutilized in clinical settings, leading to both underdiagnosis and overdiagnosis of COPD. [1,14-17]

- Forced Vital Capacity (FVC): Measures the total volume of air forcibly exhaled after taking a deep breath, providing an estimate of lung capacity.
- Forced Expiratory Volume in 1 Second (FEV1): Assesses the amount of air expelled in the first second of the FVC maneuver, indicating the efficiency of airflow.
- FEV1/FVC Ratio: Compares FEV1 to FVC and is used to evaluate airflow obstruction. In COPD, this ratio is reduced due to airflow limitation. The Global Initiative for Chronic Obstructive Lung Disease (GOLD) defines COPD based on an FEV1/FVC ratio of <0.70 post-bronchodilation, but this fixed threshold may lead to misclassification, particularly in elderly individuals. An alternative approach is the Lower Limit of Normal (LLN), which accounts for individual variations. [1,14]

Role of Spirometry in COPD Management [1,14-16]

Spirometry is a non-invasive, widely accessible diagnostic tool that plays a central role in:

- Confirming COPD
- Determining disease severity
- Guiding treatment strategies
- Monitoring disease progression

Additional tests, such as lung volume measurements and diffusion capacity assessments, provide further insights into pulmonary function. Despite its limitations, spirometry remains the cornerstone of COPD diagnosis and management, with newer classifications such as Pre-COPD and PRISm emphasizing the need for early detection and intervention.

Methods for Symptom Assessment in COPD [18,19,20]

Accurate assessment of COPD symptoms is essential for guiding treatment decisions, monitoring disease progression, and improving patient outcomes. Since symptoms do not always correlate directly with lung function decline, various standardized tools have been developed to assess the severity and impact of COPD on daily life. These tools focus on breathlessness, physical limitations, and overall health-related quality of life.

Modified Medical Research Council (mMRC) Dyspnea Scale [(19,20)]

The mMRC scale is a simple and widely used tool for assessing breathlessness (dyspnea) severity in COPD patients. It measures the degree of disability caused by dyspnea based on a five-point scale.

While easy to use, the mMRC scale is limited to dyspnea assessment and does not evaluate other COPD symptoms such as cough, sputum production, fatigue, or sleep disturbances. (Table B)

Table B. Modified Medical Research Council (mMRC) Grading of Breathlessness [19]

Grade	Description
Grade 0	No breathlessness except with strenuous exercise
Grade 1	Short of breath when hurrying on level ground or walking up a slight hill
Grade 2	Walks slower than people of the same age due to breathlessness or has to stop for breath when walking at own pace
Grade 3	Stops for breath after walking about 100 meters or after a few minutes on level ground
Grade 4	Too breathless to leave the house or breathless when dressing or undressing

COPD Assessment Test (CAT) [19,20]

The CAT questionnaire is a comprehensive, patient-reported tool that assesses the overall impact of COPD on daily life. It includes eight questions, each rated on a scale from 0 to 5, covering:

• Cough frequency

• Phlegm (sputum) production

• Chest tightness

• Breathlessness during exertion

• Limitations in daily activities

• Confidence leaving the house

• Sleep quality

• Energy levels (fatigue)

The total CAT score ranges from 0 (minimal impact) to 40 (severe impact), with higher scores indicating greater disease burden. The CAT is sensitive to changes in COPD severity and treatment response, making it a useful tool for monitoring disease progression over time.

Exacerbation History and Symptom Diaries

In addition to formal questionnaires, exacerbation frequency and daily symptom tracking are valuable for assessing COPD severity. Patients may keep symptom diaries or use digital tracking tools to record:

- Increased breathlessness
- Cough and sputum changes
- Use of rescue medications
- Hospital visits or emergency care needs

Tracking exacerbations helps identify high-risk patients and guide preventive treatment strategies.

Management and Treatment of COPD

COPD management focuses on reducing exacerbations, improving quality of life, and preventing hospitalizations. Pharmacologic therapy includes dual

bronchodilators (LAMA + LABA) for maintenance, ICS for frequent exacerbators, and triple therapy (LAMA + LABA + ICS) for persistent symptoms. Oral treatments like roflumilast (for chronic bronchitis) and azithromycin (for former smokers with recurrent exacerbations) are also used. Acute exacerbations are managed with short-acting bronchodilators, systemic corticosteroids (e.g., prednisone for 5 days), and antibiotics if infection is suspected. Non-pharmacologic strategies include pulmonary rehabilitation, smoking cessation, inhaler training, and regular follow-ups to monitor disease progression. Multidisciplinary care programs help reduce hospitalizations and improve outcomes. [12,18]

Vaccination in COPD Patients [1,22]

Vaccination is essential to prevent infections, exacerbations, and hospitalizations in COPD. GOLD 2024 recommends vaccines against influenza, pneumococcus, COVID-19, RSV, pertussis, and herpes zoster, as COPD patients have weakened immunity and increased infection risk. Despite proven benefits, vaccination uptake remains suboptimal, emphasizing the need for improved adherence.

Bronchiectasis

Definition of Bronchiectasis

Bronchiectasis is a chronic respiratory disease characterized by irreversible bronchial dilatation resulting from the destruction of airway wall components such as cartilage, muscle, and elastin. This disease is marked by persistent cough, daily mucopurulent sputum production, recurrent respiratory infections, and airway obstruction, significantly impacting patients' quality of life and increasing mortality risk.

Despite extensive research, the cause remains idiopathic in approximately 30–40% of patients. In known cases, bronchiectasis is linked to underlying conditions such as post-infectious complications (e.g., tuberculosis), immunodeficiencies, primary ciliary dyskinesia (PCD), cystic fibrosis (CF), and autoimmune diseases like rheumatoid arthritis.

Advances in molecular biology and genomics have opened new avenues to explore the underlying causes and biological complexity of the disease. [23]

Prevalence of Bronchiectasis: Global and Indian Context

Bronchiectasis is increasingly recognized as a significant respiratory disease worldwide. The prevalence of bronchiectasis varies significantly between different regions due to differences in diagnostic capabilities, genetic factors, environmental conditions, and underlying causes such as tuberculosis.

Global Prevalence of Bronchiectasis

A study by Wang et al [24] conducted a large-scale meta-analysis that included data from 15 studies covering 437,851,478 individuals. The study estimated a pooled global prevalence of bronchiectasis at 680 per 100,000 persons (95% CI: 634–727 per 100,000). However, regional variations were noted, with higher prevalence rates in certain Asian countries, suggesting a strong link between infectious etiologies and bronchiectasis development.

In the United States, a study by Weycker et al [25] analyzed retrospective cohort data using ICD9-CM coding and estimated a prevalence of 478 per 100,000 persons. The study noted that bronchiectasis cases have been increasing due to improved radiological detection and growing awareness of the disease.

A study conducted by Henkle et al [26] analyzed data from U.S. Medicare enrollees aged 65 and older between 2006 and 2014. The findings indicated that bronchiectasis is relatively common in this population, with an average prevalence of 701 per 100,000 individuals (0.701%) during the study period. The study highlighted that the prevalence increased with age and was more common in women compared to men.

A study conducted by Choi et al [27] investigated the prevalence of bronchiectasis in South Korea using data from the National Health Insurance Service. The results showed a prevalence of 464 cases per 100,000 adults (0.464%) in 2012, with a slight increase to 480 cases per 100,000 (0.48%) by 2017. The study suggested that increasing HRCT usage and awareness contributed to the rising prevalence.

A study conducted by Monteagudo et al [28] utilized primary care data from Catalonia, Spain, to assess bronchiectasis prevalence. The study reported a prevalence of 362 cases per 100,000 individuals (0.362%) in 2012, with higher rates observed in women (0.391%) compared to men (0.333%). The study emphasized the increasing burden of bronchiectasis and the need for early diagnosis and management.

A study conducted by Seitz et al. 2012 [29] examined trends in bronchiectasis among U.S. Medicare beneficiaries from 2000 to 2007. The findings revealed an increase in prevalence from 322 cases per 100,000 individuals (0.322%) in 2000 to 553 cases per 100,000 (0.553%) in 2007 among women, and from 223 to 388 cases per 100,000 (0.223% to 0.388%) among men during the same period. The study highlighted that increased HRCT use and improved awareness might have contributed to the rising prevalence.

Prevalence of Bronchiectasis in India

Bronchiectasis in India has a unique epidemiological profile, largely due to the high prevalence of tuberculosis (TB), which remains a major cause of post-infectious bronchiectasis. A study by Dhar et al [30] using data from the Indian Bronchiectasis Registry (EMBARC-India) found that among 2,195 enrolled patients, previous tuberculosis was identified as the most frequent underlying cause, accounting for 35.5% of cases. The study also highlighted that Indian patients with bronchiectasis tend to be younger (median age: 56 years) and more commonly male (56.9%) compared to Western populations.

A global registry-based study by Gómez-Olivas et al [31] analyzed data from 27,258 patients across 33 countries and found that post-infectious bronchiectasis accounted for 30.5% of cases globally, with a notably higher prevalence of post-tuberculous bronchiectasis in India at 35.5%. The study emphasized the geographical variability in bronchiectasis etiology, with infections playing a more dominant role in South Asia than in Western populations.

Further, a study by Chalmers et al [32], which examined longitudinal data from the EMBARC-India Registry, found that Indian patients exhibited more severe disease compared to their Western counterparts, with higher rates of exacerbations, hospitalizations, and chronic bacterial infections. The study underscored the urgent need for better awareness, early diagnosis, and improved management of bronchiectasis in India.

Bronchiectasis is no longer an “orphan disease” but rather a growing global health concern. While in Western countries, idiopathic bronchiectasis is a common cause, post-tuberculous and post-infectious bronchiectasis dominate in India and other

Asian regions. The high disease burden in India, coupled with a lack of standardized treatment approaches, highlights the need for more robust healthcare policies, increased screening, and improved management strategies. Addressing bronchiectasis effectively in India will require greater investment in diagnostic tools, increased access to HRCT imaging, and more widespread implementation of evidence-based treatments. [24-32]

Pathophysiology of Bronchiectasis:

The Vicious Vortex Model

The pathophysiology of bronchiectasis revolves around a self-perpetuating cycle of airway infection, inflammation, mucociliary dysfunction, and structural lung damage, collectively described as the vicious vortex model. This interconnected process accelerates disease progression and complicates management.

Airway Infection : [23,33,34]

Airway infection is central to the development and progression of bronchiectasis. Chronic infections with Pseudomonas aeruginosa, Haemophilus influenza, Mycobacterium abscessus, and fungi like Aspergillus are commonly implicated. These pathogens establish biofilms, making them resistant to antibiotics and immune responses.

- The presence of Pseudomonas aeruginosa is particularly associated with severe disease, frequent exacerbations, accelerated lung function decline, and increased mortality.
- Recent molecular studies using next-generation sequencing have shown that the bronchiectasis microbiome is heterogeneous and less diverse, with

dominance of Proteobacteria during exacerbations. Fungal species also contribute significantly to the microbiological landscape.

Airway Inflammation: [23,33,34]

Persistent airway inflammation is a hallmark of bronchiectasis. The immune response is dominated by neutrophilic inflammation, where neutrophils release destructive enzymes.

- Neutrophil elastase, in particular, plays a crucial role in airway tissue degradation, perpetuating inflammation and further impairing mucociliary clearance. High NE activity correlates with disease severity, increased bacterial load, and risk of exacerbations.
- In addition to neutrophils, eosinophilic inflammation has been observed in a subset of patients, potentially identifying a distinct endotype that may respond to corticosteroid therapy.

Mucociliary Dysfunction: [23,33,34]

Effective mucociliary clearance is the first line of defence against inhaled pathogens and environmental pollutants. In bronchiectasis, ciliary dysfunction (either primary, as in PCD, or acquired due to chronic inflammation and infection) and mucus hypersecretion lead to mucus stasis, favouring persistent infections.

- The thickened, highly viscous mucus contains high levels of DNA and proteins released from neutrophils, further impairing clearance.
- Secretory immunoglobulin A (IgA), which plays a protective role in preventing bacterial adhesion, is often deficient in bronchiectasis patients, increasing susceptibility to airway colonization.

Structural Lung Damage:[23,33,34]

Repeated cycles of infection and inflammation result in irreversible bronchial wall damage, causing permanent bronchial dilation. The destruction of elastin, muscle, and cartilage leads to tortuous airways with mucus plugging and airflow obstruction.

- Early damage starts in the small airways and progresses toward larger bronchi, eventually affecting gas exchange and leading to respiratory failure.

Classification of Bronchiectasis

Bronchiectasis can be classified based on its morphological appearance and anatomic distribution, as well as underlying causes. The classification helps in determining the cause, severity, and appropriate management strategy for each patient.

Morphological Classification [34,35,36]

1. **Cylindrical (Tubular) Bronchiectasis:**

- Characterized by uniformly dilated airways without tapering, commonly seen in post-infectious bronchiectasis and less severe forms, appears as parallel "tram-track" lines or a "signet ring" sign on CT.

2. **Varicose Bronchiectasis:**

- Shows an irregular, beaded appearance, resembling a "string of pearls."
- The airway diameter varies, alternating between narrow and dilated segments.
- Associated with more advanced disease and frequent infections.

3. Cystic (Saccular) Bronchiectasis:

- The most severe form, characterized by large, ballooned airways that form cyst-like sacs.
- Often associated with recurrent infections and significant structural damage.
- May lead to progressive respiratory failure if untreated.

Anatomic Classification [34,35,36]

I. Focal Bronchiectasis

- Affects a localized area of the lung, typically resulting from an obstructive process, causes include congenital anomalies (e.g., bronchial atresia), foreign body obstruction, broncholithiasis, and tumours.Diagnosis often requires bronchoscopy to determine the underlying cause.

II. Diffuse Bronchiectasis Involves multiple lobes or both lungs. Causes and patterns of distribution can vary:

- **Upper Lobe Predominance:** Commonly associated with cystic fibrosis, sarcoidosis, and post-radiation fibrosis.
- **Lower Lobe Predominance:** Often related to recurrent childhood infections, aspiration, and fibrotic lung diseases (e.g., idiopathic pulmonary fibrosis).
- **Middle Lobe and Lingula Predominance:** Frequently caused by nontuberculous mycobacterial infection (Mycobacterium avium-intracellulare) or immotile cilia syndrome (Kartagener's syndrome).
- **Central Predominance:** Typically seen in allergic bronchopulmonary aspergillosis (ABPA) and cartilage-deficiency disorders such as Mounier-Kuhn syndrome and Williams-Campbell syndrome.

Etiological Classification [34,35,36]

1. **Congenital Causes**

   - Cystic fibrosis, primary ciliary dyskinesia (Kartagener's syndrome), Williams-Campbell syndrome, Mounier-Kuhn syndrome.

2. **Post-Infectious Causes**

   - Tuberculosis, pertussis, pneumonia, and nontuberculous mycobacteria (NTM) infections.

3. **Immunodeficiency**

   - Hypogammaglobinaemia, HIV infection, chronic granulomatous disease.

4. **Autoimmune and Inflammatory Diseases**

   - Rheumatoid arthritis, Sjögren's syndrome, inflammatory bowel disease.

5. **Obstructive Causes**

   - Foreign body, bronchial tumors, broncholithiasis.

6. **Idiopathic**

   - In about 20–50% of cases, the underlying cause remains unknown despite extensive evaluation.

Etiology and Risk Factors of Bronchiectasis [36]

Bronchiectasis is a multifactorial chronic lung disease that arises as a result of a variety of underlying conditions, including infectious, autoimmune, allergic, genetic, and inflammatory disorders.

The most commonly identified etiology is post-infectious bronchiectasis, accounting for a significant proportion of cases. Conditions such as bacterial pneumonia, tuberculosis, viral infections, and nontuberculous mycobacterial infections are often implicated. Primary and acquired immunodeficiencies also increase susceptibility to recurrent lung infections, which can lead to bronchiectasis development.

Chronic obstructive pulmonary disease (COPD) and severe asthma are strongly associated with bronchiectasis, especially in patients with eosinophilic inflammation and mucus plugging. Additionally, autoimmune diseases like rheumatoid arthritis and connective tissue diseases are linked with more aggressive and rapidly progressive forms of bronchiectasis.

Cystic fibrosis (CF) and primary ciliary dyskinesia (PCD) are notable genetic causes, characterized by impaired mucociliary clearance leading to chronic infections. Gastroesophageal reflux disease (GERD), aspiration, and alpha-1 antitrypsin deficiency (A1AT) are also recognized contributors.

Despite extensive evaluation, approximately 38.1% of cases are classified as idiopathic due to the absence of an identifiable cause. Recognizing the underlying etiology is crucial, as it can alter management strategies and improve outcomes in about 18% of patients. [36]

Clinical Symptoms of Bronchiectasis [34,36]

Respiratory Symptoms

- Chronic Cough – Persistent and productive, often with large amounts of purulent sputum.
- Sputum Production – Copious, thick, and may be foul-smelling due to chronic bacterial colonization.
- Hemoptysis – Blood-streaked sputum or, in severe cases, massive hemoptysis due to airway damage.
- Dyspnea – Shortness of breath, particularly during exertion.
- Wheezing – May occur due to airway narrowing and mucus plugging.
- Chest Pain – Pleuritic in nature, due to recurrent infections or inflammation.[34,36]

Systemic Symptoms

- Fatigue and Malaise – Due to chronic infection and respiratory inefficiency.
- Weight Loss – Seen in advanced cases due to increased energy expenditure and reduced appetite.
- Recurrent Respiratory Infections – Frequent episodes of bronchitis or pneumonia. [34,36]

Exacerbation Symptoms

- Increased Sputum Volume and Purulence – A sign of acute bacterial infection.
- Worsening Dyspnea – Greater difficulty in breathing compared to baseline.
- Fever and Chills – Indicating active infection.

These symptoms are crucial for the clinical diagnosis and management of bronchiectasis, often requiring imaging and sputum analysis for confirmation.[34,35]

Clinical Characteristics of Bronchiectasis [37,38,39]

Physical Signs of Bronchiectasis : (Based on clinical examination findings)

General Inspection:

- Clubbing of fingers – Common in long-standing disease due to chronic hypoxia.
- Cyanosis – May be present in severe cases indicating hypoxemia.
- Cachexia and weight loss – Observed in advanced cases with chronic respiratory distress.

Respiratory Examination:

Inspection:

- Increased chest wall movement on the affected side in localized disease.
- Use of accessory muscles in cases of respiratory distress.
- Barrel chest – Possible in severe or advanced cases with air trapping.

Palpation:

- Decreased chest expansion on the affected side in cases of localized bronchiectasis.
- Increased tactile vocal fremitus over areas of consolidation due to retained secretions.

Percussion:

- Dullness to percussion over areas of significant mucus plugging or lung consolidation.

Auscultation:

- Coarse inspiratory crackles (crepitations) – Typically heard at the lung bases, and may be persistent even after coughing.
- Wheezing – Associated with airway narrowing due to inflammation or secretions.
- Rhonchi – Due to airway obstruction from thick mucus.
- Bronchial breath sounds – In areas of lung consolidation.
- Prolonged expiration – Reflecting airflow limitation.

Cardiovascular Signs (in Severe Cases):

- Signs of right heart failure (cor pulmonale), Raised jugular venous pressure (JVP).Pedal edema. Loud pulmonary component of the second heart sound (P2).These findings aid in the clinical diagnosis and assessment of disease severity in bronchiectasis

Bronchiectasis assessment tools [40,41,42]

Quality of Life-Bronchiectasis (QoL-B)

- A bronchiectasis-specific health-related quality of life (HRQoL) questionnaire, Comprises eight domains:
- Respiratory symptoms, physical function, role function, vitality, emotional function, social function, treatment burden, and health perceptions.

- Scored separately, making it useful for targeted interventions but lacking a single overall score.
- Widely used in clinical trials to assess treatment effects.

St. George's Respiratory Questionnaire (SGRQ)

- A validated tool for assessing chronic respiratory diseases (COPD, bronchiectasis, etc.).
- Measures three domains: symptoms, activity limitations, and impact on daily life.
- Produces a total score (0-100), where higher scores indicate worse health.
- More comprehensive than bronchiectasis-specific tools, but may capture symptoms not entirely relevant to bronchiectasis.

Leicester Cough Questionnaire (LCQ)

- Assesses cough-related quality of life with three domains: physical, psychological, and social.
- Scored 3 to 21, with higher scores indicating better quality of life.
- Commonly used in clinical research to evaluate cough severity in bronchiectasis and other chronic respiratory conditions.

Bronchiectasis Health Questionnaire (BHQ)

- A brief, validated, and repeatable patient-reported health status questionnaire.
- Contains 10 items, generating a single total score for overall health status.
- Strong correlation with SGRQ and lung function (FEV1 % predicted).
- Highly repeatable (intraclass correlation coefficient 0.89), making it reliable for longitudinal assessments.

Bronchiectasis Severity Index (BSI)

A composite scoring system to predict:

- Mortality risk, Exacerbation frequency, Hospitalization risk.
- Includes parameters like age, BMI, lung function (FEV1%), exacerbations, colonization status (Pseudomonas aeruginosa), and radiological severity.
- Categorizes patients into mild, moderate, or severe disease groups.
- Useful for prognosis and clinical decision-making.

FACED Score

- A simplified version of BSI to assess disease severity. Based on five factors:
- FEV1, Age, Chronic colonization (Pseudomonas aeruginosa), Extension (radiological extent of disease), Dyspnea (MRC scale). Scores range 0-7, with higher scores indicating worse disease prognosis.

Bronchiectasis Exacerbation and Symptom Tool (BEST)

- A symptom diary designed to detect exacerbations and track day-to-day symptom fluctuations.
- Includes six symptom categories:
- Cough, sputum volume, sputum color, breathlessness, fatigue, and systemic symptoms.
- Helps in early exacerbation detection, potentially reducing hospital admissions.
- Validated against other quality-of-life tools like SGRQ, LCQ, and CAT.

Each tool serves a different purpose, with QoL-B, BHQ, SGRQ, and LCQ focusing on patient-reported outcomes, while BSI and FACED assess disease severity and prognosis. BEST is useful for monitoring daily symptoms and exacerbations.

Assessment through Bronchiectasis Health Questionnaire (BHQ) [41,42]

The Bronchiectasis Health Questionnaire (BHQ) is a validated, 10-item tool designed to assess health status in bronchiectasis patients, providing a single overall score for easier clinical interpretation.

Development and Validation

- Developed through a three-phase process, including 206 patients.
- Strong correlation with St. George's Respiratory Questionnaire ($r = -0.82$, $p<0.001$).
- Moderately correlated with lung function (FEV1% predicted, $r = -0.27$, $p=0.001$).
- Repeatability confirmed over two weeks (ICC = 0.89).

Structure and Use

- Covers fatigue, breathlessness, cough, sputum production, and sleep disruption.
- Scores range from 0 to 100, with higher scores indicating better health.
- Translated into 11 languages for global use.

Clinical Relevance

- Brief, easy to administer, and reliable for tracking health status.

- Identifies disease severity, exacerbations ($p<0.001$), and hospitalizations ($p=0.001$).
- Practical for both clinical practice and research.

The BHQ is a concise, effective, and patient-centered tool for assessing bronchiectasis impact.

Radiological Diagnosis of Bronchiectasis [23,43]

The gold standard for diagnosing bronchiectasis is high-resolution computed tomography (HRCT) of the chest. It offers superior sensitivity and precision compared to chest radiography, allowing for detailed visualization of airway abnormalities.

Key radiological signs used to confirm bronchiectasis include:

- Airway dilation, where the diameter of the bronchus exceeds that of the adjacent pulmonary artery (airway-to-artery ratio > than 1).
- Lack of tapering, where the bronchial diameter remains constant or widens as it extends towards the lung periphery.
- Bronchial wall thickening, indicative of chronic inflammation or infection.
- Visibility of airways in the peripheral lung regions, which are normally invisible due to their small size .

Management of Bronchiectasis [44,45]

Bronchiectasis management focuses on airway clearance, infection control, anti-inflammatory therapy, pulmonary rehabilitation, and comorbidity management. Airway clearance techniques (e.g., active cycle breathing, postural drainage, oscillatory devices) help mucus clearance, with hyperosmolar agents (hypertonic

saline, mannitol) aiding secretion mobilization, while dornase alfa is not recommended. [31,32]

Infection control involves antibiotics based on sputum culture, with amoxicillin-clavulanate, doxycycline, and macrolides for typical infections and ciprofloxacin or IV antibiotics for Pseudomonas aeruginosa. Long-term macrolides reduce exacerbations but require monitoring, while inhaled antibiotics (gentamicin, colistin, tobramycin) are used for chronic infections.[44,45]

Anti-inflammatory therapy includes ICS for asthma/COPD overlap, oral corticosteroids for ABPA, and statins for potential anti-inflammatory effects (31,32). Pulmonary rehabilitation and regular exercise improve lung function and reduce exacerbations. [44,45]

Comorbidities such as GERD and IgG deficiencies require specific treatments, while surgery or lung transplantation is considered for severe, refractory cases .[31,32,44,45]

Preventative strategies include influenza, pneumococcal vaccines and smoking cessation to slow disease progression. A multidisciplinary, personalized approach is key to improving outcomes and quality of life. [22,44,45]

Bronchiectasis in COPD

COPD and Bronchiectasis: A Distinct Clinical Phenotype

Bronchiectasis and COPD frequently coexist, resulting in a bronchiectasis-COPD overlap (BCO) syndrome that significantly alters the clinical course and prognosis of COPD patients. The coexistence of these conditions defines a distinct

clinical phenotype associated with worse lung function, increased frequency of exacerbations, and higher morbidity and mortality compared to COPD alone. [3]

Prevalence and Clinical Characteristics

The prevalence of bronchiectasis in patients with COPD varies between 4% and 72%, depending on the diagnostic criteria and population characteristics.

A study conducted by Sangtam et al [5] in North-East India reported a prevalence of 17.7% among COPD patients, with female patients showing a slightly higher prevalence (19.45%) compared to males (15.4%). Despite the observed difference, the gender association was not statistically significant. Bronchiectasis was associated with significantly lower forced expiratory volume in one second (FEV1) and increased frequency of exacerbations, although the latter did not reach statistical significance in this particular study. Bronchiectasis in COPD patients is primarily diagnosed through HRCT, which reveals hallmark features such as permanent airway dilation and bronchial wall thickening. Patients often present with chronic cough, daily sputum production, recurrent infections, and wheezing, with dyspnea being the most prevalent symptom, reported in 100% of the study participants. [5]

In another study by Dou et al [6], the prevalence and clinical significance of bronchiectasis in patients with emphysema-predominant COPD were examined. Among 1,739 COPD patients, 8.1% were diagnosed with bronchiectasis using high-resolution computed tomography (HRCT). The prevalence of bronchiectasis was significantly higher in emphysema-predominant COPD patients (16.5%) compared to non-emphysema-predominant patients (10.3%), suggesting a closer link between bronchiectasis and emphysema than previously thought. Patients with coexisting bronchiectasis had more severe airflow limitation (FEV1% predicted) and were more

likely to develop pulmonary hypertension and cor pulmonale compared to those without bronchiectasis. The severity of bronchiectasis correlated with both emphysema extent (r = 0.226) and the degree of airflow limitation (r = -0.371), indicating that worsening lung function increases the risk of bronchiectasis.

The study concluded that emphysema extent and reduced lung function are independent predictors of bronchiectasis in COPD patients, highlighting the importance of early diagnosis and comprehensive management in this group to reduce complications and improve outcomes. [6]

In a cross-sectional study conducted by Silva et al [37], the clinical, functional, and tomographic characteristics of patients with severe chronic obstructive pulmonary disease (COPD) with and without bronchiectasis were analyzed. HRCT was used to identify bronchiectasis in 24.5% of the 98 patients studied. The presence of bronchiectasis was significantly associated with bilateral distribution and predominantly lower lobe involvement. Patients with bronchiectasis had a greater drop in oxygen saturation during the six-minute walk test (ΔSpO2) and worse forced vital capacity (FVC) compared to those without bronchiectasis.

Additionally, sputum cultures revealed that the presence of potentially pathogenic microorganisms (PPM), such as *Pseudomonas aeruginosa*, increased the likelihood of having bronchiectasis by nearly fivefold. The study concludes that bronchiectasis in COPD is associated with worse lung function, chronic inflammation, and increased airway colonization, underscoring the need for early detection and personalized treatment strategies for these patients. [37]

A study conducted by Patel et al [44] investigated the prevalence of bronchiectasis in patients with moderate-to-severe COPD using high-resolution

computed tomography (HRCT). The study included 54 stable COPD patients who underwent HRCT imaging to assess the presence of bronchiectasis. It was found that 50% of the patients exhibited bronchiectatic changes. The study further highlighted that patients with bronchiectasis had significantly higher levels of airway inflammation, more frequent bacterial colonization, and prolonged exacerbations compared to those without bronchiectasis. The authors concluded that the presence of bronchiectasis in COPD patients represents a distinct clinical phenotype associated with more severe disease progression.

A study conducted by Martínez-García et al [45] to evaluate the clinical implications of bronchiectasis in COPD patients. This prospective cohort study analyzed 201 patients with moderate-to-severe COPD and found that 57.6% had bronchiectasis as detected by HRCT. The study demonstrated that the presence of bronchiectasis was strongly associated with increased exacerbation frequency, a higher prevalence of potentially pathogenic microorganisms (PPMs), and greater systemic inflammation. Additionally, patients with COPD and bronchiectasis had a significantly higher risk of mortality, indicating that bronchiectasis is an independent prognostic factor in COPD.

A study conducted by Mao et al [46] analyzed the prevalence and clinical impact of bronchiectasis in a large cohort of 406 COPD patients. The results showed that 34.7% of the patients had coexisting bronchiectasis, and this subgroup exhibited worse lung function, higher rates of chronic bacterial colonization, and more frequent hospitalizations. The study further stratified patients based on the severity of bronchiectasis and found that those with more extensive bronchiectatic changes had worse clinical outcomes. The authors concluded that the coexistence of bronchiectasis

in COPD patients correlates with a distinct disease progression pattern characterized by a higher burden of exacerbations and greater healthcare utilization.

A study conducted by O'Brien et al [47] investigated the prevalence of bronchiectasis in 110 primary care patients presenting with acute exacerbations of COPD. The study employed HRCT imaging and found that 29% of these patients had bronchiectatic changes, despite many of them not meeting the classical diagnostic criteria for COPD. This finding suggests that undiagnosed bronchiectasis may be contributing to the disease burden in a subset of COPD patients. The study also reported that patients with bronchiectasis had more significant airflow obstruction and a greater likelihood of requiring hospitalization during exacerbations.

A study conducted by Polverino et al [48] analyzed data from the European Bronchiectasis Registry (EMBARC), which included 16,730 patients diagnosed with bronchiectasis. The study found that 25.9% of these patients had a co-diagnosis of COPD, suggesting a substantial overlap between the two conditions. The study highlighted that patients with both bronchiectasis and COPD had worse clinical outcomes, including higher rates of exacerbations, more frequent hospitalizations, and a greater burden of chronic bacterial colonization. The authors suggested that COPD patients with bronchiectasis should be considered as a distinct phenotype requiring targeted management strategies.

Predisposing Factors for the Development of Bronchiectasis in COPD Patients:

Bronchiectasis is a chronic, irreversible dilation of the bronchi that frequently develops in COPD patients due to persistent inflammation, airway obstruction, mucus stasis, and recurrent infections. Understanding the predisposing factors for bronchiectasis in COPD patients is crucial because COPD-bronchiectasis overlap

syndrome (CBOS) is associated with more severe airflow limitation, increased exacerbations, higher rates of hospitalization, greater bacterial colonization, and poorer prognosis.

i) Severe airflow limitation

One of the most significant risk factors for bronchiectasis development in COPD is severe airflow limitation. Reduced FEV_1% predicted contributes to weakened mucociliary clearance, loss of airway structural integrity, and higher bacterial colonization, all of which promote chronic infection and inflammation. A study by Dou et al [6] on 1,739 COPD patients found that 140 patients (8.1%) had radiologically confirmed bronchiectasis, with a mean FEV_1% predicted of 36.5% compared to 48.3% in COPD-only patients ($p<0.001$). A strong negative correlation was observed between FEV_1% and bronchiectasis severity ($r = -0.371$, $p < 0.001$), confirming that worsening airflow limitation increases bronchiectasis risk. Similarly, Sangtam et al [5] found that the mean FEV_1% predicted in bronchiectasis patients was 35.6%, compared to 47.2% in non-bronchiectasis COPD patients ($p<0.05$). In a meta-analysis of 881 patients, Ni et al [38] reported that the mean FEV_1% was 39.1% in COPD-bronchiectasis patients compared to 50.7% in COPD-only patients ($p<0.001$). The prevalence of bronchiectasis was significantly higher in GOLD 3-4 COPD patients (23.7%) than in GOLD 1-2 patients (6.5%). Mechanistically, severe obstruction leads to hyperinflation and dynamic airway collapse, mucus hypersecretion, and impaired clearance, which increase bacterial overgrowth. Chronic inflammation further contributes to bronchial wall remodeling and dilation. [5,6,38]

ii) Chronic airway infections and colonization by Potentially pathogenic microorganisms

Chronic bacterial colonization is another major risk factor for bronchiectasis in COPD patients. Potentially pathogenic microorganisms, including Pseudomonas aeruginosa, Haemophilus influenzae, Moraxella catarrhalis, and Streptococcus pneumoniae, are frequently found in the airways of these patients, leading to persistent airway inflammation and progressive lung damage. A study by Gatheral et al [7] found that 69% of COPD-bronchiectasis patients were colonized with Pseudomonas aeruginosa, compared to only 21% in COPD-only patients ($p<0.001$). Similarly, Ni et al [38] reported that Pseudomonas aeruginosa was present in 32.4% of COPD-bronchiectasis patients compared to 8.1% in COPD-only patients ($p<0.01$). Sangtam et al [5] found that Haemophilus influenzae was the most common isolate in bronchiectasis patients (54.5%), followed by Pseudomonas aeruginosa (18.2%).

High bacterial loads ($>10^6$ CFU/mL) were associated with significantly elevated CRP levels (8.2 mg/L vs. 4.5 mg/L, $p=0.01$). Additionally, Pseudomonas aeruginosa colonization was linked to an increased exacerbation rate of 3.5 per year, compared to 1.8 in non-colonized patients ($p<0.001$). Chronic infections persist due to bacterial biofilm formation in bronchiectatic airways, which makes infections difficult to clear. Neutrophilic inflammation further worsens airway destruction, and protease and cytokine release contribute to progressive airway dilation.[5,7,38]

iii) Frequent exacerbations

Frequent exacerbations (≥2 per year) are another important predictor of bronchiectasis development in COPD patients.Exacerbations trigger acute airway inflammation, leading to long-term structural damage, increased bacterial

colonization, and accelerated lung function decline. In a study by Martinez-Garcia et al [2] , COPD-bronchiectasis patients had an exacerbation rate of 3.2 per year compared to 1.7 per year in COPD-only patients ($p<0.001$). Similarly, Ni et al [38] found that patients with ≥2 exacerbations per year had a 62% increased risk of bronchiectasis (OR 1.62, $p=0.008$). Mechanistically, exacerbations trigger airway inflammation and structural damage, and the post-exacerbation period is associated with increased bacterial colonization and mucus retention. Each exacerbation contributes to an accelerated decline in pulmonary function, further predisposing airways to bronchial dilation. [2,38]

iv) Emphysema, Chronic bronchitis, and Bronchiectasis

The relationship between emphysema, chronic bronchitis, and bronchiectasis development in COPD patients is complex. Emphysema-predominant COPD is associated with a loss of alveolar attachments, increasing airway collapse and mucus trapping. In a study by Dou et al [6] bronchiectasis was found in 16.5% of emphysema-predominant COPD patients compared to 10.3% in non-emphysema COPD patients ($p=0.01$). Emphysema severity, as measured by the emphysema index (EI), was an independent predictor of bronchiectasis (OR 1.99, $p=0.008$). Chronic bronchitis, on the other hand, is characterized by persistent mucus hypersecretion, bacterial colonization, and airway inflammation, all of which contribute to bronchiectasis development. In a study by Martinez-Garcia et al [2] 67% of COPD-bronchiectasis patients exhibited chronic bronchitis symptoms, compared to 42% of COPD-only patients ($p<0.001$).[2,6]

v) Systemic inflammation

Systemic inflammation plays a crucial role in bronchiectasis development in COPD patients. Various blood biomarkers provide insights into the inflammatory burden and its impact on disease progression. COPD-bronchiectasis patients often exhibit lower hemoglobin (Hb) levels, which may indicate anemia of chronic disease due to persistent inflammation and hypoxia. Mean Hb levels in COPD-bronchiectasis patients were found to be 12.4 g/dL compared to 13.8 g/dL in COPD-only patients (p=0.001). Total leukocyte count (WBC) is also elevated in COPD-bronchiectasis due to chronic infection and inflammation, with a mean WBC count of 9.3×10^9/L vs. 7.6×10^9/L in COPD-only patients (p=0.002). [7] Elevated high-sensitivity C-reactive protein (HSCRP) levels were observed in COPD-bronchiectasis patients (6.2 mg/L vs. 3.9 mg/L in COPD-only, p<0.01), correlating with frequent hospitalizations (OR 2.1, p=0.005), worse lung function (FEV_1% predicted <40%), and higher mortality risk [7]. Serum albumin levels were significantly lower in COPD-bronchiectasis patients (3.7 g/dL vs. 4.2 g/dL in COPD-only, p<0.001), reflecting a higher inflammatory burden and poor nutritional status.[7] Other markers such as fibrinogen (3.7 g/L vs. 3.4 g/L, p=0.03) and erythrocyte sedimentation rate (ESR) (23.1 mm/h vs. 18.4 mm/h, p=0.01) were also elevated in COPD-bronchiectasis patients, indicating persistent systemic inflammation. [7]

vi) Smoking

Smoking is a well-established risk factor for the development and progression of both COPD and bronchiectasis. Chronic exposure to tobacco smoke leads to airway inflammation, destruction of alveolar structures, and impaired mucociliary clearance, all of which contribute to increased mucus retention, bacterial colonization, and

chronic infections. Smoking is also associated with oxidative stress, which further damages lung tissue and impairs immune responses, making the lungs more susceptible to recurrent infections. [5, 6]

Several studies have demonstrated a strong correlation between smoking history and the presence of bronchiectasis in COPD patients. A study by Sangtam et al [5] observed that COPD patients with bronchiectasis had a significantly higher cumulative smoking history compared to those without bronchiectasis, with a mean pack-year history of 47.2 vs. 35.1 ($p=0.03$). Similarly, Dou et al [6] observed that the prevalence of bronchiectasis was higher in heavy smokers (>40 pack-years) compared to light smokers (<20 pack-years) ($p<0.05$).

vii) Nutritional Status and BMI

Malnutrition and low body mass index (BMI) are frequently observed in COPD patients with bronchiectasis and are associated with worse disease outcomes. Poor nutritional status contributes to muscle wasting, immune dysfunction, and reduced respiratory muscle strength, all of which exacerbate the severity of lung disease. Several studies have demonstrated that COPD patients with bronchiectasis have lower BMI and poorer nutritional status compared to those without bronchiectasis.

A study by Gatheral et al [7] reported that COPD-bronchiectasis patients had a significantly lower mean BMI of 21.5 vs. 24.3 in COPD-only patients ($p<0.01$). Similarly, Ni et al [38] found that BMI <18.5 was associated with a 1.9-fold increased risk of bronchiectasis ($p=0.002$).Lower BMI was also associated with increased exacerbation frequency, reduced exercise capacity, and higher mortality risk.

Serum albumin levels, an important marker of nutritional status, were also significantly lower in COPD-bronchiectasis patients. A study found that mean serum albumin levels were 3.7 g/dL in COPD-bronchiectasis vs. 4.2 g/dL in COPD-only ($p<0.001$) [7]. Low albumin levels were correlated with higher exacerbation frequency, severe weight loss, and increased hospitalization rates.[7,38]

viii) Dyspnea and Functional Capacity

Dyspnea, or breathlessness, is a hallmark symptom of COPD and is further exacerbated by the presence of bronchiectasis. Functional capacity, as measured by the Modified Medical Research Council (MMRC) dyspnea scale and the 6-minute walk distance (6MWD) test, is often significantly worse in COPD-bronchiectasis patients compared to those with COPD alone.

A study by Gatheral et al [7] found that MMRC scores were significantly higher in COPD-bronchiectasis patients (2.7 vs. 1.9 in COPD-only, $p<0.001$), indicating greater breathlessness. Similarly, Martinez-Garcia et al. (2017) [2] reported that patients with MMRC ≥ 3 had a 2.3-fold increased likelihood of having bronchiectasis ($p=0.01$).

The 6-minute walk distance (6MWD) test is an important measure of functional capacity and exercise tolerance. COPD-bronchiectasis patients often exhibit reduced 6MWD due to increased dyspnea, airflow limitation, and muscle wasting. Ni et al [38] observed that mean 6MWD was 342 meters in COPD-bronchiectasis patients vs. 396 meters in COPD-only patients ($p<0.05$). Furthermore, patients with 6MWD <350 meters had a significantly higher exacerbation frequency and hospital admission rates.

Impact of Bronchiectasis in COPD Patients :

Bronchiectasis in patients with chronic obstructive pulmonary disease (COPD) defines a distinct clinical phenotype that is associated with more severe disease, higher health care utilization, and increased morbidity.

i) Increased Exacerbations and Hospitalization

COPD patients with bronchiectasis experience more frequent exacerbations and longer hospital stays compared to those without bronchiectasis. According to Gatheral et al [7] these patients had a significantly higher annual respiratory admission rate, reflecting the increased clinical burden.

ii) Chronic Infections and Microbial Colonization

Chronic colonization by potentially pathogenic microorganisms (PPMs), particularly Pseudomonas aeruginosa, is common in COPD patients with bronchiectasis. This colonization contributes to persistent airway inflammation, increased exacerbations, and poor clinical outcomes. [38]

iii) Worse Lung Function and Quality of Life

Patients with comorbid bronchiectasis exhibit lower FEV1 values and more rapid lung function decline, indicating more severe airflow obstruction. Health-related quality of life is significantly impaired due to increased dyspnea, chronic sputum production, & fatigue. [2]

iv) Body Mass Index and Nutritional Status

Low BMI and poor nutritional status are more prevalent in COPD patients with bronchiectasis, and these factors are associated with worse outcomes, including

increased hospitalizations and higher mortality. Malnutrition exacerbates disease progression by weakening respiratory muscles and reducing immune defense, making these patients more susceptible to recurrent infections. [38]

v) Poor Prognosis and Mortality

Bronchiectasis is considered an independent predictor of poor prognosis in COPD patients. Its presence correlates with higher mortality rates and increased complications, necessitating comprehensive and personalized management.

The coexistence of bronchiectasis in COPD patients amplifies the clinical burden by increasing exacerbations, infections, and mortality. Regular monitoring of lung function, nutritional status, and early treatment of infections is essential to improve long-term outcomes. Tailored management strategies can help reduce complications and improve the quality of life in these patients.[2,7, 37)]

Management Strategies in Bronchiectasis-COPD overlap (BCO)

Management of bronchiectasis-COPD overlap requires a multidisciplinary and personalized approach, focusing on airway clearance, infection control, and prevention of exacerbations.

i. Airway Clearance Therapy

Effective clearance of airway secretions is essential for all patients with bronchiectasis. Chest physiotherapy and the use of devices like oscillatory positive expiratory pressure (PEP) help reduce mucus retention and improve lung function. Pulmonary rehabilitation and exercise also contribute to improving symptoms and overall health-related quality of life. [3,34)]

ii. Antibiotic Therapy

- Long-term macrolides (e.g., azithromycin) have shown to reduce exacerbations in BCO patients by targeting chronic airway inflammation and bacterial colonization.
- Inhaled antibiotics, such as tobramycin and colistin, are recommended for patients with chronic Pseudomonas aeruginosa infection to reduce bacterial load and prevent recurrent infections
- Acute exacerbations require culture-guided antibiotics to target the specific pathogens identified through sputum analysis. [3,44,45]

iii. Anti-inflammatory Therapy

Inhaled corticosteroids (ICS) are used selectively in BCO patients with eosinophilic inflammation or coexisting asthma.

However, routine use of ICS in BCO without these indications may increase the risk of infections, particularly with nontuberculous mycobacteria (NTM). New therapies targeting neutrophilic inflammation are under investigation to reduce airway damage and improve outcomes. [3,31,32]

iv. Microbiome-based Strategies

With advances in microbiome research, there is growing interest in targeted therapies that modulate the airway microbiome. These include probiotic approaches and bacteriophage therapy, although more clinical trials are needed to validate their effectiveness. [3]

v. Addressing Comorbidities

Managing underlying conditions such as gastroesophageal reflux disease (GERD), immunodeficiency, and connective tissue disorders is crucial in improving disease control. Nutritional support and vaccination against respiratory pathogens (influenza and pneumococcus) are recommended for all patients.[44,45]

British Thoracic Society (BTS) Guideline for Management of Bronchiectasis in Adults (2019) [46,47]

Management of bronchiectasis centers on airway clearance and infection control. All patients should be taught effective airway clearance techniques by a respiratory physiotherapist, with options such as active cycle of breathing techniques, oscillating positive expiratory pressure, and gravity-assisted drainage. Mucoactive agents like carbocysteine or hypertonic saline may be trialed in patients who have difficulty expectorating sputum, while recombinant human DNase is not recommended. For patients experiencing three or more exacerbations annually, long-term antibiotic therapy particularly macrolides like azithromycin or inhaled antibiotics for P. aeruginosa colonization is advised, with careful monitoring for side effects and antimicrobial resistance.

Other important aspects of care include ensuring patients receive annual influenza and pneumococcal vaccinations. Bronchodilators may be used if the patient has co-existing asthma or COPD, and corticosteroids should be reserved for those with overlapping conditions such as ABPA or IBD. Pulmonary rehabilitation is recommended for functionally limited patients, and nutritional status should be monitored regularly.

Patients with localized disease unresponsive to optimal medical therapy may be considered for surgical resection, while lung transplantation is an option for younger patients with advanced disease, particularly those with severe airflow limitation or rapid clinical deterioration. Follow-up in secondary care is recommended for patients with severe disease, frequent exacerbations, chronic infection with pathogens like P. aeruginosa or MRSA, or those requiring long-term antibiotic therapy. Routine monitoring should include annual clinical review, sputum analysis, lung function tests, and assessment of disease progression. [46,47]

Prognosis and Future Directions

The presence of bronchiectasis in COPD patients is associated with higher mortality, reduced lung function, and poorer quality of life. Longitudinal studies are necessary to further explore the natural history of bronchiectasis in COPD and identify biomarkers that can guide early diagnosis and personalized treatment.

Future therapies may include anti-inflammatory agents, microbiome-targeting interventions, and biologics to control inflammation and infection. [(2,6,7,17)]

Martinez-Garcia et al [2] emphasized the need for clinical trials focused on reducing bacterial loads and improving lung function in patients with BCO. Such trials will help determine the most effective strategies for reducing exacerbations and improving long-term outcomes.

MATERIALS AND METHODS

- **Study Design**

This was a cross-sectional observational study conducted to determine the prevalence and predisposing factors of bronchiectasis in COPD patients.

- **Study Period and Duration**

The study was conducted over a 12-month period, from March 2023 to March 2024.

- **Study Place**

The study was carried out at KLE'S Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi, a tertiary care teaching hospital that caters to a large number of respiratory disease patients.

- **Sample Size Calculation**

The sample size was calculated using the finite sample size formula:

$$n = \frac{N \times z1-\alpha^2 \times p\,(100-p)}{[d^2 \times (N-1) + z1-\alpha^2 \times p \times (100-p)]}$$

On an average 450 -650 COPD patients attending hospital in a year

N = Estimated Population size =450,

p = prevalence of bronchiectasis,

d =Error (Relative), $z1-\alpha$ = critical value for given confidence

N = 450, p = 50% (from previous studies)[7],

d= 15 % of p = 0.15 x 50 = 7.5

For 95% confidence,

$\underline{z1-\alpha = 1.96 \approx 2\ n = 450* 2\ 2 * 50*(100-50)}$

$[(7.5)\ 2* (450-1)+ 2\ 2* 50* (100-50)]$

$n = 450* 2\ 2 *50 *(100-50)\ (7.5)\ 2* (450-1)+ 2\ 2* 50* (100-50)$

$n = 450* 4*50*50\ (7.5)\ 2* (449)+ 4* 50* (50)$

$n = 450* 4*50*50\ 56.25* (449)+ 10000$

$n = 4500000\ 25256.25+ 10000 = 4500000\ 35256.25$

$n = 127.6 \approx 128$

Considering 5% Non response, a sample size of 128 + 6.4 ≈ 135, hence a total of 146 subjects were included in the study.

- **Study Protocol**

- Patient Selection and Recruitment
  - COPD patients attending the respiratory medicine department at KLE Hospital were screened for inclusion.
  - All patients meeting the inclusion and exclusion criteria were recruited.
  - A systematic sampling technique was adopted.
  - Written informed consent was obtained before study participation.
- **Clinical Assessment**

- Detailed clinical history was recorded, including:
  - Number of previous exacerbations in the last year.
  - Hospitalization history in the last 2 years.
  - History of tuberculosis, chronic infections, and antibiotic use.
  - Smoking history (pack-years calculation).
- Physical examination included:
  - Measurement of vitals (BP, HR, respiratory rate, temperature).
  - Anthropometric measurements (height, weight, BMI calculation).

- Respiratory system examination for wheezing, crackles, or rhonchi.

➢ **Study Outcomes**

- Primary Outcome

  Prevalence of bronchiectasis in COPD patients based on HRCT findings.

- Secondary Outcomes

  Risk factors associated with the development of bronchiectasis in COPD, including:

  - Age, gender, BMI, smoking history, disease duration.
  - Pulmonary function impairment (FEV1, FVC, FEV1/FVC ratio).
  - Number of exacerbations and hospitalizations.
  - Microbiological findings (sputum culture for infections, especially Pseudomonas aeruginosa).
  - Nutritional and inflammatory markers (serum albumin levels, leukocyte counts, etc.).
  - Bronchiectasis Health Questionnaire

➢ **Inclusion Criteria**

- Clinically confirmed COPD cases (based on GOLD 2023 criteria).
- Patients aged >40 years.

➢ **Exclusion Criteria**

- Patients with secondary immunodeficiency states (HIV, on immunosuppressants).
- Active tuberculosis or previous pulmonary TB with severe lung damage (“Destroyed lung”).
- Other chronic lung diseases (e.g., ILD, cystic fibrosis, pulmonary fibrosis).
- History of childhood measles or whooping cough.

- Known cases of bronchiectasis diagnosed during childhood.

➢ **Methods of Data Collection**

- Baseline Data Collection
    - Demographic details were recorded, including age, gender, occupation, smoking status, and BMI.
- Symptom severity was assessed using:
    - COPD Assessment Test (CAT) score.
    - Modified Medical Research Council (mMRC) Dyspnea Scale.
    - 6-Minute Walk Distance (6MWD) test.
    - Pulmonary function testing (PFT) was performed to assess:
    - FEV1, FVC, FEV1/FVC ratio, Peak Expiratory Flow Rate (PEFR).
- Exacerbation history (frequency, severity, treatment details) was documented.
- Hospitalization history in the last 2 years was noted.
- Comorbidities (diabetes, hypertension, cardiovascular disease) were recorded.
- Microbiological assessment included:
    - Sputum culture for bacterial colonization (especially Pseudomonas aeruginosa).
    - Sputum AFB test to rule out tuberculosis.
    - Radiological evaluation (HRCT thorax) was performed to classify bronchiectasis.

➢ **Investigations**

- Lung Function Tests (Spirometry/PFTs)
    - FEV1, FVC, FEV1/FVC ratio, Peak Expiratory Flow Rate (PEFR) were measured.

- HRCT Thorax (High-Resolution Computed Tomography) was used to diagnose bronchiectasis. Diagnosis was confirmed using the following criteria in HRCT Thorax :
    - Broncho-arterial ratio >1 (bronchial diameter greater than adjacent pulmonary artery).
    - Lack of tapering of bronchial lumen.
    - Visibility of bronchus within 10 mm of the pleural surface.
- Chest X-ray was performed to detect additional lung pathology.
- Blood Tests and Microbiological Studies
- Serum albumin was measured as a marker of nutritional status.
- Total leukocyte count and differential count were assessed for inflammation.
- Sputum culture was performed to detect bacterial colonization.

➢ **Ethical Clearance**

The study was approved by the JNMC Institutional Ethics Committee.Ethical guidelines for human research were strictly followed.

➢ **Informed Consent Process**

- Written informed consent was obtained before enrollment.
- Patients were explained:
    - Study objectives, procedures, and potential risks/benefits.
    - Confidentiality of their data.
    - Right to withdraw from the study at any time without affecting treatment.

➢ **Statistical Analysis**

- Data entry and processing were performed using Microsoft Excel & SPSS v22 software.

- Descriptive statistics were used for demographics, lung function, and clinical variables.
- Chi-square test was used to assess associations between categorical variables.
- Student's t-test/Mann-Whitney U test was applied for continuous variables.
- A p-value < 0.05 was considered statistically significant.

RESULTS

Table 1. Baseline Characteristics: COPD vs COPD with Bronchiectasis

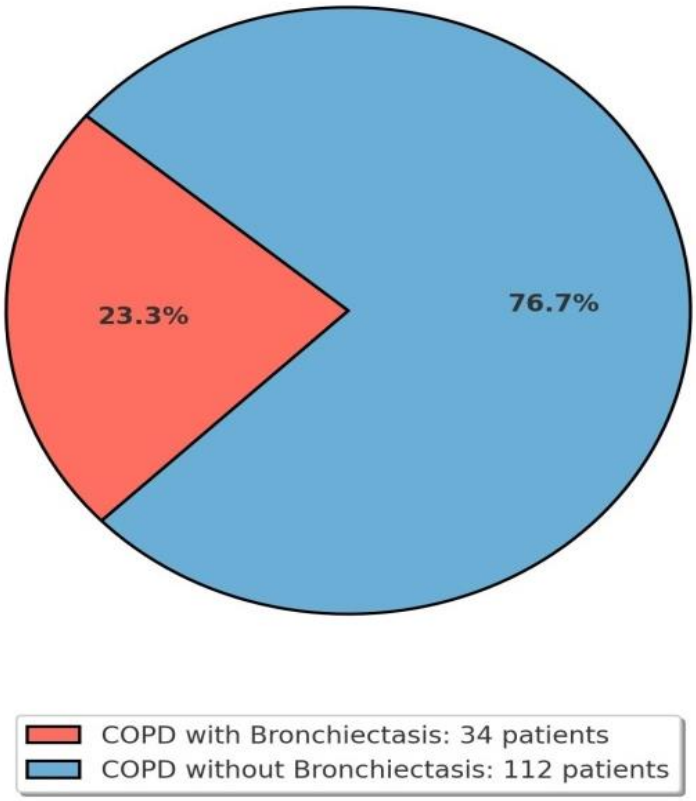
CHARACTERISTIC	COPD (N=112)	COPD WITH BRONCHIECTASIS (N=34)	P-VALUE
AGE (YEARS)	67.3 ± 9.2	67.1 ± 10.4	0.927
GENDER (MALE/FEMALE)	Male: 86 (76.8%) Female: 26 (23.2%)	Male: 31 (91.2%) Female: 3 (8.8%)	0.110
BMI (KG/M²)	24.1 ± 3.7	21.5 ± 4.3	0.003
FEV1 (% PREDICTED)	59.4 ± 12.4	54.5 ± 10.6	0.026
FVC (% PREDICTED)	86.3 ± 18.6	77.7 ± 15.0	0.008
FEV1/FVC RATIO	0.67 ± 0.05	0.69 ± 0.07	0.191
FEF25-75 (% PREDICTED)	45.5 ± 15.3	39.8 ± 14.7	0.053
PEFR (% PREDICTED)	49.9 ± 19.1	42.4 ± 14.9	0.018
MMRC GRADE	1.7 ± 0.8	2.4 ± 0.7	<0.001
CAT SCORE	14.9 ± 4.1	18.9 ± 4.4	<0.001
GOLD ABE CLASSIFICATION	A: 54 (48.2%) B: 54 (48.2%) E: 4 (3.6%)	A: 1 (2.9%) B: 12 (35.3%) E: 21 (61.8%)	<0.001
GOLD GRADE (1–4)	GOLD 1: 3 (2.7%) GOLD 2: 83 (74.1%) GOLD 3: 22 (19.6%) GOLD 4: 4 (3.6%)	GOLD 1: 0 (0.0%) GOLD 2: 19 (55.9%) GOLD 3: 15 (44.1%) GOLD 4: 0 (0.0%)	0.023
SYMPTOM DURATION (YEARS)	7.1 ± 3.8	9.4 ± 4.5	0.009
HOSPITAL ADMISSIONS (LAST 2 YRS)	1.3 ± 0.9	3.1 ± 0.8	<0.001
SMOKING HISTORY	Smoker: 71 (63.4%) Non-smoker: 41(36.6%)	Smoker: 25 (73.5%) Non-smoker: 9 (26.5%)	0.376
HTN	44 (39.3%)	21 (61.8%)	0.035
T2DM	29 (25.9%)	17 (50.0%)	0.015
CKD	0 (0.0%)	2 (5.9%)	0.081
IHD	15 (13.4%)	12 (35.3%)	0.009
CLD	0 (0.0%)	1 (2.9%)	0.526

Table 1. Compares the baseline characteristics of the patients, involved in the study. It compares COPD-only (n=112) patients with those having COPD and bronchiectasis (n=34), highlighting differences in lung function, symptom burden, disease severity, and comorbidities.

COPD with bronchiectasis patients had lower BMI (21.5 ± 4.3 vs. 24.1 ± 3.7, p=0.003) and worse lung function, with significantly reduced FEV1 (54.5 ± 10.6 vs. 59.4 ± 12.4, p=0.026), FVC (77.7 ± 15.0 vs. 86.3 ± 18.6, p=0.008), and PEFR (42.4 ± 14.9 vs. 49.9 ± 19.1, p=0.018). Symptom burden was higher in this group, reflected by higher MMRC grade (2.4 ± 0.7 vs. 1.7 ± 0.8, p<0.001) and CAT score (18.9 ± 4.4 vs. 14.9 ± 4.1, p<0.001). Most were in GOLD group E (61.8%), indicating frequent exacerbations, while COPD-only patients were mainly in groups A/B (48.2% each, p<0.001).

Disease progression was worse in the bronchiectasis group, with longer symptom duration (9.4 ± 4.5 vs. 7.1 ± 3.8 years, p=0.009) and more hospital admissions (3.1 ± 0.8 vs. 1.3 ± 0.9, p<0.001). Comorbidities were also more common, including HTN (61.8% vs. 39.3%, p=0.035), T2DM (50.0% vs. 25.9%, p=0.015), and IHD (35.3% vs. 13.4%, p=0.009).

Fig 1. Prevalence of Bronchiectasis in COPD patients



The pie (fig. 1) chart illustrates the prevalence of bronchiectasis among a total of 146 COPD patients, 34 patients (23.3%) had coexisting bronchiectasis, 112 patients (76.7%) did not have bronchiectasis,

Table 2. Comparison of Age, Gender, and BMI: COPD vs COPD with Bronchiectasis

Characteristic	COPD (n=112)	COPD with Bronchiectasis (n=34)	P-value
Age (years)	67.3 ± 9.2	67.1 ± 10.4	0.927
Male, n (%)	86 (76.8%)	31 (91.2%)	0.110
Female, n (%)	26 (23.2%)	3 (8.8%)	0.110
BMI (kg/m²)	24.1 ± 3.7	21.5 ± 4.3	0.003

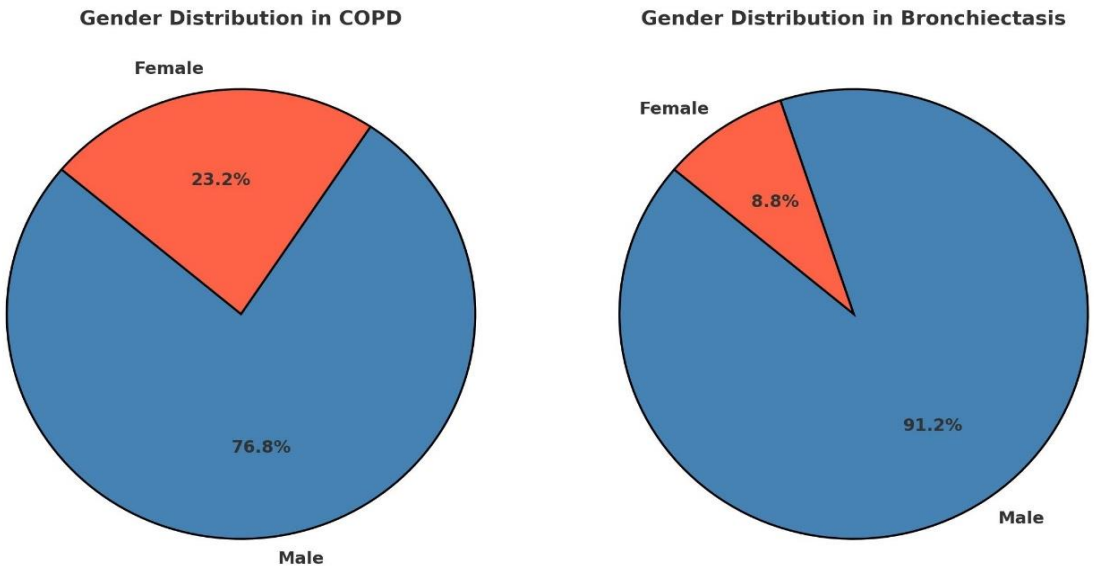
Demographic Characteristics

A total of 112 COPD patients and 34 Bronchiectasis patients were included in this study. The mean age of COPD patients was 67.3 ± 9.2 years, while the mean age of Bronchiectasis patients was 67.1 ± 10.4 years. The difference between the two groups was not statistically significant ($p = 0.927$), indicating that age distribution did not play a distinguishing role between these conditions. (Table 2)

Gender Distribution.

Among COPD patients, 76.8% were male, while 23.2% were female. In contrast, the Bronchiectasis group had a higher proportion of males (91.2%), with only 8.8% of patients being female. Despite the noticeable male predominance in the Bronchiectasis group, this difference was not statistically significant ($p = 0.110$). (Table 2 and Fig 2)

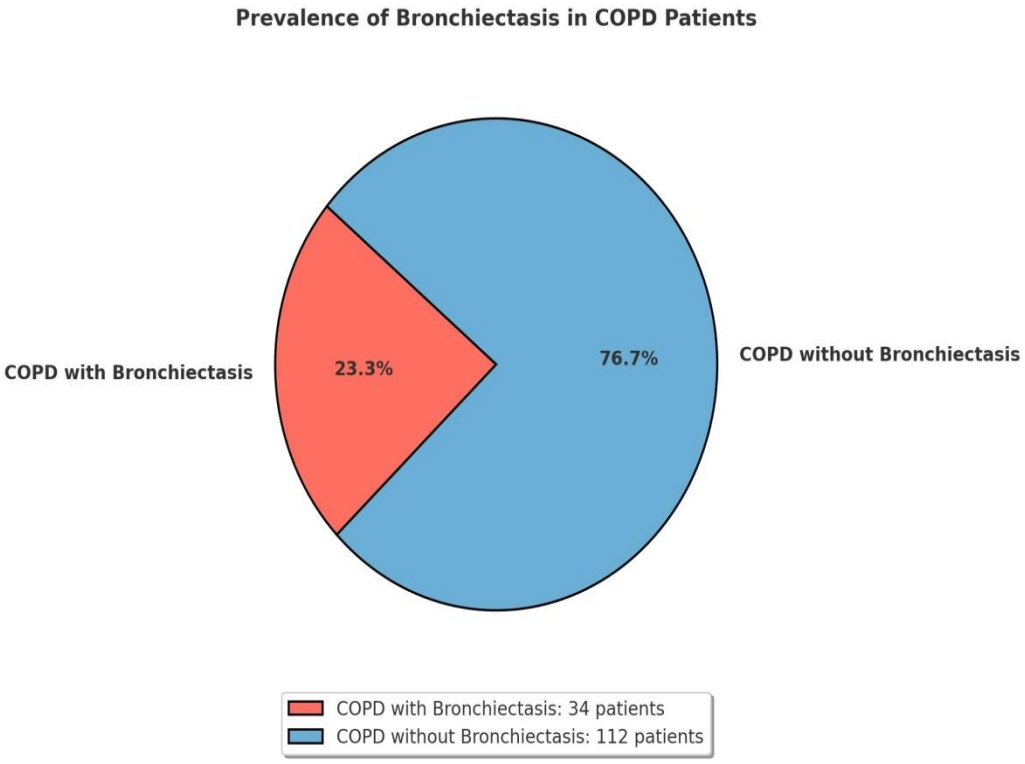
Fig 2. Gender distribution in COPD and COPD with Bronchiectasis group



Body Mass Index (BMI) Comparison

The mean BMI among COPD patients was 24.1 ± 3.7 kg/m², while in the Bronchiectasis group, it was significantly lower at 21.5 ± 4.3 kg/m². This difference was found to be statistically significant ($p = 0.003$), suggesting that Bronchiectasis patients had a significantly lower BMI compared to COPD patients. (Table 2)

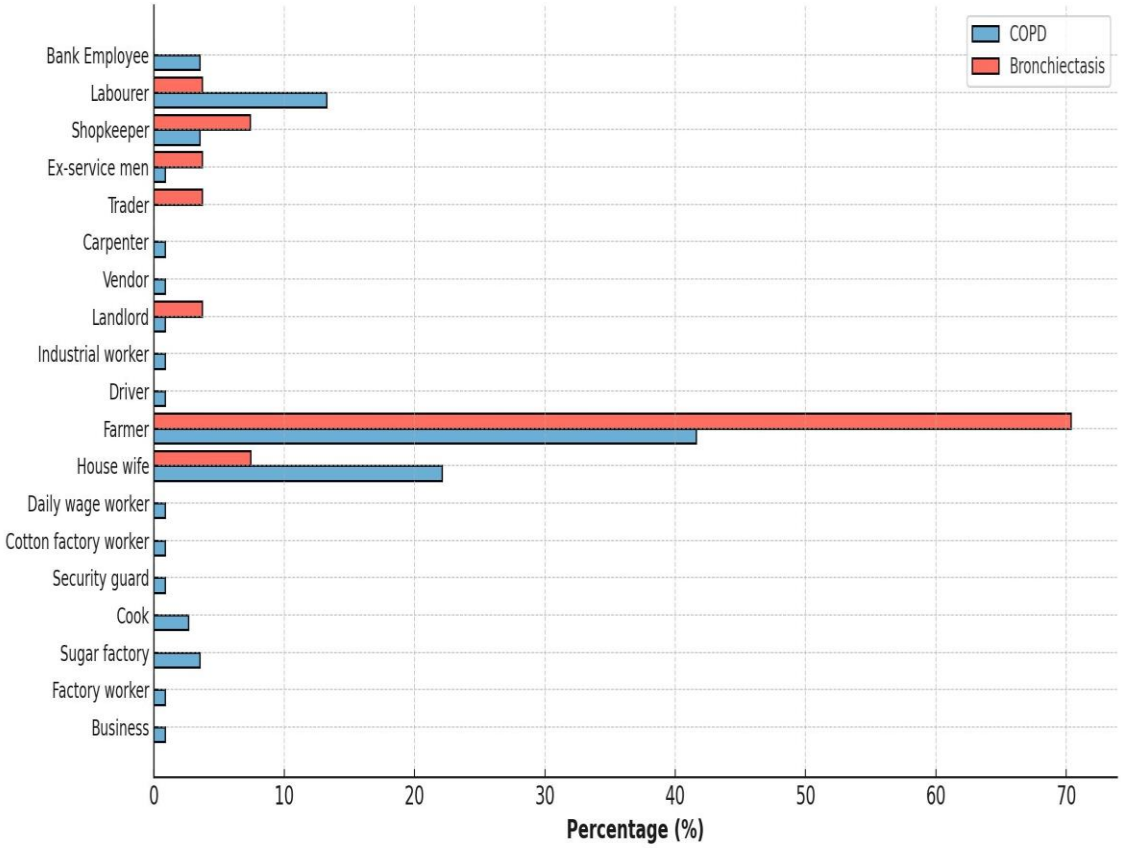
Fig 3. Prevalence of Bronchiectasis in COPD Patients



The given pie chart (Fig 3) illustrates the prevalence of bronchiectasis among COPD patients in the study population. The total number of COPD patients in this dataset is 146, out of which, 34 patients (23.3%) were found to have coexisting bronchiectasis and 112 patients (76.7%) had COPD without bronchiectasis.

This suggests that nearly one-fourth of COPD patients in this study also exhibit bronchiectasis, indicating a significant overlap between the two conditions.

Fig 4. Comparison of occupational Status



The bar chart (Fig 4) compares the occupational distribution of COPD and bronchiectasis patients. Farmers form the largest group in both categories, with a higher percentage in bronchiectasis patients. Housewives and labourers are also common, with labourers more prominent in COPD. Other occupations like shopkeepers, traders, ex-servicemen, and landlords are present in smaller numbers. Certain occupations, such as bank employees, security guards, and cooks, appear only in COPD patients.

Table 3. Prevalence of comorbidities

Comorbidity	COPD (n=112)	COPD with Bronchiectasis (n=34)	P-Value
Hypertension (HTN)	44 (39.3%)	21 (61.8%)	0.035
Type 2 Diabetes Mellitus (T2DM)	29 (25.9%)	17 (50.0%)	0.015
Chronic Kidney Disease (CKD)	0 (0.0%)	2 (5.9%)	0.081
Ischemic Heart Disease (IHD)	15 (13.4%)	12 (35.3%)	0.009
Chronic Liver Disease (CLD)	0 (0.0%)	1 (2.9%)	0.526

The bar chart (Fig 5) and table (Table 3) illustrate the prevalence of comorbidities among COPD and bronchiectasis patients. Hypertension (HTN) is the most common comorbidity, affecting 61.8% of bronchiectasis patients compared to 39.3% of COPD patients. Similarly, Type 2 Diabetes Mellitus (T2DM) is more frequent in bronchiectasis (50%) than in COPD (25.9%). Ischemic Heart Disease (IHD) is also significantly higher in bronchiectasis (35.3%) compared to COPD (13.4%). While Chronic Kidney Disease (CKD) and Chronic Liver Disease (CLD) are less prevalent, they are more frequently observed in bronchiectasis patients.

Fig 5. Comorbidities Comparison: COPD vs COPD with Bronchiectasis group

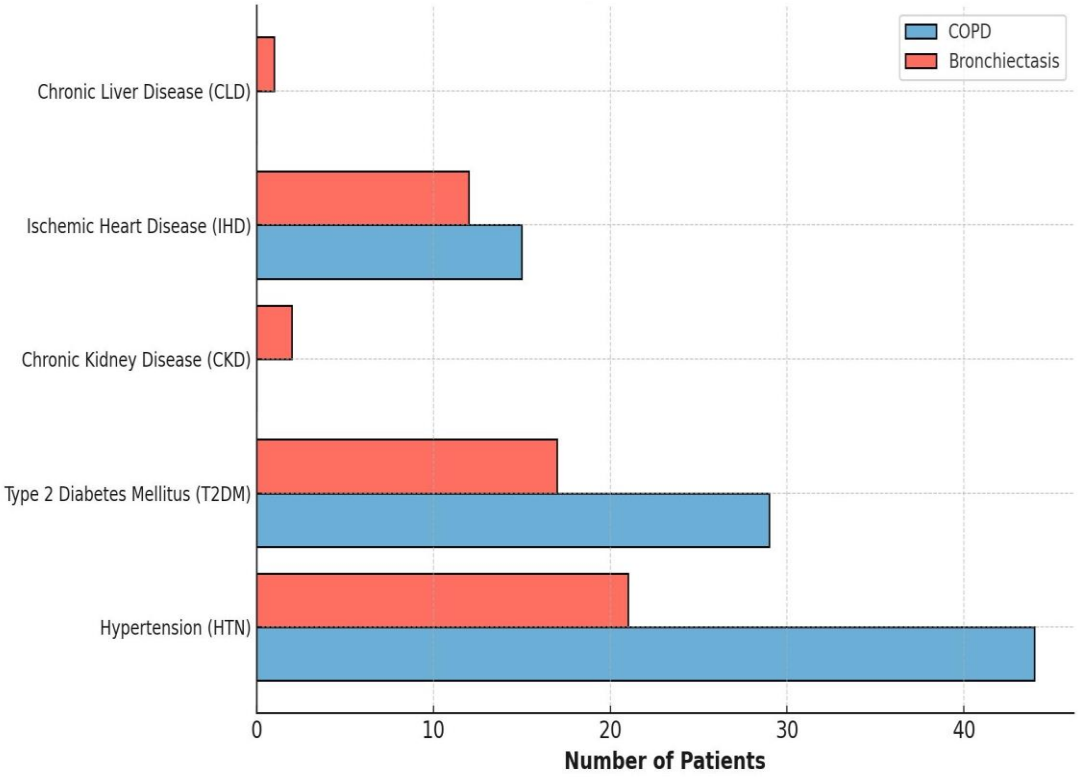
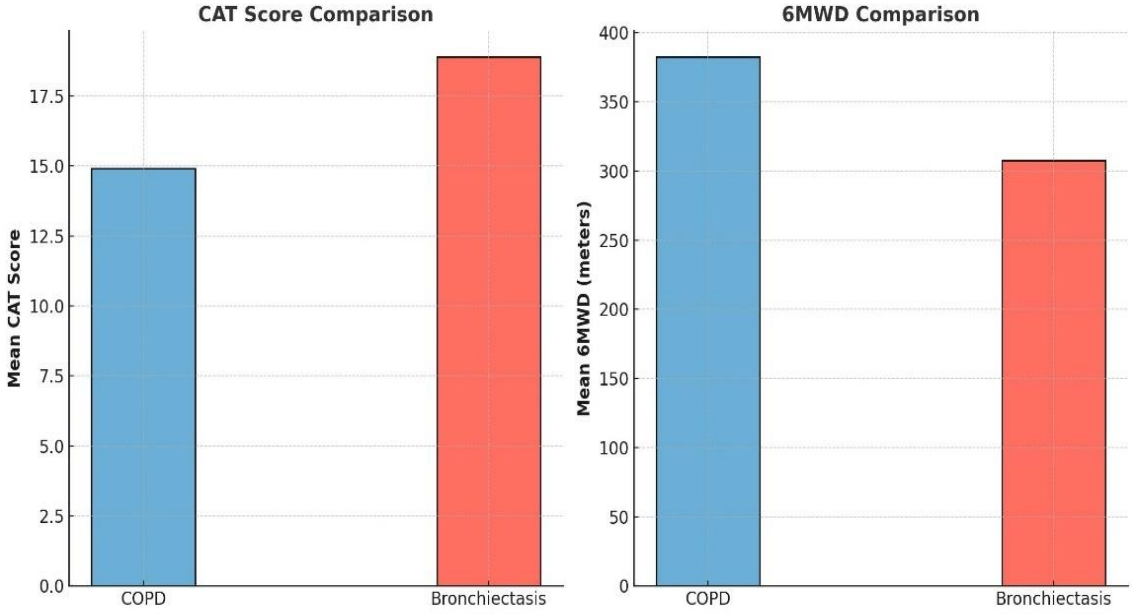


Table 4. Comparison of CAT Score and 6MWD

Parameter	COPD (n=112)	COPD with Bronchiectasis (n=34)	P-Value
CAT Score (Mean ± SD)	14.9 ± 4.1	18.9 ± 4.4	<0.001
6MWD (meters, Mean ± SD)	382.7 ± 126.9	307.6 ± 70.8	0.001

Fig 6. Comparison of CAT Score and 6MWD: COPD vs COPD with Bronchiectasis



The table (Table 4) and bar chart (Fig 6) compare the CAT score and 6MWD between COPD and bronchiectasis patients. The CAT score, which measures symptom burden, was observed to be significantly higher in bronchiectasis patients (18.9 ± 4.4) compared to COPD patients (14.9 ± 4.1), ($P<0.001$), indicating greater symptom severity in bronchiectasis.

Conversely, the 6MWD, a marker of exercise capacity, is lower in bronchiectasis patients (307.6 ± 70.8 m) compared to COPD patients (382.7 ± 126.9 m), with a P-value of 0.001, suggesting reduced physical endurance in bronchiectasis. The bar charts visually depict these differences, reinforcing that bronchiectasis patients experience more symptoms and greater functional impairment than COPD patients.

Table 5. Comparison of MMRC Grades

MMRC Grade	COPD (n=112)	COPD with Bronchiectasis (n=34)	P-Value
Grade 1	48 (42.9%)	4 (11.8%)	<0.001
Grade 2	44 (39.3%)	12 (35.3%)	0.725
Grade 3	16 (14.3%)	14 (41.2%)	<0.001
Grade 4	4 (3.6%)	4 (11.8%)	0.041

The bar chart (Fig 7) and table (Table 5) compare the Modified Medical Research Council (MMRC) dyspnea grades between COPD and bronchiectasis patients. Grade 1 dyspnea is significantly more common in COPD patients (42.9%) than in bronchiectasis (11.8%) ($p < 0.001$). Grade 3 and 4 dyspnea are more frequent in bronchiectasis (41.2% and 11.8%, respectively) compared to COPD (14.3% and 3.6%), with significant p-values (<0.001 and 0.041). Grade 2 dyspnea is similar in both groups ($p = 0.725$). The chart visually reflects that higher MMRC grades are more common in bronchiectasis, indicating greater dyspnea severity in this group.

Fig 7. Comparison of MMRC Grades: COPD vs COPD with Bronchiectasis

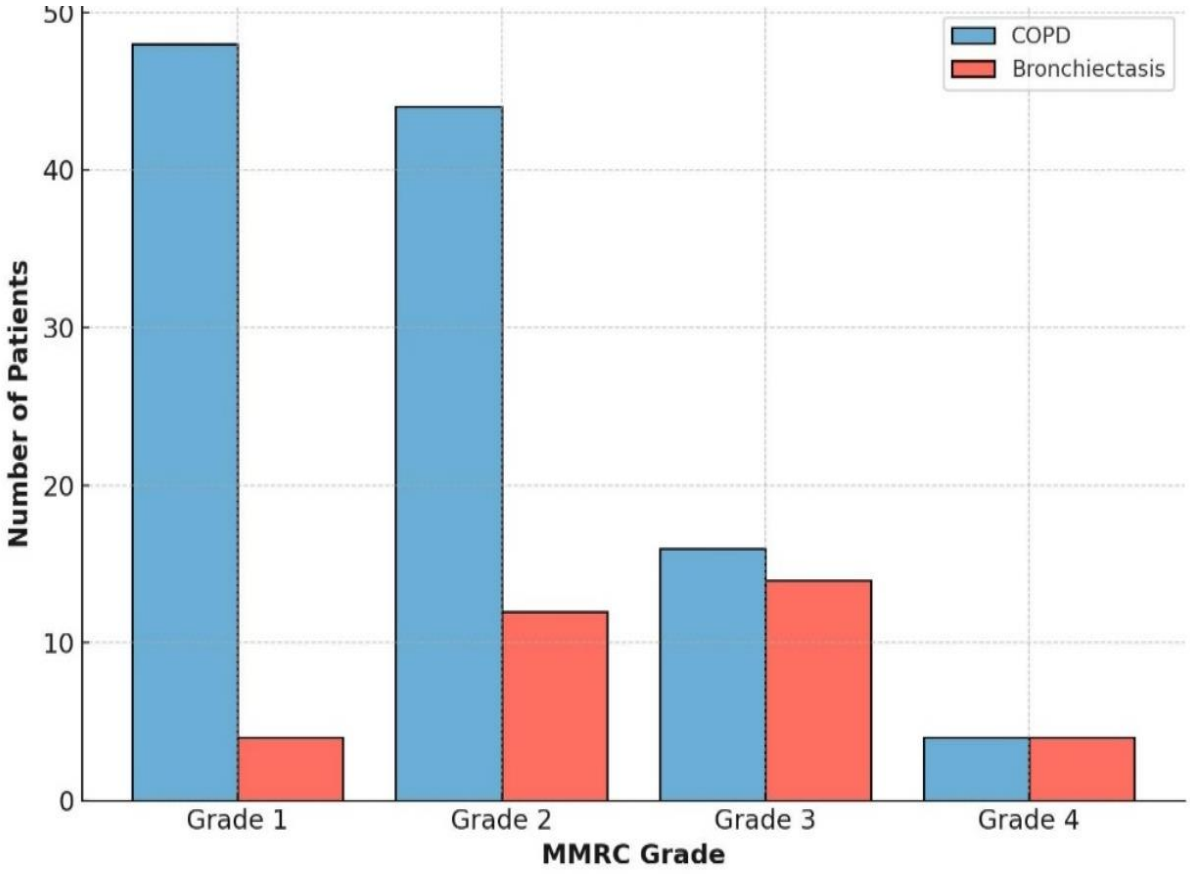


Table 6. Comparison of Pulmonary Function Tests

Pulmonary Function Test	COPD (n=112)	COPD with Bronchiectasis (n=34)	P-Value
FEV1 (% predicted)	59.4 ± 12.4	54.5 ± 10.6	0.026
FVC (% predicted)	86.3 ± 18.6	77.7 ± 15.0	0.008
FEV1/FVC ratio in %	67 ± 5	69 ± 7	0.191
FEF 25-75 (% predicted)	45.5 ± 15.3	39.8 ± 14.7	0.053
PEFR (% predicted)	49.9 ± 19.1	42.4 ± 14.9	0.018

The bar chart (Fig 8) and table (Table 6) compare pulmonary function test (PFT) parameters between COPD and bronchiectasis patients. FEV1 (% predicted) and FVC (% predicted) were lower in bronchiectasis patients (54.5 ± 10.6 and 77.7 ± 15.0) compared to COPD patients (59.4 ± 12.4 and 86.3 ± 18.6), with significant p-values (0.026 and 0.008). PEFR is also lower in bronchiectasis (42.4 ± 14.9) vs. COPD (49.9 ± 19.1) ($p = 0.018$). FEV1/FVC ratio and FEF25-75% show no significant difference ($p = 0.191, 0.053$). The chart highlights reduced lung function in bronchiectasis across multiple parameters compared to COPD.

Fig 8. Comparison of Pulmonary Function Tests: COPD vs COPD with Bronchiectasis

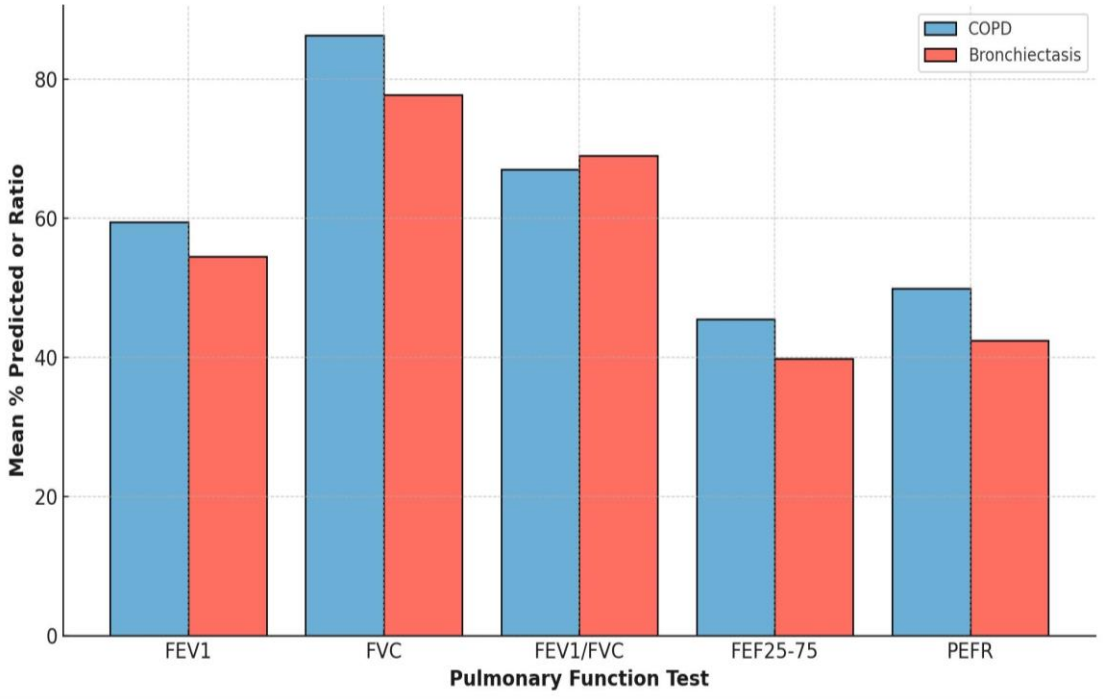


Table 7. Comparison of GOLD ABE Classification

GOLD ABE Classification	COPD (n=112)	COPD with Bronchiectasis (n=34)	P-Value
A (Low Risk, Less Symptoms)	54 (48.2%)	1 (2.9%)	<0.001
B (Low Risk, More Symptoms)	54 (48.2%)	12 (35.3%)	<0.001
E (Frequent Exacerbator)	4 (3.6%)	21 (61.8%)	<0.001

The table (Table 7) and bar chart (Fig 9) compare GOLD ABE classification between COPD and bronchiectasis patients. In COPD, most patients fall into Group A (48.2%) and Group B (48.2%), with very few in Group E (3.6%). In contrast, bronchiectasis patients were predominantly in Group E (61.8%), indicating frequent exacerbations, while only 2.9% were in Group A and 35.3% in Group B. The p-values (<0.001) indicate significant differences between the two groups. The chart visually reflects this distribution, with Group E (frequent exacerbators) being more common in bronchiectasis than in COPD.

Fig 9. Comparison of GOLD ABE Classification: COPD vs COPD with Bronchiectasis

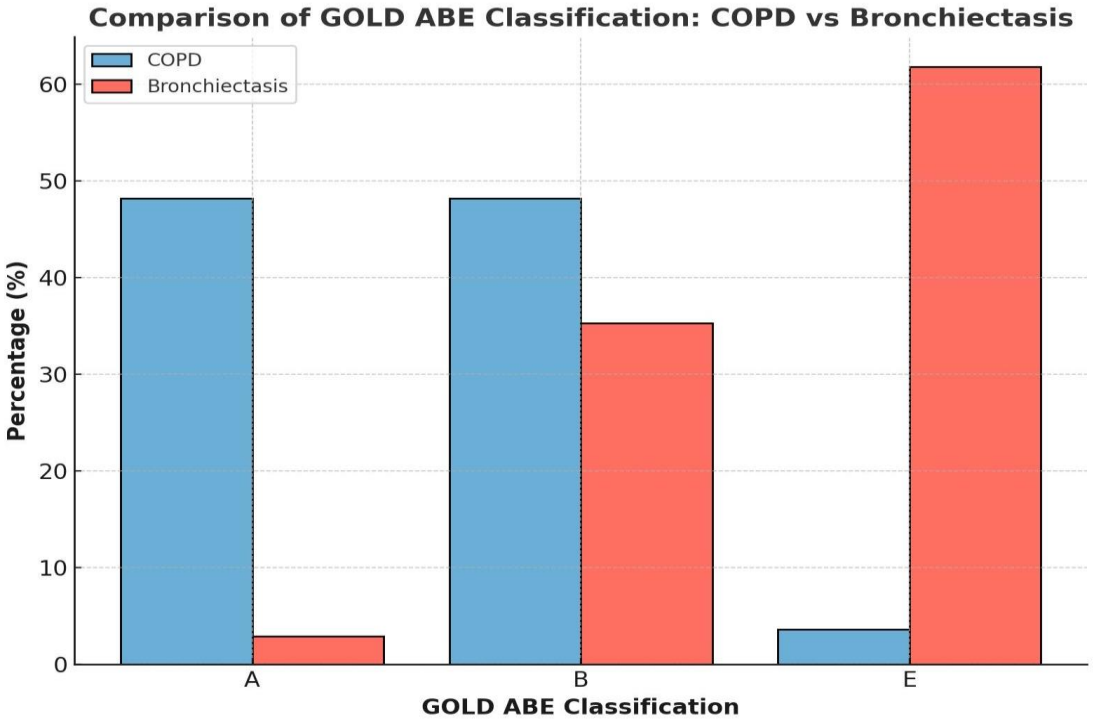


Table 8. Comparison of GOLD Classification:

GOLD Grade	COPD (n=112)	COPD with Bronchiectasis (n=34)	P-Value
1 (Mild)	2.65%	0.00%	0.035
2 (Moderate)	74.34%	51.85%	0.035
3 (severe)	19.47%	48.15%	0.035
4 (very severe)	3.54%	0.00%	0.035

The table (Table 8) and bar chart (Fig 10) compare GOLD grading between COPD and bronchiectasis patients. In COPD, the majority of patients fall under GOLD Grade 2 (74.34%), followed by Grade 3 (19.47%), with a small proportion in Grade 1 (2.65%) and Grade 4 (3.54%). In bronchiectasis, a larger proportion is in Grade 3 (48.15%), with fewer in Grade 2 (51.85%), and none in Grade 1 or 4. The p-values (0.035 for all grades) indicate significant differences between the groups. The bar chart visually highlights that bronchiectasis patients are more likely to have severe airflow limitation (Grade 3) compared to COPD.

Fig 10. Comparison of GOLD Classification: COPD vs COPD with Bronchiectasis

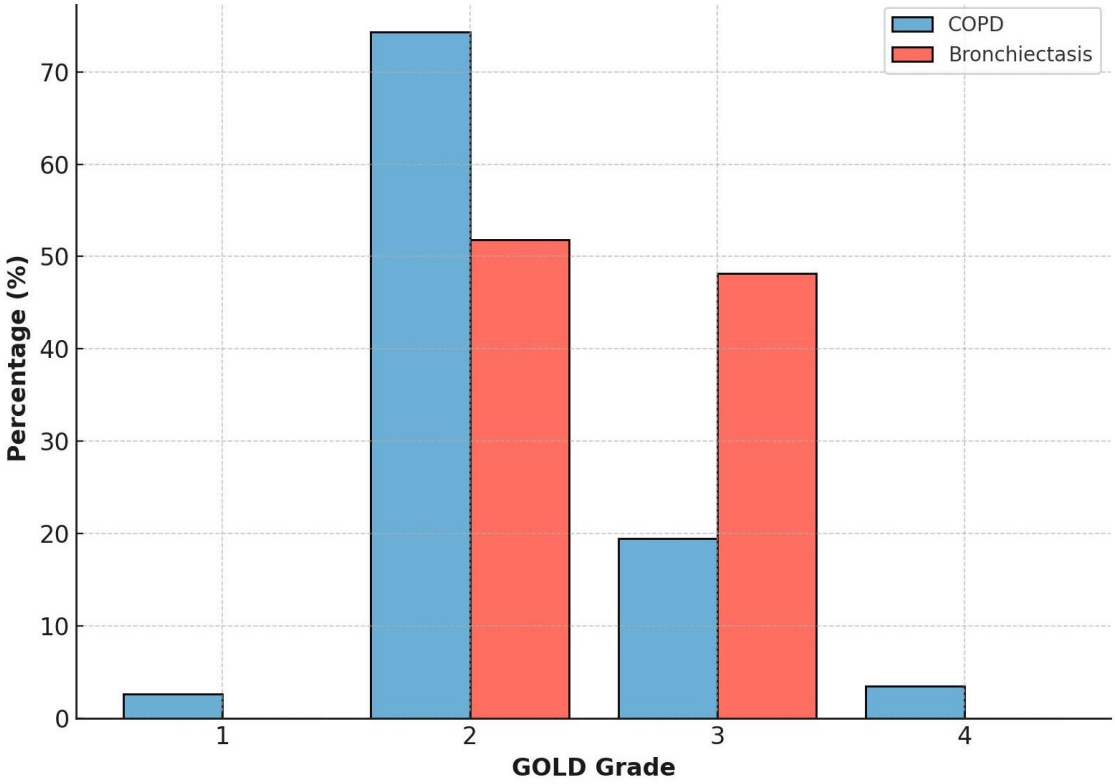


Table 9. Comparison of Smoking History, Symptoms Duration, and History of Previous Admissions

Smoking History	**Smoker: 71(63.4%)** **Non-smoker: 41 (36.6%)**	**Smoker: 25 (73.5%)** **Non-smoker: 9 (26.5%)**	**0.376**
Symptoms Duration (Years, Mean ± SD)	7.1 ± 3.8	9.4 ± 4.5	0.009
Previous Admissions History (Last 2 Years, Mean ± SD)	1.3 ± 0.9	3.1 ± 0.8	<0.001

The table (Table 9) and bar chart (Fig 11) compare smoking history, symptom duration, and previous hospital admissions between COPD and bronchiectasis patients. Smoking history was similar in both groups (COPD: 63.4% smokers, bronchiectasis: 73.5% smokers, $p = 0.376$), showing no significant difference. Symptom duration was longer in bronchiectasis (9.4 ± 4.5 years) compared to COPD (7.1 ± 3.8 years) ($p = 0.009$). Previous admissions in the last 2 years were significantly higher in bronchiectasis (3.1 ± 0.8) compared to COPD (1.3 ± 0.9) ($p < 0.001$), indicating a higher frequency of hospitalizations. The bar charts visually depict these differences, highlighting that bronchiectasis patients tend to have longer symptom duration and more frequent hospital admissions than COPD patients.

Fig 11. Comparison of Smoking History, Symptoms Duration, and History of Previous Admissions: COPD vs COPD with Bronchiectasis

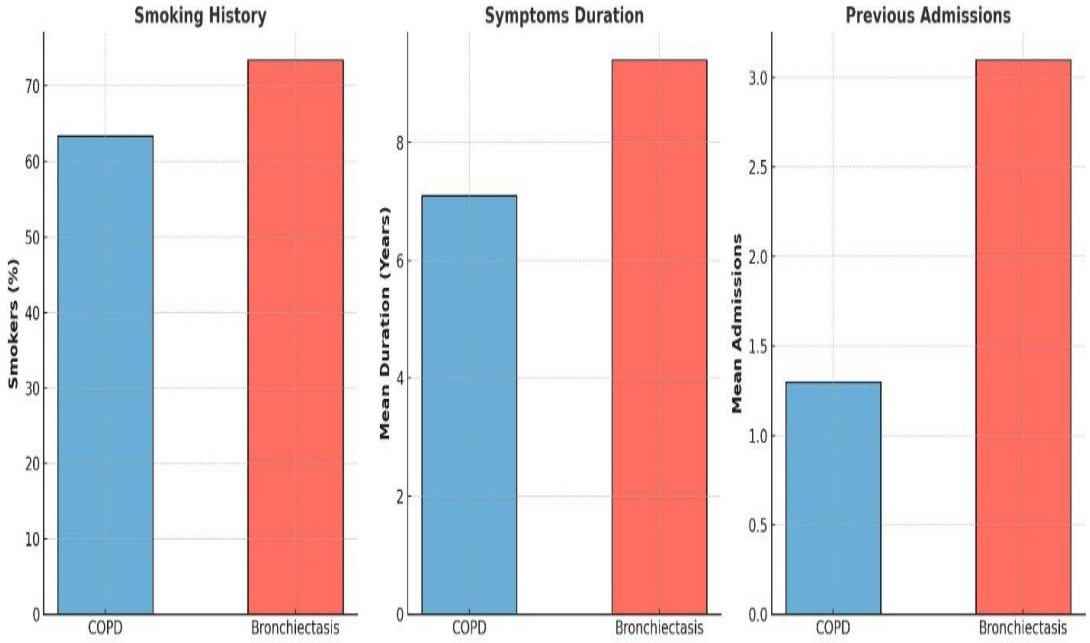


Table 10. Comparison of Chronic Bronchitis and Emphysema Phenotypes.

Phenotype	COPD (n=112)	COPD with Bronchiectasis (n=34)	P-Value
Chronic Bronchitis (CB), n (%)	24 (21.4%)	23 (67.6%)	<0.001
Emphysema (E), n (%)	88 (78.6%)	11 (32.4%)	<0.001

The table (Table 10) and pie chart (Fig 12) compare chronic bronchitis (CB) vs. emphysema predominance in COPD and bronchiectasis patients. In COPD, the majority of patients have emphysema (78.6%), while only 21.4% present with chronic bronchitis. In contrast, bronchiectasis patients predominantly exhibit chronic bronchitis (67.6%), with only 32.4% showing emphysema predominance. The p-values (<0.001) indicate a statistically significant difference between groups.

The pie charts visually highlight these differences, showing that emphysema is more common in COPD, whereas chronic bronchitis predominates in bronchiectasis patients.

Fig 12. Comparison of Chronic Bronchitis and Emphysema Phenotypes: in COPD vs COPD with Bronchiectasis groups

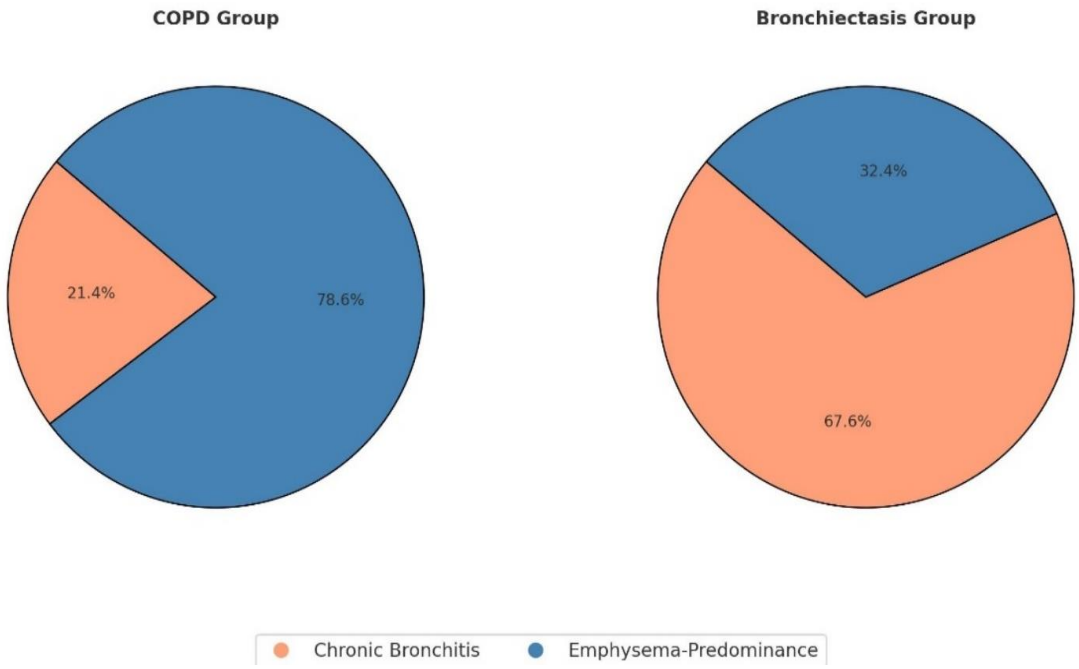


Table 11. comparison of Sputum culture isolates

ORGANISMS ISOLATED IN SPUTUM CULTURES	COPD	COPD WITH BRONCHIECTASIS	P-VALUE
ACINETOBACTOR BAUMANNII	0 (0.0%)	1 (2.94%)	0.537
COAGULASE NEGATIVE STREPTOCOCCI	1 (0.89%)	2 (5.88%)	0.290
ECOLI	0 (0.0%)	1 (2.94%)	0.537
ENTEROCOCCUS FAECALIS	3 (2.68%)	2 (5.88%)	0.742
KLEBSIELLA OXYTOCA	0 (0.0%)	2 (5.88%)	0.092
KLEBSIELLA PNEUMONIA	1 (0.89%)	3 (8.82%)	0.075
MRSA	0 (0.0%)	1 (2.94%)	0.537
NOG	14 (12.5%)	3 (8.82%)	0.826
PSEUDOMONAS AUREGINOSA	1 (0.89%)	4 (11.76%)	0.018
STAPH AUREUS	0 (0.0%)	1 (2.94%)	0.537
SPUTUM CULTURES UNAVAILABLE	84 (75.0%)	7 (20.59%)	0.003
COMMENSALS ISOLATED	8 (7.14%)	7 (20.59%)	0.095
TOTAL	112 (100.00%)	34 (100.00%)	

Fig 13 A. Sputum culture isolates in COPD group

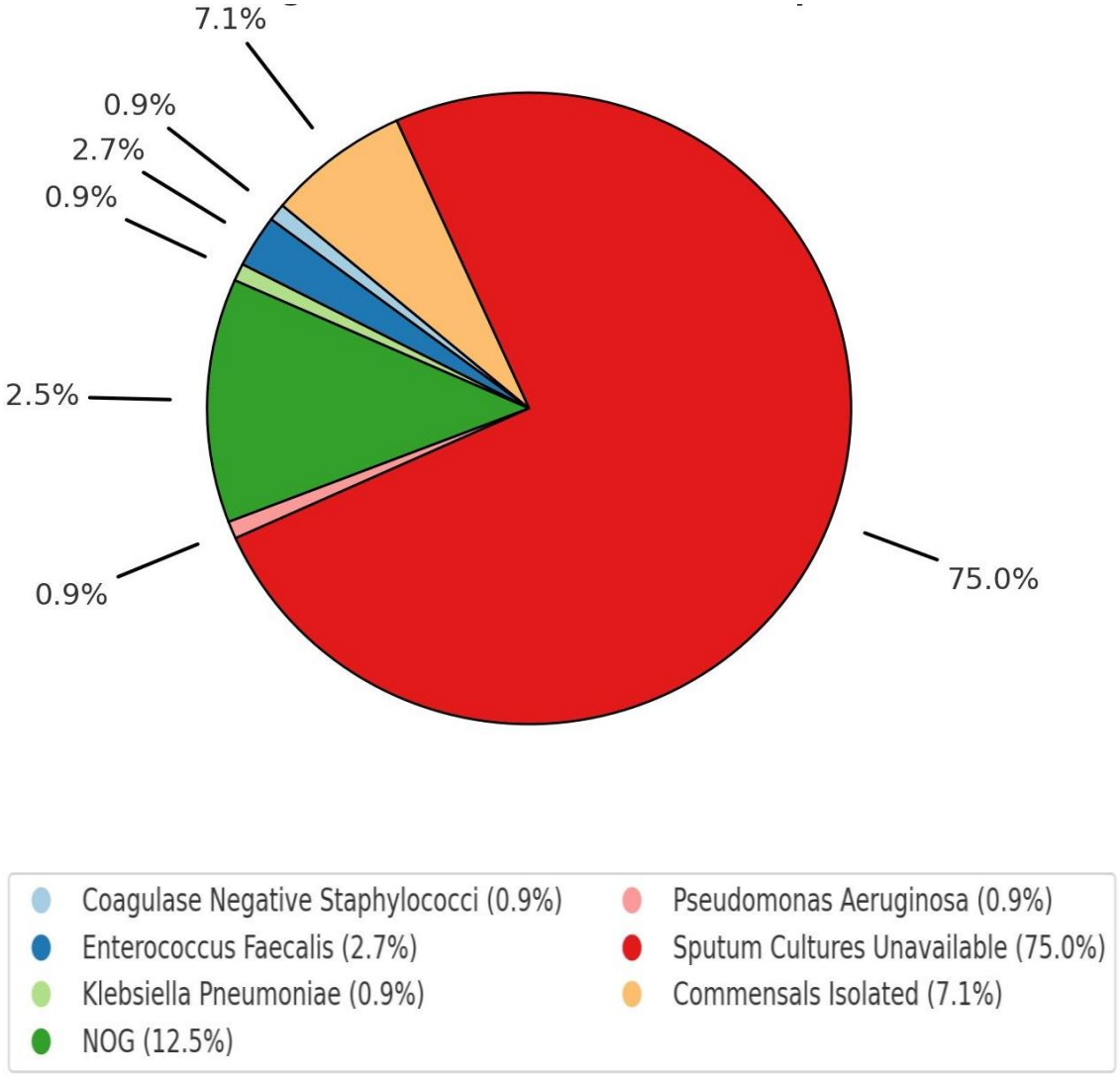
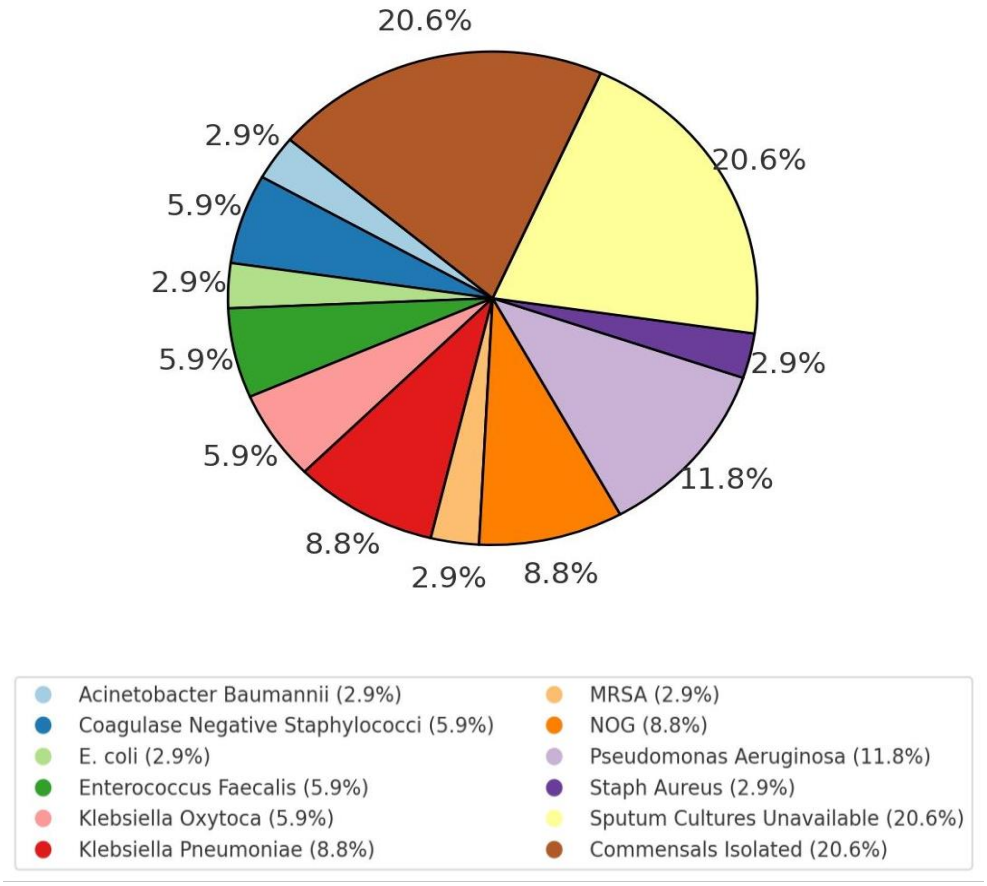


Fig 13 B. Sputum culture isolates in COPD with bronchiectasis group



The table (Table 11) and pie charts (Fig 13 A, Fig 13 B) summarize the distribution of organisms isolated in sputum cultures from COPD and COPD with bronchiectasis patients. In COPD, sputum cultures were unavailable in 75%, with NOG (12.5%) and commensals (7.14%) being the most detected organisms. In contrast, in COPD with bronchiectasis, sputum cultures were unavailable in only 20.59%, with commensals (20.59%) and Pseudomonas aeruginosa (11.76%) being more prevalent.

A significant difference (p=0.018) was found in Pseudomonas aeruginosa presence between groups, and sputum culture availability was significantly higher in bronchiectasis cases (p=0.003). The data highlights microbial differences in these conditions, with Pseudomonas aeruginosa being notably more frequent in bronchiectasis.

Table 12. Comparison of Blood Parameters

Blood Parameter	COPD (n=112)	COPD with Bronchiectasis (n=34)	P-Value
Hemoglobin (g/dL, Mean ± SD)	12.29 ± 2.04	12.04 ± 2.50	0.553
Total Leukocyte Count (×10³/µL, Mean ± SD)	7.12 ± 2.77	10.86 ± 4.07	<0.001
Neutrophils (% of total leukocytes, Mean ± SD)	55.11 ± 14.19	77.47 ± 12.72	<0.001
Serum Albumin (g/dL, Mean ± SD)	3.96 ± 1.38	3.32 ± 0.84	0.011

The table (Table 12) and bar chart (Fig 14) compare blood parameters between COPD and bronchiectasis patients. Hemoglobin levels were similar in both groups ($p = 0.553$). However, total leukocyte count (10.86 ± 4.07 vs. 7.12 ± 2.77, $p < 0.001$) and neutrophil percentage (77.47 ± 12.72 vs. 55.1 ± 14.19, $p < 0.001$) were significantly higher in bronchiectasis, indicating increased systemic inflammation. Serum albumin levels were lower in bronchiectasis (3.32 ± 0.84) compared to COPD (3.96 ± 1.38) ($p = 0.011$). The bar chart visually reflects these differences, highlighting higher inflammatory markers in bronchiectasis patients.

Fig 14. Comparison of Blood Parameters: COPD vs COPD with Bronchiectasis

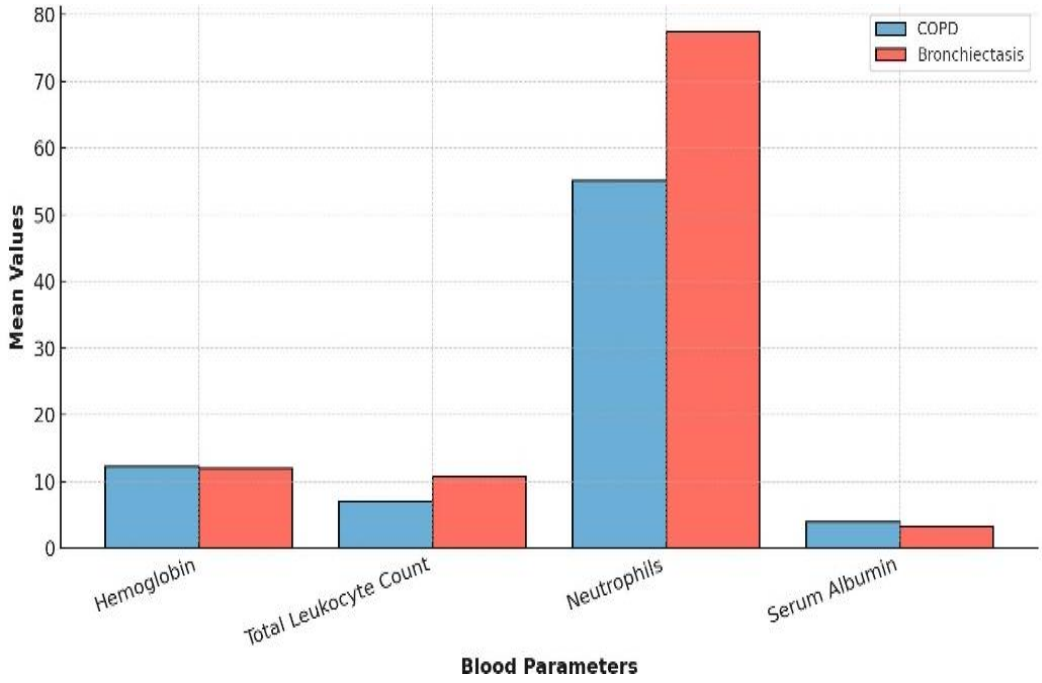


Table 13. Comparison of BMI and Albumin

Parameter	COPD (n=112)	COPD with Bronchiectasis (n=34)	P-Value
BMI (kg/m², Mean ± SD)	24.1 ± 3.7	21.5 ± 4.3	0.003
Serum Albumin (g/dL, Mean ± SD)	3.96 ± 1.38	3.32 ± 0.84	0.011

The table (Table 13) and bar chart (Fig 15) compare BMI and serum albumin levels between COPD and bronchiectasis patients. BMI was significantly lower in bronchiectasis (21.5 ± 4.3) compared to COPD (24.1 ± 3.7) ($p = 0.003$), indicating a higher prevalence of malnutrition in bronchiectasis patients. Similarly, serum albumin levels were lower in bronchiectasis (3.32 ± 0.84) compared to COPD (3.96 ± 1.38) ($p = 0.011$), suggesting greater systemic inflammation or nutritional deficiency. The bar charts visually represent these differences, showing that bronchiectasis patients have lower BMI and serum albumin compared to COPD patients.

Fig 15. Comparison of BMI and Albumin: COPD vs COPD with Bronchiectasis

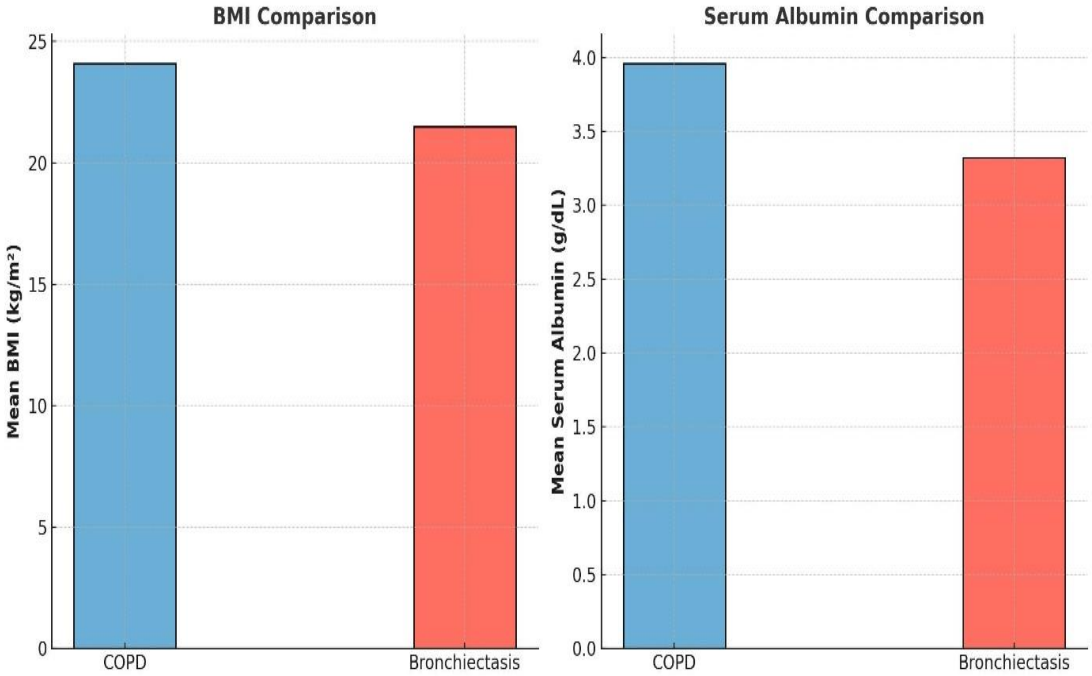
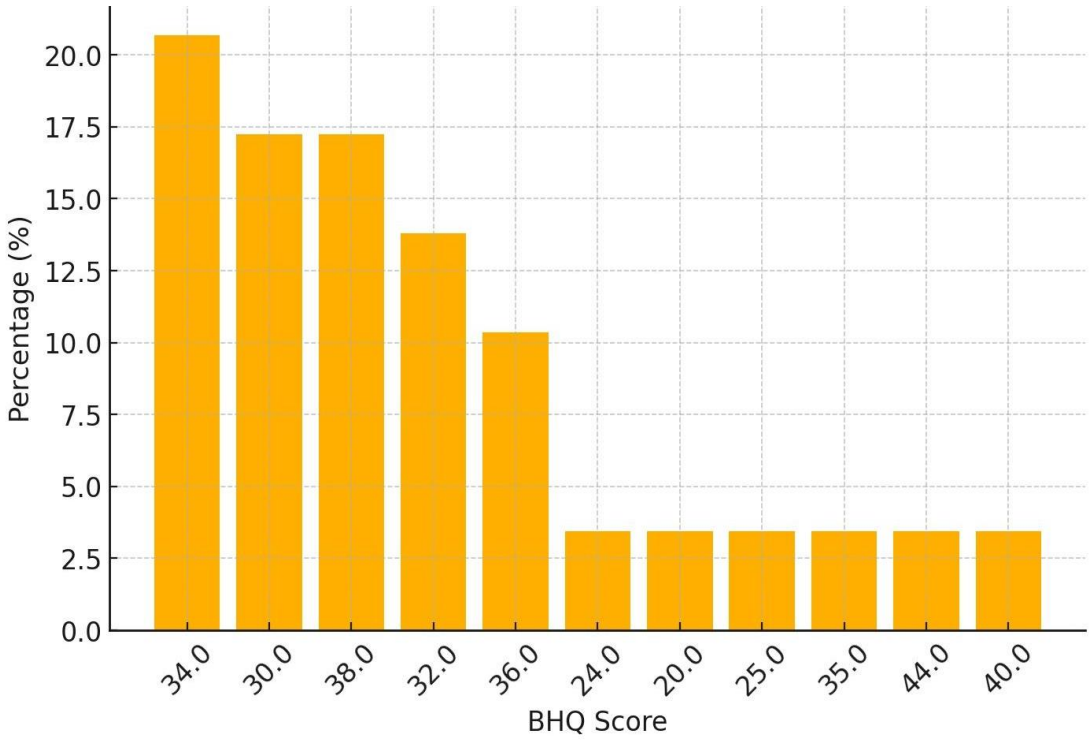


Fig 16. Bronchiectasis Health Questionnaire scores in COPD with bronchiectasis patients



The bar graph (Fig 16) illustrates the distribution of Bronchiectasis Health Questionnaire (BHQ) scores among patients with bronchiectasis. The most common BHQ scores observed were 34 and 38, each comprising around **20.7% and 17.2%** of the patients, respectively. Scores of 30 and 32 also show a similar distribution, with approximately **17.2% and 13.8%** of patients falling into these categories.

The least common BHQ score in this dataset is **36**, accounting for **10.3%** of the bronchiectasis group. This distribution suggests a variability in health-related quality of life among bronchiectasis patients, with a tendency toward moderate BHQ scores.

Table 14. Radiological types of Bronchiectasis

TYPE OF BRONCHIECTASIS	COUNT	PERCENTAGE (%)
CYSTIC	13	38.235
TRACTION	11	32.353
CYLINDRICAL	8	23.529
TUBULAR	2	5.882
Total	34	100.0

The table (Table 14) pie chart (Fig 17) illustrate the distribution of bronchiectasis types among 34 patients. The most common type was cystic bronchiectasis (38.2%), followed by traction bronchiectasis (32.4%) and cylindrical bronchiectasis (23.5%), while tubular bronchiectasis was the least common (5.9%). The pie chart visually represents these proportions, highlighting that cystic and traction bronchiectasis are the predominant forms observed in this group.

Fig 17. Radiological types of Bronchiectasis

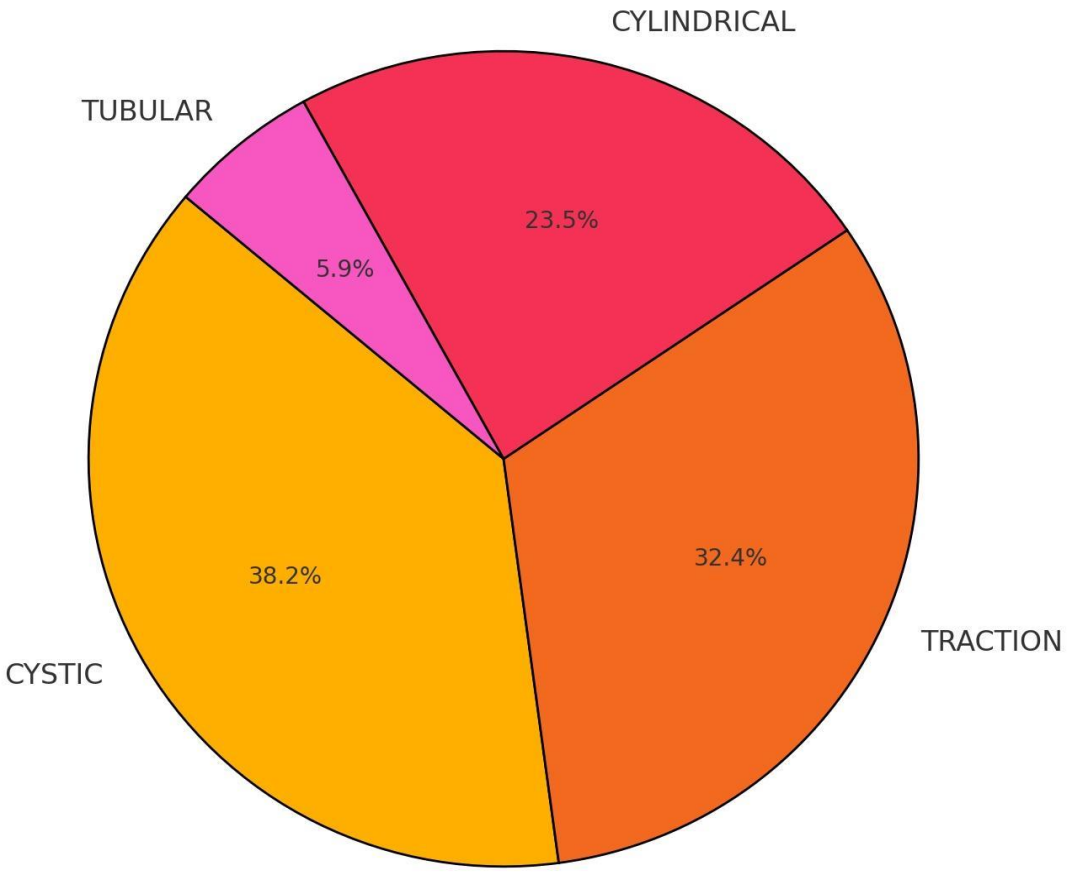


Table 15. Unilateral/Bilateral Distribution of Bronchiectasis

Unilateral/Bilateral Bronchiectasis	Count	Percentage (%)
BILATERAL	20	58.824
UNILATERAL	14	41.176
Total	34	100.0

The table (Table 15) and bar chart (Fig 18) display the distribution of unilateral and bilateral bronchiectasis among 34 patients. Bilateral bronchiectasis was more common (58.8%), indicating disease involvement in both lungs, while unilateral bronchiectasis was present in 41.2% of cases, affecting only one lung. The bar chart visually represents this distribution, showing a higher prevalence of bilateral bronchiectasis in the study population.

Fig 18. Unilateral/Bilateral Distribution of Bronchiectasis

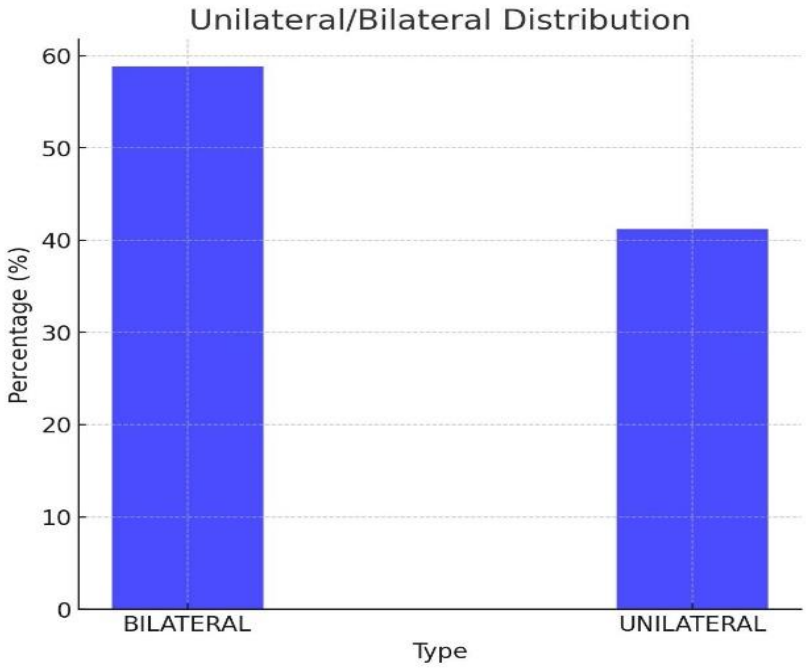


Table 16. Proximal/Distal Distribution of Bronchiectasis

PROXIMAL/DISTAL BRONCHIECTASIS	COUNT	PERCENTAGE (%)
DISTAL	24	70.588
PROXIMAL	10	29.412
TOTAL	34	100.0

The table (Table 16) and bar chart (Fig 19) display the distribution of proximal and distal bronchiectasis among 34 patients. Distal bronchiectasis was more common (70.6%), indicating a predominant involvement of the peripheral airways. In contrast, proximal bronchiectasis was observed in 29.4% of cases, affecting the larger, central bronchi. The bar chart visually represents this distribution, emphasizing that bronchiectasis more frequently affects the distal airways.

Fig 19. Proximal/Distal Distribution of Bronchiectasis

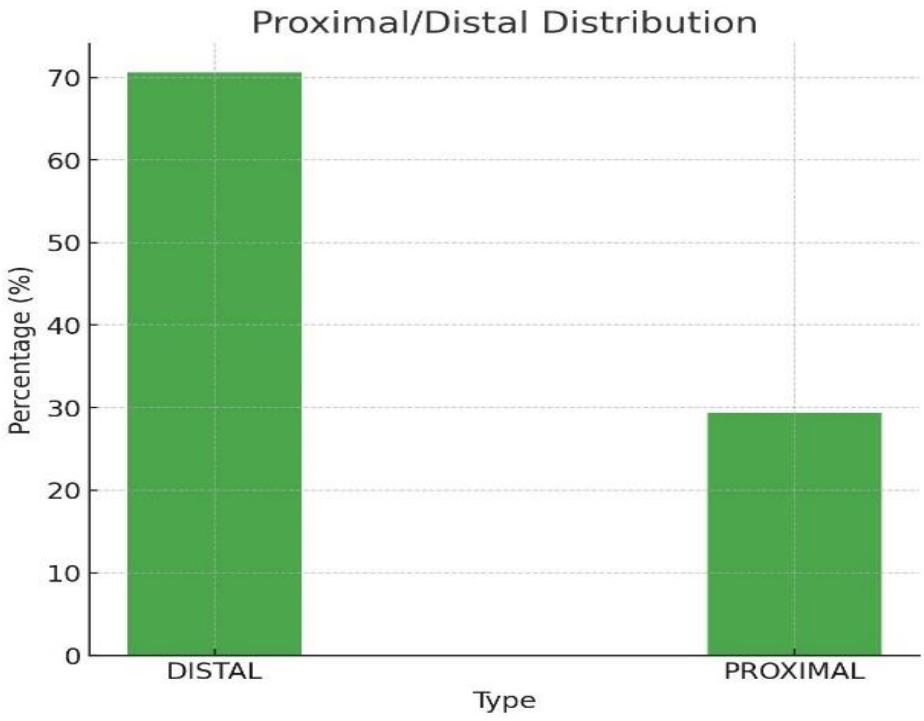


Table 17. Other findings in HRCT Thorax

HRCT Findings	Percentage
Pleural Effusion Present	15.90%
PAH Present	11.85%
Cardiomegaly Present	8.90%

The table (Table 17) resents the distribution of key HRCT findings in the study population. Pleural effusion was the most common finding, observed in 15.9% of cases, followed by pulmonary arterial hypertension (PAH) in 11.85%, and cardiomegaly in 8.9% of cases. These findings indicate associated complications commonly seen in chronic lung diseases.

DISCUSSION

Baseline Characteristics and Clinical Differences

This study compares the baseline characteristics of COPD-only (n=112) patients with those having COPD and bronchiectasis (n=34), emphasizing key differences in lung function, symptom burden, disease severity, and comorbidities.

Gender and age, as mentioned, did not significantly differ in our cohort between those with and without bronchiectasis. Males were predominant in both groups (≈91% in bronchiectasis vs 77% in COPD-only, p = 0.110), reflecting the demographics of smoking-related COPD in our region. Some studies, like Sangtam et al [5] even observed a slightly higher bronchiectasis prevalence in female COPD patients (though not significant), suggesting no strong sex predilection when exposure factors are accounted for. Age likewise was not a discriminator once disease duration was considered. Therefore, neither advanced age nor female sex appears to inherently predispose to bronchiectasis in COPD – it is more about the clinical phenotype and history as outlined above.

Patients with COPD and bronchiectasis had a significantly lower BMI (21.5 ± 4.3 kg/m²) compared to those with COPD alone (24.1 ± 3.7 kg/m², p=0.003). Lung function parameters were also notably reduced in the bronchiectasis group, including FEV1 (% predicted) (54.5 ± 10.6 vs. 59.4 ± 12.4, p=0.026), FVC (% predicted) (77.7 ± 15.0 vs. 86.3 ± 18.6, p=0.008), and PEFR (% predicted) (42.4 ± 14.9 vs. 49.9 ± 19.1, p=0.018). These findings suggest that bronchiectasis contributes to greater airflow obstruction and impaired respiratory mechanics.

The symptom burden was significantly higher in the COPD with bronchiectasis group. The MMRC grade was 2.4 ± 0.7, compared to 1.7 ± 0.8 in COPD-only patients ($p<0.001$), indicating more pronounced dyspnea. Similarly, the CAT score was 18.9 ± 4.4 in the bronchiectasis group versus 14.9 ± 4.1 in COPD-only patients ($p<0.001$), reflecting worse health-related quality of life. In terms of GOLD classification, most COPD with bronchiectasis patients were categorized in group E (61.8%), which is associated with frequent exacerbations, whereas COPD-only patients were primarily in groups A and B (48.2% each, $p<0.001$). In GOLD grading, the bronchiectasis group had a higher proportion of GOLD grade 3 (44.1%), while 74.1% of COPD-only patients were in grade 2 ($p=0.023$). These results indicate that bronchiectasis is linked to more severe COPD phenotypes.

Regarding disease progression, COPD with bronchiectasis patients had longer symptom durations (9.4 ± 4.5 years vs. 7.1 ± 3.8 years, $p=0.009$), suggesting earlier disease onset or slower resolution of symptoms. Moreover, their hospital admissions in the last two years (3.1 ± 0.8 vs. 1.3 ± 0.9, $p<0.001$) were significantly higher, emphasizing the greater healthcare burden and the need for more intensive disease management.

Comorbidities were also more prevalent in the COPD with bronchiectasis group. Hypertension (HTN) was present in 61.8% of these patients compared to 39.3% in COPD-only patients ($p=0.035$). Type 2 diabetes mellitus (T2DM) was also significantly higher (50.0% vs. 25.9%, $p=0.015$), along with ischemic heart disease (IHD) (35.3% vs. 13.4%, $p=0.009$). Although chronic kidney disease (CKD) and chronic liver disease (CLD) were slightly more frequent in the bronchiectasis group, the differences were not statistically significant. These findings highlight the increased systemic disease burden in COPD patients with bronchiectasis.

Prevalence of Bronchiectasis in COPD Patients

In our study the prevalence of bronchiectasis among COPD patients was 23.3% among COPD patients (34 of 146 cases). This prevalence falls within the range reported in literature [4-8], though it is on the lower end of the wide spectrum noted across different studies. Prior investigations have found prevalence of bronchiectasis varies from ~4% of mild COPD cases up to over 50% in severe COPD cohorts. Chalmers et al [4] reported in a meta-analysis that up to 54.3% of COPD patients had radiological bronchiectasis, though estimates varied greatly depending on the population and diagnostic methods. Martinez-Garcia et al [2] also observed that approximately 50% of moderate-to-severe COPD patients may have coexistent bronchiectasis when systematically evaluated with HRCT. Conversely, some large-scale studies have found more modest rates, Dou et al [6] observed an overall prevalence of only 8.1% in a Chinese COPD cohort (mean FEV_1 ~45% predicted), increasing to ~16.5% in those with emphysema-predominant COPD. Our prevalence of ~23% is comparable to studies focusing on severe COPD or hospital-based samples. Notably, a recent Indian study by Sangtam et al [5] reported a bronchiectasis prevalence of 17.7% among COPD patients in a tertiary center, slightly lower than ours; this difference could reflect regional variations or differences in patient selection (e.g. our cohort may have included patients with more frequent exacerbations or referred for evaluation of symptoms, thus increasing detection rate). Overall, our finding reinforces that a substantial minority (approximately one in four to one in five) of COPD patients have coexistent bronchiectasis, consistent with the growing recognition of COPD-bronchiectasis overlap as a common and clinically relevant phenotype.

It Is important to interpret prevalence in light of patient characteristics and diagnostic approach. Our study, like most, relied on HRCT scans to detect bronchiectasis, which is considered the gold standard. Many cases of bronchiectasis in COPD remain underdiagnosed in routine practice (where CT is not performed unless clinically suspected). Therefore, studies that proactively scan COPD patients (especially those with advanced disease or frequent infections) tend to report higher prevalence. [34,35]

Gatheral et al [7] found bronchiectasis in 69% of COPD patients in their UK cohort – an unusually high figure likely due to including patients with recurrent infections referred for CT. The variability in severity of COPD among study populations also influences prevalence, bronchiectasis is far more common in severe (GOLD 3–4) COPD than in mild disease. A meta-analysis by Ni et al [38] observed bronchiectasis in 23.7% of GOLD 3–4 patients vs. only 6.5% of GOLD 1–2. Our cohort predominantly included moderate-to-severe COPD (no patients were GOLD 1, and ~97% were GOLD ≥2), which aligns with the mid-range prevalence we observed. In summary, our prevalence data is similar to the previous studies while considering disease severity and diagnostic intensity, and they underscore that a notable proportion of COPD patients – especially those with advanced disease – have coexistent bronchiectasis.

Predisposing Risk Factors

In exploring why certain COPD patients develop bronchiectasis, our study identified multiple predisposing risk factors. We will discuss each factor in detail, highlighting its significance in our results.

i) Severe Airflow Limitation (Low FEV $_1$)

Worsening airflow obstruction emerged as a significant factor associated with bronchiectasis. COPD patients with bronchiectasis had poorer lung function on spirometry, mean FEV $_1$ was lower (54.5 ± 10.6% vs 59.4 ± 12.4% predicted in those without bronchiectasis; p = 0.026) and a greater proportion were in GOLD stage 3 (48% vs 19% in COPD-only). This was similar to the well-established trend, bronchiectasis is more prevalent in more advanced COPD. [4-8]

In our study, essentially all patients with bronchiectasis belonged to GOLD 2–3, with none in the mildest or very severe extremes, but notably nearly half were having GOLD 3 stage (severe obstruction) compared to only ~20% of COPD-only patients (p = 0.035). The association between low FEV_1 and bronchiectasis is consistently reported. [2]

Martinez-Garcia et al [2] observed that severe COPD ($FEV_1 \leq 50\%$ predicted) was an independent predictor of bronchiectasis, with nearly a four-fold higher odds (OR ~3.9, 95%CI 1.4–10.5) of having bronchiectasis compared to moderate COPD. Similarly, Dou et al [6] reported that COPD patients with bronchiectasis had significantly worse airflow limitation (mean FEV $_1$ ~36% vs 48% predicted in COPD-only, p < 0.001). A meta-analysis by Ni et al [38] also confirmed that COPD-bronchiectasis patients tend to have a much lower FEV $_1$% (pooled mean ~39% vs 51% in COPD-only; p<0.001). These converging data strongly suggest that more severe airflow obstruction predisposes COPD patients to developing bronchiectasis. (2,6,38)

ii) Frequent Exacerbations and Hospitalizations

A history of frequent acute exacerbations of COPD (AECOPD) emerged as one of the strongest distinguishing factors for bronchiectasis in our study. By the GOLD ABE classification, 61.8% of COPD patients with bronchiectasis were "frequent exacerbators" (Group E), compared to only 3.6% of those without bronchiectasis ($p < 0.001$). In practical terms, bronchiectasis patients had suffered far more exacerbations requiring healthcare utilization; they averaged 3.1 ± 0.8 hospital admissions in the past 2 years, versus 1.3 ± 0.9 in COPD-only patients ($p < 0.001$). This dramatic difference suggests that exacerbation-prone COPD patients are much more likely to develop bronchiectasis, or conversely that coexisting bronchiectasis itself leads to more frequent exacerbations – in reality, both may be true, reinforcing a self-perpetuating cycle.

Our findings are similar to those of other studies, Martinez-Garcia et al [2] who observed that COPD patients with bronchiectasis had an exacerbation rate of ~3.2 per year vs 1.7 per year in those without ($p<0.001$). Ni et al [38] further quantified that having ≥2 exacerbations/year increases the odds of bronchiectasis by about 60% (OR 1.62, $p = 0.008$). Frequent exacerbations, especially if they are severe enough to require hospitalization, thus appear both a marker and a risk factor for the COPD-bronchiectasis overlap. [2,38]

iii) Chronic Airway Infection and Colonization

Chronic bacterial infection of the airways is a pivotal factor in the pathogenesis of bronchiectasis, and our data provide indirect evidence of this in the COPD-bronchiectasis group. Although we did not culture sputum in all patients, we observed that bronchiectasis patients had significantly elevated biomarkers of infection/inflammation: their white blood cell counts (WBC) were markedly higher

(10.86 ± 4.07 vs 7.12 ± 2.77 ×10^9/L; p < 0.001), as were their neutrophil percentages (77.5% vs 55.1%; p < 0.001). Clinically, nearly two-thirds of our COPD-bronchiectasis patients presented with a chronic bronchitic phenotype (productive cough), suggesting ongoing mucus hypersecretion likely due to infection. These findings indicate that persistent airway infection and inflammation were much more pronounced in the overlap patients. This is in agreement with studies showing COPD-bronchiectasis patients often harbor pathogenic bacteria chronically.

Gatheral et al [7] reported that 69% of COPD patients with bronchiectasis had colonization with Pseudomonas aeruginosa a particularly virulent organism in bronchiectasis, as compared to only 21% of COPD patients without bronchiectasis. Similarly, a meta-analysis observed that Pseudomonas organisms were present in ~32% of COPD-bronchiectasis cases vs 8% in COPD-only (p<0.01). Even less opportunistic organisms like Haemophilus influenzae often chronically infect COPD-bronchiectasis airways (in one study, H. influenzae was the most common isolate, in 54.5% of cases). Chronic infection with such organisms perpetuates airway injury: Pseudomonas in particular is associated with more frequent exacerbations and faster lung function decline in COPD. Our cohort's high neutrophil counts and low albumin (discussed later) likely reflect this burden of infection/inflammation.

The pathophysiological rationale is the classic "vicious cycle" (or vortex) of bronchiectasis, airway colonization by bacteria leads to continual neutrophilic inflammation, which damages the mucociliary apparatus and structural support of the bronchi, which in turn causes mucus stasis and further bacterial growth. In COPD patients, the risk of entering this cycle is elevated due to pre-existing impaired clearance and often a history of acute infections (exacerbations). Pseudomonas and other organisms form biofilms in the bronchiectatic airways, making eradication

difficult and leading to persistent infection despite therapy. Neutrophils, while attempting to fight infection, release proteases (like neutrophil elastase) that destroy elastin and cartilage in the bronchial wall, causing permanent dilation. Thus, chronic infection is both a cause and effect of bronchiectasis. Our findings underscore that the COPD patients who developed bronchiectasis were likely caught in this vicious cycle of infection. From a clinical standpoint, this advocates for actively searching for chronic respiratory infections in COPD patients with worsening symptoms, and for aggressive management (e.g., long-term macrolides, inhaled antibiotics, airway clearance) once bronchiectasis is identified to reduce the bacterial load. [3,12,21,33,34,36,37]

iv) COPD Phenotype: Chronic Bronchitis vs. Emphysema

Our results indicate that the clinical phenotype of COPD plays a role in bronchiectasis risk. We observed a striking predominance of the chronic bronchitis phenotype among COPD patients with bronchiectasis, 67.6% of the overlap group had chronic bronchitic features, compared to only 21.4% of those with COPD alone ($p < 0.001$). Conversely, the emphysema-predominant phenotype was much more common in COPD-only patients (78.6%) than in those with bronchiectasis (32.4%). This suggests that COPD patients characterized by mucus hypersecretion and airway inflammation (i.e. chronic bronchitis) are more prone to developing bronchiectasis than those with pure emphysema in our cohort. This finding correspond with the intuitive expectation that chronic mucus retention in bronchitic COPD can foster recurrent infections, thereby causing bronchiectatic changes. Similarly, Martinez-Garcia et al [2] observed that about 67% of COPD patients with bronchiectasis had chronic bronchitis symptoms, vs 42% of COPD patients without bronchiectasis. The mucus hypersecretion in chronic bronchitis likely contributes to the stagnation of secretions and infection that drive bronchiectasis.

Interestingly, some studies have also implicated emphysema itself as a risk factor for bronchiectasis, highlighting a complex interplay. Dou et al [6] observed bronchiectasis to be significantly more frequent in emphysema-predominant COPD patients (16.5%) compared to those without emphysematous changes (10.3%, p = 0.01). In their analysis, greater emphysema severity (quantified by CT emphysema index) independently predicted bronchiectasis (OR ~1.99). The proposed mechanism is that loss of alveolar attachments in emphysema leads to loss of support for small airways and traction bronchiectasis – essentially, dilated floppy airways due to surrounding tissue destruction. When compared with results our study, one possibility is that in our clinically characterized sample, many of those classified as "emphysema type" did not have as much cough or infection symptoms, and were less likely to be investigated with CT unless other indications arose. Meanwhile, those with chronic bronchitic symptoms and frequent exacerbations were more readily identified as having bronchiectasis. It could also be that emphysema contributes to bronchiectasis in a more subclinical way (detected radiologically), whereas chronic bronchitis leads to more clinical bronchiectasis (symptomatic). In any case, both major COPD phenotypes can be associated with bronchiectasis: chronic bronchitis via mucus plugging and infection, and emphysema via structural mechanics of airway traction and bullous changes. Our data emphasize the former, but we acknowledge that an emphysema-heavy patient is not immune to bronchiectasis – especially if other risk factors like infections or severe airflow limitation are present. [2,4,6-8,35]

v) Smoking History

Cigarette smoking is the primary risk factor for COPD itself, and it also contributes to the pathogenesis of bronchiectasis through its effects on airway defense mechanisms. In our study, surprisingly, the proportion of ever-smokers was not

significantly different between COPD patients with bronchiectasis and those without (73.5% vs 63.4%; p = 0.376). This suggests that simply being a smoker (yes/no) did not distinguish the groups – likely because the vast majority of our COPD patients overall had substantial smoking exposure. However, it is important not to dismiss smoking as a risk factor. It may be that the degree of smoking exposure is what matters. Although we did not record pack-years in every case in our results, other studies have shown that heavier smoking history correlates with development of bronchiectasis in COPD patients. [2,5,6,16,34,36,38]

Sangtam et al [5] found that COPD-bronchiectasis patients had a higher cumulative smoking exposure (mean ~47 pack-years) than COPD-only patients (~35 pack-years; p = 0.03). Dou et al [6] similarly reported that the prevalence of bronchiectasis was greater in heavy smokers (>40 pack-years) compared to light smokers (<20 pack-years). Smoking contributes to bronchiectasis by causing chronic airway inflammation, impairing mucociliary clearance (through cilia damage and mucus hyperplasia), and reducing immune function in the lungs. These effects lead to chronic bronchitis and make the airways more susceptible to persistent infection and structural damage. Indeed, smoke exposure generates oxidative stress that can directly injure airway epithelium and degrade elastin, compounding the structural changes when infections occur. [5,6,34,36,38]

vi) Duration of Disease symptoms

It has been observed in our study that longer duration of COPD symptoms was associated with coexistent bronchiectasis. Patients with COPD-bronchiectasis reported a significantly longer history of respiratory symptoms (9.4 ± 4.5 years) than those with COPD alone (7.1 ± 3.8 years; p = 0.009). This implies that bronchiectasis tends to manifest in patients who have had COPD for many years, reinforcing the idea

that it often develops as a late complication in the natural history of COPD. With more years of disease, patients accumulate more exposures to exacerbations and infections and more progressive lung damage, all of which contribute to bronchiectasis. [7,8,34,36] A systematic review by Ni Y etal [38] also identified longer symptom duration as a significant risk factor for bronchiectasis in COPD, in addition to factors like exacerbations and hospitalization history.

It Is important to note that while longer COPD duration increases the chance to develop bronchiectasis, the age of the patient per se was not a differentiating factor in our study. The mean age of COPD patients with bronchiectasis (67.1 years) was virtually the same as those without (67.3 years; p = 0.927). This suggests it's not chronological age but rather "disease age" how long one has had active COPD symptoms that matters. Many of our patients were of similar age, but those who had earlier onset of COPD or more continuous symptoms over the years accumulated more damage. This distinction highlights that bronchiectasis in COPD is often a consequence of cumulative disease burden over time, rather than just getting older. From a clinical perspective, a COPD patient with a long-standing history (e.g. decades of chronic bronchitis) who now has worsening sputum production or recurrent infections should raise suspicion for bronchiectasis. Early imaging in such long-duration cases might be helpful in diagnosing bronchiectasis at an earlier stage, potentially allowing interventions to slow its progression. [2,7,8,33,34,36]

vii) Nutritional Status (Low BMI and Albumin)

Malnutrition and low body mass index (BMI) were strongly associated with the presence of bronchiectasis in our COPD patients. We found that the bronchiectasis group had a significantly lower BMI (21.5 ± 4.3 kg/m²) compared to the COPD-only group (24.1 ± 3.7; p = 0.003). In fact, 23.5% of the COPD-bronchiectasis patients in

our study were underweight (BMI < 18.5) versus only 7.1% of COPD-only patients. Additionally, serum albumin, a marker of nutritional status and systemic inflammation, was lower in the bronchiectasis group (3.32 ± 0.84 g/dL vs 3.96 ± 1.38 g/dL; p = 0.011). These findings were similar to those from other studies. Gatheral et al [7] observed that COPD-bronchiectasis patients had a mean BMI about 3 units lower than COPD patients (21.5 vs 24.3, p < 0.01). A meta-analysis by Ni et al [38] observed that a BMI < 18.5 was associated with nearly 2-fold higher risk of bronchiectasis in COPD (OR ~1.9, p = 0.002). Low BMI in COPD is often a sign of systemic disease severity. Our data indicate that those with coexisting bronchiectasis are often the sickest and most catabolic COPD patients.

The mechanisms linking poor nutritional status to bronchiectasis are multifaceted. Low BMI may simply be a proxy indicator for more severe COPD (and frequent illness), which itself predisposes to bronchiectasis. Additionally, malnutrition can directly impair immune defenses – for example, deficiencies in protein and micronutrients can weaken the respiratory epithelium's barrier function and reduce the activity of immune cells. In our bronchiectasis patients, the combination of low BMI, low albumin, and high WBC suggests a state of ongoing inflammation and catabolism. This state could predispose to a downward spiral, inflammation causes weight loss and immune dysfunction, which leads to more infection and inflammation. Thus, BMI can serve as a simple clinical indicator: a COPD patient who is losing weight or has a low BMI should prompt consideration of complicating factors like bronchiectasis or other comorbidities that might be driving a high inflammatory state. [34-38, 40]

viii) Comorbidities and Other Factors

An interesting finding in our study was that COPD patients with bronchiectasis tended to have more chronic comorbid conditions. Specifically, we observed higher prevalence of hypertension (61.8% vs 39.3%), Type 2 diabetes (50.0% vs 25.9%), and ischemic heart disease (35.3% vs 13.4%) in the bronchiectasis group, compared to COPD-only patients. While our study was not primarily designed to assess comorbidities, these differences suggest that the overlap syndrome may be associated with a greater systemic disease burden.

Finally, environmental and genetic factors can play a role. Many of our patients were farmers by occupation (in both groups), indicating biomass smoke exposure or dust could be a contributing factor to COPD and possibly to airway damage leading to bronchiectasis. While our data did not show a clear occupational risk signal (the distribution of farmers, laborers, etc. was similar overall). Genetic predispositions like alpha-1 antitrypsin deficiency are known to cause early emphysema and have been linked to bronchiectasis as well, although we did not test for this in our cohort and it would be rare in our population. If present, such genetic factors could underlie some cases of severe COPD that progress to bronchiectasis. [34,36,39]

In summary, the presence of bronchiectasis in a COPD patient should prompt enhanced and tailored care. Our study's real-world data provide clinicians with specific red flags (e.g. low BMI, frequent exacerbations) to identify such patients. By intervening on the modifiable risk factors (e.g. preventing exacerbations, treating infections, improving nutrition), we can potentially slow the vicious cycle of decline. The concept of a distinct COPD-bronchiectasis phenotype also raises the question of

whether treatment guidelines should more explicitly address this overlap – something that future clinical trials will need to inform. [2,5-8]

Limitations

While our study provides valuable insights, it is not without limitations that must be acknowledged when interpreting the findings:

Cross-Sectional Design: This study was observational and cross-sectional, meaning we captured a snapshot in time. Thus, we cannot definitively establish causality or temporal sequence. A longitudinal study following COPD patients over time to see who develops bronchiectasis would better clarify cause-effect.

Single-Center, Moderate Sample Size: Our data were derived from a single tertiary care center with 146 COPD patients, of whom 34 had bronchiectasis. The relatively small number of bronchiectasis cases limits the statistical power to detect more subtle differences and may overestimate some associations. Additionally, being a single-center study, our patient population (and local environmental factors) may not be fully representative of all COPD patients.

Selection Bias: There is potential selection bias in which patients received HRCT scans and were included. We included COPD patients and then identified bronchiectasis among them, by screening all or most with HRCT. However, it's possible that patients with more symptoms (e.g. chronic sputum or frequent exacerbations) were more likely to have undergone CT imaging in the first place. If so, the true prevalence of bronchiectasis in an unselected COPD population could differ, and our identified risk factors might be somewhat accentuated due to how patients were selected for evaluation.

Unmeasured Confounders: We did not evaluate some potential risk factors for bronchiectasis. If a number of bronchiectasis cases were due to old TB (undiagnosed), that could confound the relationship with COPD factors. We also did not assess immunological status (Ig levels) or genetic markers (e.g. A1AT levels) which can predispose to bronchiectasis.

No Multivariate Analysis: Our analysis primarily compared characteristics between groups. We did not perform a multivariable regression to identify independent predictors of bronchiectasis within COPD. Multivariate regression was not feasible due to a small sample size, highly correlated variables, and some predictors perfectly distinguishing the groups. These factors limited the model's ability to estimate reliable coefficients. Expanding the dataset and refining variable selection would enhance the analysis.

Possible Reverse Causation: Some risk factors we identified might actually be consequences of bronchiectasis rather than predisposing causes. For instance, the lower BMI could be a result of bronchiectasis-related systemic inflammation and recurrent infections, rather than a cause of bronchiectasis. Similarly, higher exacerbation frequency might be partly an outcome of having bronchiectasis (which predisposes to infections). Because of our study design, we must be cautious in labeling these as "predisposing factors" – they are strongly associated features, and likely part of a cycle of mutual reinforcement. Only a prospective study could fully disentangle this.

Despite these limitations, our study provides a valuable data-driven profile of COPD patients at risk for bronchiectasis. The limitations highlight areas for future research – such as prospective cohort studies, inclusion of broader populations, and interventional trials focusing on this overlap group.

CONCLUSION

This cross-sectional study highlights that approximately one in four COPD patients in the cohort also have bronchiectasis forming a more severe clinical phenotype, the prevalence of bronchiectasis in COPD patients was 23.3%. COPD-bronchiectasis overlap patients experience worse respiratory symptoms, including more frequent exacerbations, greater functional impairment, and higher inflammation compared to those with COPD alone.

Several key risk factors contribute to the development of bronchiectasis in COPD, including severe airflow limitation, frequent exacerbations requiring hospitalization, chronic airway infections (especially with P. aeruginosa), chronic bronchitic phenotype, prolonged COPD duration, malnutrition, and systemic inflammation. These factors likely interact synergistically, creating a vicious cycle that worsens respiratory health. While the study's findings align with existing literature, differences such as the predominance of chronic bronchitis over emphysema in this cohort suggest further investigation is needed.

The study underscores the Importance of early recognition and intervention for bronchiectasis in COPD patients. Clinicians should consider HRCT scans for patients with risk factors like frequent infections or high sputum production, enabling targeted management strategies such as intensive airway clearance and antimicrobial therapy. Addressing modifiable risk factors, including vaccination, smoking cessation, and nutritional support, could also help prevent disease progression. Additionally, further research, including prospective studies and clinical trials, is essential to establish optimal management approaches for COPD-bronchiectasis overlap patients.

By improving early diagnosis and comprehensive care, clinicians can potentially reduce exacerbations, enhance quality of life, and improve survival outcomes in this high-risk population.

SUMMARY

- The study included a total of 146 patients with COPD.
- Among them, 112 patients (76.7%) had COPD only, while 34 patients (23.3%) had COPD with coexisting bronchiectasis.
- The mean age was similar between the groups: 67.3 years in the COPD-only group and 67.1 years in the COPD-bronchiectasis group, with no significant difference.
- A higher proportion of males was observed in the bronchiectasis group (91.2%) compared to the COPD-only group (76.8%), though this difference was not statistically significant.
- Patients with bronchiectasis had a significantly lower BMI (21.5 vs. 24.1, p=0.003).
- Serum albumin levels were also significantly lower in the bronchiectasis group (3.32 vs. 3.96, p=0.011), indicating malnutrition and systemic inflammation.
- Pulmonary function tests showed more severe airflow obstruction in the bronchiectasis group.
- FEV1 was significantly lower in the bronchiectasis group (54.5% vs. 59.4%, p=0.026).
- FVC was also lower in the bronchiectasis group (77.7% vs. 86.3%, p=0.008).
- PEFR was significantly reduced in the bronchiectasis group (42.4% vs. 49.9%, p=0.018).

- The MMRC dyspnea score was higher in patients with bronchiectasis (2.4 vs. 1.7, $p<0.001$), indicating greater breathlessness.
- The CAT score was also significantly worse in the bronchiectasis group (18.9 vs. 14.9, $p<0.001$), reflecting a higher symptom burden.
- Exercise capacity, measured by the six-minute walk distance, was significantly lower in the bronchiectasis group (307.6m vs. 382.7m, $p=0.001$).
- Patients with bronchiectasis experienced more frequent exacerbations (61.8% vs. 3.6%, $p<0.001$).
- They also had more hospitalizations over two years (3.1 vs. 1.3, $p<0.001$).
- The prevalence of hypertension was higher in the bronchiectasis group (61.8% vs. 39.3%, $p=0.035$).
- Diabetes was also more common among bronchiectasis patients (50% vs. 25.9%, $p=0.015$).
- Heart disease was significantly more prevalent in the bronchiectasis group (35.3% vs. 13.4%, $p=0.009$).
- Bronchiectasis was Higher in the chronic bronchitis phenotype (67.6%) as compared to emphysema phenotype.
- A higher infection burden was noted in the bronchiectasis group, with Pseudomonas detected in 11.76% of cases compared to none in the COPD-only group ($p=0.018$).

- The most common radiological types of bronchiectasis were cystic (38.2%), traction (32.4%), and cylindrical (23.5%).

- Bilateral disease was more common in bronchiectasis patients (58.8%), with distal airway involvement seen in 70.6% of cases.

- Smoking rates were similar between the groups (73.5% vs. 63.4%, p=0.376).

- The duration of symptoms was significantly longer in the bronchiectasis group (9.4 vs. 7.1 years, p=0.009).

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ANNEXURES

ANNEXURE – I - INFORMED CONSENT FORM

Dear Mr. /Mrs. /Dr. _____, you are kindly requested to enroll yourself in a research study tilted, **"**EVALUVATION OF PREVALENCE AND PREDISPOSING FACTORS FOR BRONCHIECTASIS IN COPD PATIENTS - A CROSS SECTIONAL STUDY**."** being conducted by **Dr. ADITYA V PATIL**, a post graduate student in M.D. RESPIRATORY MEDICINE and the study will be carried out under the direct supervision and guidance of **Dr G.S.GAUDE**, Professor, Department of RESPIRATORY MEDICINE, Jawaharlal Nehru Medical College, Belgaum.

You have been requested to participate in this as you fit into the laid-out criteria for a study 'subject'/ participant.

Your participation in the study is voluntary. During the study you will be subjected to lung function test, routine chest X-ray, HRCT Thorax. Your decision whether or not to participate in the study will not affect your treatment in any form. If you decide to participate you are free to withdraw at any time.

TITLE OF THE STUDY: EVALUVATION OF PREVALENCE AND PREDISPOSING FACTORS FOR BRONCHIECTASIS IN COPD PATIENTS - A CROSS SECTIONAL STUDY.

PURPOSE OF THE STUDY: To study the prevalence of bronchiectasis in Chronic obstructive pulmonary disease (COPD) and the predisposing factors for development of bronchiectasis, if we can know the magnitude of bronchiectasis and predisposing factors associated with Chronic obstructive pulmonary disease patients in developing

bronchieactasis, we can take measures to reduce the frequency of exacerbations and improve the quality of life in these patients.

PROCEDURES INVOLVED: If you agree to enroll yourself in my study, you will be subjected to clinical examination which will involve assessment of your vitals, general physical examination and focussed systemic examination. You will then be subjected to Lung function test, Chest X-ray and HRCT.

RISKS AND BENEFITS:

There are no potential risks involved in this study

Benefits of taking part in this research: By taking part in this study, we can know the prevalence, predisposing factors and magnitude of bronchiectasis on Chronic obstructive pulmonary disease (COPD) patients and we can take measures to reduce the frequency of exacerbations and improve the quality of life in these patients.

VOLUNTARY PARTICIPATION / WITHDRAWAL FROM THE STUDY: Taking part in the study is voluntary. You may choose not to enroll yourself in this study and may choose to leave the study anytime in between.

ALTERNATIVES: Your decision regarding participation in study will not change present or future health care services offered to you at KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum. You would simply be excluded from the study if you wish to, and all your details shall be kept confidential and you will get the routine line of management.

PRIVACY AND CONFIDENTIALITY: All data collected or disclosed by you during the course of participation of study, will be kept fully confidential. If, however during the course it becomes necessary for the progress of the course to disclose the

identity, it would be done so only after your informed & written consent. The only people to know that you are a research subject are members of the research team. No information about you will be disclosed to other without your written permission except: In emergency to protect your rights AND welfare. If required by law.

AUTHORIZATION TO PUBLISH RESULT: The results of the study may be used to publish an article. When the results of research are published or discussed in a conference, no information will be displayed that would disclose your identity. Any information obtained in connection with this study and that can be identified with you will remain confidential.

FINANCIAL INCENTIVES FOR PARTICIPATION: No additional costs shall be incurred upon you for the purpose of this study. It is purely being done with the idea of research and all the cost of study will be borne by the investigator.

COMPENSATION: In the event that you become injured as a result of taking part in this study, treatment will be offered to you at KLES **Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum**, or you will be given information about where to receive medical care. However, no reimbursement, compensation or free medical care will be given.

QUESTIONS/CONTACT DETAILS:

You shall be free to contact the below mentioned name & addresses anytime during the study period for any clarification or help as you may desire for.

PRINCIPAL INVESTIGATOR:

if you have any question or complaints with regard to your right as study participant you may contact **Dr Harsha Hegde,** Chairperson, Ethical committee of JNMC, 0831-2473777 Extension 405

CONSENT STATEMENT

I am making a voluntary decision to participate in the study **"EVALUATION OF PREVALENCE AND PREDISPOSING FACTORS FOR BRONCHIECTASIS IN COPD PATIENTS - A CROSS SECTIONAL STUDY."**

My signature below indicates that I have decided to participate and I have read the information provided above or the information provided above has been read to me in the language that I understand best. I was given the opportunity to ask questions and that they have been answered to my satisfaction.

Name of the participant:

Signature or left thumb impression of the participant:

Name of the witness:

Signature or left thumb impression of the witness:

Name of the investigator:

Signature of the investigator:

ANNEXURE II

PERFORMA

CASE NO		IP/OP NO	
NAME			
AGE/SEX OCCUPATION		ADDRESS	

Complaints and Case history		
Past history		
Previous hospitalization in the last 2years due to COPD exacerbations		
Family history		
Personal history • ALCOHOLISM • SMOKING • COMORBIDITIES		
Treatment history		
Vitals :		
Temperature		
Pulse		
Respiratory rate		
Blood pressure		
PHYSICAL EXAMINATION:	Yes	No
Pallor		
Icterus		
Cyanosis		
Clubbing		
Lymphadenopathy		
Edema		

ANTHROPOMETRY VALUES HEIGHT	
WEIGHT	
BMI	
SPIROMETRY VALUE PERCENTAGE FEV1	
FVC	
FEV1/FVC	
STAGE OF COPD **(According to GOLD 2023)**	

Investigations **Sputum investigations :** **Serum albumin :** **Others :**	
HRCT findings	
Others Details	

1. MODIFIED MRC DYSPNEA SCALE

MODIFIED MRC DYSPNEA SCALE[a]

PLEASE TICK IN THE BOX THAT APPLIES TO YOU | ONE BOX ONLY | Grades 0 - 4

mMRC Grade 0.	I only get breathless with strenuous exercise.	☐
mMRC Grade 1.	I get short of breath when hurrying on the level or walking up a slight hill.	☐
mMRC Grade 2.	I walk slower than people of the same age on the level because of breathlessness, or I have to stop for breath when walking on my own pace on the level.	☐
mMRC Grade 3.	I stop for breath after walking about 100 meters or after a few minutes on the level.	☐
mMRC Grade 4.	I am too breathless to leave the house or I am breathless when dressing or undressing.	☐

[a] Fletcher CM. BMJ 1960; 2: 1662.

2. CAT SCORE

Your name:_____

Today's date:_____

How is your COPD? Take the COPD Assessment Test™ (CAT)

This questionnaire will help you and your healthcare professional to measure the impact that COPD (Chronic Obstructive Pulmonary Disease) is having on your wellbeing and daily life. Your answers and test score can be used by you and your healthcare professional to help improve the management of your COPD and gain the greatest benefit from the treatment.

For each item below, place a mark (X) in the box that best describes your current situation. Please ensure that you only select one response for each question.

Example: I am very happy [0] [X] [2] [3] [4] [5] I am very sad

			SCORE
I never cough	0 1 2 3 4 5	I cough all the time	☐
I have no phlegm (mucus) on my chest at all	0 1 2 3 4 5	My chest is full of phlegm (mucus)	☐
My chest does not feel tight at all	0 1 2 3 4 5	My chest feels very tight	☐
When I walk up a hill or a flight of stairs I am not out of breath	0 1 2 3 4 5	When I walk up a hill or a flight of stairs I am completely out of breath	☐
I am not limited to doing any activities at home	0 1 2 3 4 5	I am completely limited to doing all activities at home	☐
I am confident leaving my home despite my lung condition	0 1 2 3 4 5	I am not confident leaving my home at all because of my lung condition	☐
I sleep soundly	0 1 2 3 4 5	I do not sleep soundly because of my lung condition	☐
I have lots of energy	0 1 2 3 4 5	I have no energy at all	☐

TOTAL SCORE ☐☐

3. BRONCHIECTASIS HEALTH QUESTIONNAIRE

1

Bronchiectasis Health Questionnaire (BHQ)

This questionnaire is designed to assess how bronchiectasis affects your life. Please read each question carefully and answer by SELECTING the response that best applies to you. It is important that you answer all questions as honestly as you can.

Participant ID				

Date							
D	D	M	M	Y	Y	Y	Y

Visit Number

Participant ID				

2

1. In the last 2 weeks, I have been tired.

1. All of the time ☐
2. Most of the time ☐
3. A good bit of the time ☐
4. Some of the time ☐
5. A little of the time ☐
6. Hardly any of the time ☐
7. None of the time ☐

2. In the last 2 weeks, I have been much slower at doing things than other people of my age.

1. All of the time ☐
2. Most of the time ☐
3. A good bit of the time ☐
4. Some of the time ☐
5. A little of the time ☐
6. Hardly any of the time ☐
7. None of the time ☐

3. In the last 2 weeks, I have felt anxious.

1. All of the time ☐
2. Most of the time ☐
3. A good bit of the time ☐
4. Some of the time ☐
5. A little of the time ☐
6. Hardly any of the time ☐
7. None of the time ☐

Participant ID					
					3

4. In the last 2 weeks, my chest has felt clear.

1. All of the time ☐
2. Most of the time ☐
3. A good bit of the time ☐
4. Some of the time ☐
5. A little of the time ☐
6. Hardly any of the time ☐
7. None of the time ☐

5. In the last 2 weeks, I have been embarrassed because of my phlegm (sputum).

1. All of the time ☐
2. Most of the time ☐
3. A good bit of the time ☐
4. Some of the time ☐
5. A little of the time ☐
6. Hardly any of the time ☐
7. None of the time ☐

6. In the last 2 weeks, I have felt short of breath.

1. All of the time ☐
2. Most of the time ☐
3. A good bit of the time ☐
4. Some of the time ☐
5. A little of the time ☐
6. Hardly any of the time ☐
7. None of the time ☐

Participant ID					

4

7. In the last 2 weeks, my sleep has been disrupted because of my bronchiectasis.

1. Every night ☐
2. Most nights ☐
3. Several nights ☐
4. Some nights ☐
5. Occasionally ☐
6. Rarely ☐
7. Never ☐

8. In the last 2 weeks, I have had coughing bouts.

1. Every day ☐
2. Most days ☐
3. Several days ☐
4. Some days ☐
5. Occasionally ☐
6. Rarely ☐
7. Never ☐

9. In the last 2 weeks, my phlegm (sputum) contained blood.

1. Every time ☐
2. Most times ☐
3. Several times ☐
4. Sometimes ☐
5. Occasionally ☐
6. Rarely ☐
7. Never ☐

Participant ID					

5

10. In the last <u>one year</u>, I have taken courses of antibiotics for a chest infection.

1. More than five times ☐
2. Five times ☐
3. Four times ☐
4. Three times ☐
5. Twice ☐
6. Once ☐
7. None ☐

Thank you for completing this questionnaire!

ANNEXURE III: PHOTOGRAPHS

Photograph 1 : Spirometer

Photograph 2 : 128-slice CT scanner (General Electronies (GE) Revolution, single-tube machine) used for the study

ANNEXURE IV: MASTER CHART

S. No	Patient name	Age	gender M = (Male, F= Female)	occupation	height IN CM	weight IN KG	BMI	comorbidities	family history	Smoking history	Symtoms since (in years)	physical examination	Pulse Rate	BP (MMHG)	Spo2 %	RS	chest xray	GOLD GRADE	predominant: Chronic Bronchitis (CB)/ Emphysema(E)	FEV1	FVC	FEV1/FVC	FEF25-75%	PEFR	6MWD	CAT SCORE	Previous Admission in last 2 years due to exacerbations	GOLD ABE	MMRC GRADE	HB %	TC	Neutrophils %	Albumin	ORGANISM ISOLATED IN SPUTUM	HRCT THORAX - EMPHYSEMATOUS CHANGES (COPD)	BRONCHIECTASIS changes in hrct	Type of bronchiectasis	BHQ - SUBJECTS WITH BRONCHIECTASIS	Extent of bronchiectasis	Unilateral/Bilateral bronchiectasis	Proximal/Distal bronchiectasis	PAH in hrct	Pleural effusion in hrct	OTHER FINDINGS in hrct
1	PIRAJI PATIL	72	M	FARMER	162	57	21.71925011	NIL	NAD	NIL	15	WNL	78	130/80	95%	NVBS WITH SCATTERED CREPTS	COPD CHANGES	2	CB	57.6	72.5	0.69	50	22.3	220	24	5	E	3	16.7	8.9	76	3.5	KLEBSIELLA OXYTOCA	PRESENT	PRESENT	CYSTIC	32	RIGHT UPPER LOBE, RIGHT MIDDLE LOBE, RIGHT LOWER LOBE	BILATERAL	DISTAL	NIL	NIL	EMPHYSEMATOUS BULLAE PRESENT
2	MAHADEVAPPA RAYAR	63	M	FARMER	164	72	26.76977989	HTN, DM,IHD	NAD	PRESENT	5	BARREL SHAPED CHEST	86	140/90	95%	RHONCHI	COPD CHANGES	3	E	45.76	65.37	0.7	38	40	330	12	3	E	2	12.8	12.2	68	4.2	NIL	PRESENT	ABSENT								
3	SIDDALINGAPPA GURANNAVAR	66	M	SHOP KEEPER	165	71	26.07897153	HTN	NAD	PRESENT	5	WNL	78	130/80	96	NVBS	COPD CHANGES	3	E	45.76	65.37	0.7	24	28	220	12	2	B	2	11.7	5.4	12	5.4	NIL	PRESENT	ABSENT								
4	MAINUDDIN KHADRISAB	71	M	FARMER	164	86	31.97501487	DM	COPD	PRESENT	4	BARREL SHAPED CHEST	90	130/90	94	NVBS	NORMAL	2	E	65.12	93.03	0.7	52	40	321	12	2	B	1	10.9	5.5	16	4.2	NIL	PRESENT	ABSENT								
5	FAKIRGOUDA PATIL	70	M	FARMER	162	69	26.29172382	NIL	NAD	PRESENT	8	WNL	74	120/70	96	NVBS	NORMAL	3	E	39.6	59.84	0.66	72	75	228	10	2	B	2	9.2	6.8	38	3.8	NIL	PRESENT	ABSENT								
6	MALLAPPA KALASANNAVAR	78	M	TRADER	170	58	20.06920415	HTN, DM	NAD	PRESENT	12	WNL	94	130/90	94	RHONCHI	NORMAL	3	CB	42.24	60.34	0.7	52	42	221	23	4	E	3	12	7.8	76	4	ENTEROCOCCUS FAECALIS	PRESENT	PRESENT	TRACTION	30	RIGHT LOWER LOBE, LEFT MIDDLE LOBE, LEFT LOWER LOBE	BILATERAL	DISTAL	NIL	NIL	NIL
7	LAXMI KOLLANATTI	42	F	HOUSE WIFE	150	70	31.11111111	HTN	NAD	NIL	2	WNL	70	120/70	96	NVBS	NORMAL	2	E	63.36	90.5	0.7	66	52	332	18	1	A	2	12.5	5.6	42	3.8	NIL	PRESENT	ABSENT								
8	IRAPPA TALWAR	73	M	DAILY WAGE WORKR	158	48	19.22768787	NIL	NAD	PRESENT	5	WNL	68	130/90	96	NVBS	NORMAL	4	CB	20.24	36.96	0.55	25	23	208	18	2	B	2	11.2	12.5	65	3.6	COMMENSALS	PRESENT	ABSENT								
9	BALACHANDRA DASAKA	65	M	SECURITY GUARD	163	52	19.57168128	HTN,IHD	NAD	PRESENT	12	WNL	86	140/80	96	NVBS	NORMAL	4	CB	25.52	44.88	0.57	13	42	230	13	2	B	1	12.5	9.8	25	3.9	NIL	PRESENT	ABSENT								
10	SHAKUNTALA KHALATE	62	F	HOUSE WIFE	158	74	29.64268547	HTN, IHD	NAD	NIL	10	WNL	76	110/70	96	NVBS	COPD CHANGES	3	E	45.76	65.37	0.7	24	28	318	18	2	B	2	9.8	6.5	40	4.6	NIL	PRESENT	ABSENT								
11	SHIVAJI PATIL	63	M	FARMER	154	42	17.70956316	HTN,T2DM,IHD	NAD	PRESENT	12	WNL	77	130/90	96	NVBS WITH SCATTERED CREPTS	NORMAL	3	CB	45.76	65.37	0.7	24	28	220	24	4	E	3	12.9	14.6	77	3.2	COMMENSALS	PRESENT	PRESENT	CYLINDRICAL	24	RIGHT UPPER LOBE, RIGHT LOWER LOBE, LEFT LOWER LOBE	BILATERAL	DISTAL	NIL	NIL	NIL
12	NIRMALA	65	F	HOUSE WIFE	156	56	23.01117686	DM	NAD	NIL	10	WNL	88	120/70	96	NVBS	NORMAL	3	E	44	70.4	0.63	33	40	441	24	2	B	3	10.2	7.2	34	3.8	NIL	PRESENT	ABSENT								
13	APPASAHEB	74	M	FARMER	166	62	22.4996371	HTN	ASTHM	PRESENT	15	WNL	90	120/70	96	NVBS	NORMAL	2	E	63.36	90.5	0.7	66	52	400	25	1	A	3	15	6.4	36	3.9	NIL	PRESENT	ABSENT								
14	DURGAPPA	70	M	FARMER	163	56	21.07719523	NIL	NAD	PRESENT	10	WNL	68	130/90	94	NVBS	NORMAL	2	E	52.8	89.6	0.59	39	52	300	13	2	B	1	10.7	6.8	40	3.5	NIL	PRESENT	ABSENT								
15	SUDHA SAWANT	67	F	HOUSE WIFE	154	42	17.70956316	HTN, IHD	NAD	NIL	16	WNL	96	100/60	94	B/L BASAL CREPTS	NORMAL	3	CB	49.28	70.4	0.7	25	27	321	18	2	B	2	11.2	11	52	3.8	ACINETOBACTOR BAUMANNII	PRESENT	PRESENT	CYLINDRICAL	20	RIGHT UPPER LOBE, RIGHT LOWER LOBE, LEFT MIDDLE LOBE, RIGHT LOWER LOBE	BILATERAL	DISTAL	PRESENT	NIL LEFT MINIMAL EFFUSION	CARDIOMEGALY
16	ANANDI	57	F	HOUSE WIFE	142	45	22.3170006	NIL	NAD	NIL	2	WNL	70	130/90	94	NVBS	NORMAL	2	E	52.8	75.43	0.5	28	32	330	18	1	A	2	11.2	2.6	55	3.9	NIL	PRESENT	ABSENT								
17	PARVATEVVA	86	F	HOUSE WIFE	148	54	24.65303141	HTN,DM,IHD	NAD	NIL	10	WNL	98	110/70	95	NVBS	NORMAL	2	E	70.4	100.57	0.7	20	22	322	12	2	A	1	9.8	5.4	21	4	NIL	PRESENT	ABSENT								
18	HANUMAPPA	72	M	LABOURER	147	45	20.82465639	HTN,DM	NAD	PRESENT	10	WNL	72	120/70	96	NVBS	NORMAL	4	E	29.04	41.49	0.7	15	6	209	12	3	C	1	9.7	8.6	54	3.5	COMMENSALS	PRESENT	ABSENT								
19	SHASHIKALA	56	F	FARMER	168	56	19.84126984	HTN	NAD	NIL	2	WNL	68	140/80	96	NVBS	NORMAL	2	CB	52.8	75.43	0.7	36	40	393	24	3	B	3	10.2	6.5	45	2.9	NIL	PRESENT	ABSENT								
20	NOORAHEMAD	58	M	FARMER	170	60	20.76124567	DM	NAD	PRESENT	1	BARREL SHAPED	74	130/90	96	NVBS	COPD CHANGES	3	CB	45.76	65.37	0.7	38	40	330	12	2	B	1	16.8	11.5	70	2.8	COMMENSALS	PRESENT	ABSENT								
21	SIDDAGOUDA N	72	M	FARMER	168	55	19.48696145	HTN	NAD	NIL	6	WNL	88	110/70	94	NVBS	COPD CHANGES	3	CB	39.6	56.57	0.7	22	27	280	18	1	B	3	12.8	6.8	43	4.2	NIL	PRESENT	ABSENT								
22	SIDDALINGAPPA	68	M	FARMER	157	40	16.22783886	NIL	ASTHM	PRESENT	4	WNL	76	120/70	96	NVBS	NORMAL	2	CB	54.56	77.94	0.7	52	43	250	18	0	A	2	8.6	12.5	60	2.1	NIL	PRESENT	ABSENT								
23	SIDDAGOUDA NIRWANI	67	M	FARMER	158	49	19.6282647	NIL	NAD	PRESENT	7	WNL	77	110/70	99	B/L BASAL CREPTS	NORMAL	3	CB	46	66	0.69	28	38	311	24	3	E	3	12.5	5.4	56	3.1	COMMENSALS	PRESENT	PRESENT	CYLINDRICAL	25	RIGHT MIDDLE LOBE, RIGHT LOWER LOBE	UNILATERAL	DISTAL	NIL	LEFT SIDED PE	NIL
24	PRAKASH	54	M	KOOLI	159	58	22.94213045	NIL	NAD	PRESENT	3	WNL	78	110/70	98	NVBS	NORMAL	3	E	45	59	0.69	34	28	260	13	1	B	2	11.2	12.4	60	5.2	NIL	PRESENT	ABSENT								
25	BASAVARAJ	48	M	DRIVER	156	66	27.12031558	NIL	NAD	NIL	2	WNL	68	140/80	96	NVBS	NORMAL	2	E	65.12	93.03	0.7	42	40	303	12	1	A	0	14	11.2	48	3.2	NOG	PRESENT	ABSENT								
26	SHAKUNTALA	80	F	HOUSE WIFE	157	47	19.06771066	HTN	NAD	NIL	10	WNL	69	110/70	96	NVBS	COPD CHANGES	2	CB	69.52	99.31	0.7	56	59	392	18	1	A	2	12.5	3.2	56	3.8	NIL	PRESENT	ABSENT								
27	HUSSAINBAI	82	F	HOUSE WIFE	158	56	22.43230252	DM	NAD	NIL	12	WNL	70	120/70	94	NVBS	NORMAL	2	E	65.12	103.04	0.63	40	59	398	16	1	A	2	13.5	10	60	4.2	NIL	PRESENT	ABSENT								
28	KAMAPPA	75	M	LABOURER	170	70	24.22145329	HTN	NAD	PRESENT	10	WNL	110	130/70	96	NVBS	NORMAL	2	E	65.12	95.76	0.68	31	48	401	18	2	A	3	16.7	12.6	68	2.8	NIL	PRESENT	ABSENT								

29	SHIVALINGAYYA KOLLURMATH	66	M	FARMER	162	60	22.86236854	DM	NAD	PRESENT	7	BARREL SHAPED	72	140/80	94	NVBS	NORMAL	2	E	58.08	100.8	0.58	72	40	410	20	3	B	2	11.7	4.8	32	3.8	NOG	PRESENT	ABSENT								
30	SHOBHA JAKATI	61	F	HOUSE WIFE	143	39	19.07183725	NIL	NAD	NIL	5	WNL	90	110/70	94	NVBS	NORMAL	2	CB	54.56	80.24	0.68	52	43	389	16	3	B	2	16	12	45	3.6	NIL	PRESENT	ABSENT								
31	VIRABHADRAPPA H	84	M	LAND LOARD	165	60	22.03856749	NIL	NAD	PRESENT	15	WNL	74	120/70	96	NVBS	COPD CHANGES	2	CB	56.32	82.82	0.68	47	88	408	14	2	B	2	9.8	5.6	46	3.5	NIL	PRESENT	ABSENT								
32	SAHADEV PEJOLLI	56	M	INDUSTRIAL WORKER	157	49	19.8791026	HTN, DM	NAD	PRESENT	4	WNL	86	110/80	98	NVBS	NORMAL	2	CB	65.12	93.03	1	66	50	309	18	3	E	2	17	14.5	96	3.2	KLEBSIELLA OXYTOCA	PRESENT	PRESENT	TRACTION	34	LEFT UPPER LOBE, RIGHT UPPER LOBE, RIGHT MIDDLE LOBE, RIGHT LOWER LOBE	BILATERAL	DISTAL	NIL	NIL	NIL
33	RAYAPPA KITTUR	74	M	SHOPKEEPER	164	68	25.2825699	HTN, IHD	NAD	PRESENT	10	WNL	76	110/70	96	NVBS	NORMAL	2	E	73.04	104.34	0.7	48	39	409	8	0	A	0	12.6	5.8	68	5.4	NIL	PRESENT	ABSENT								
34	SHIVANAND	72	M	LABOURER	160	78	30.46875	HTN	NAD	PRESENT	12	WNL	98	110/70	94	NVBS	COPD CHANGES	2	E	77.44	123.2	0.63	50	68	392	10	0	A	1	14	6.4	65	4.2	NIL	PRESENT	ABSENT								
35	AMANULLA	65	M	FARMER	163	52	19.57168128	NIL	COPD	PRESENT	6	WNL	78	120/70	94	NVBS	NORMAL	2	E	75.68	111.29	0.68	72	75	399	8	1	A	0	12	5.6	60	2.8	NIL	PRESENT	ABSENT								
36	MALLAVVA	65	F	HOUSE WIFE	154	80	33.73250126	HTN	NAD	NIL	8	WNL	98	110/70	97	NVBS	NORMAL	2	E	74.8	106.86	0.7	55	60	445	12	0	A	2	10.2	5.5	64	3.6	NIL	PRESENT	ABSENT								
37	NAGAIAH SUBAIAH	48	M	COOK	155	57	23.72528616	NIL	NAD	PRESENT	2	WNL	78	110/80	96	NVBS	COPD CHANGES	2	E	70.4	100.57	0.7	42	66	349	14	3	E	1	11	4.5	52	4.5	NIL	PRESENT	ABSENT								
38	GANGUBAI	70	F	HOUSE WIFE	147	51	23.60127725	HTN	NAD	NIL	12	WNL	68	140/80	99	NVBS	NORMAL	3	E	46	66	0.69	28	38	270	16	3	B	3	11.6	8.6	70	3.2	NIL	PRESENT	ABSENT								
39	ABDULRAZAQ PHANIBAND	68	M	SHOP KEEPER	158	48	19.22768787	IHD, CKD	NAD	PRESENT	12	WNL	58	110/80	96	NVBS	NORMAL	3	CB	39.6	59.4	0.66	17	28	228	18	3	B	3	7.8	6.8	50	5.6	COMMENSALS	PRESENT	PRESENT	TRACTION	34	RIGHT LOWER LOBE	UNILATERAL	DISTAL	NIL	LEFT MILD PLEURAL EFFUSION	NIL
40	RUKMINI	72	F	HOUSE WIFE	165	70	25.71166208	HTN	NAD	NIL	16	WNL	86	110/70	98	NVBS	NORMAL	2	E	58	74	0.6	24	28	356	20	2	B	2	8.6	12.5	68	5.4	COAGULASE NEGATIVE STREPTOCOCCI	PRESENT	ABSENT								
41	RAYAPPA	72	M	EX MILITARY	155	66	27.47138398	NIL	NAD	PRESENT	12	WNL	98	140/80	96	NVBS	NORMAL	2	E	66.88	98.35	0.68	54	68	384	10	2	B	0	12.6	11	93	4.8	NOG	PRESENT	ABSENT								
42	LAXMAPPA KARIGAR	81	M	FARMER	164	52	19.33372992	HTN, CKD	NAD	NIL	20	WNL	94	150/100	95	NVBS	COPD CHANGES	3	CB	46	66	0.69	28	38	300	16	3	E	2	11.5	11.2	96	3	COAGULASE NEGATIVE STREPTOCOCCI	PRESENT	PRESENT	CYSTIC	35	RIGHT MIDDLE LOBE, RIGHT LOWER LOBE	UNILATERAL	DISTAL	NIL	NIL	NIL
43	LAKKAPPA	58	M	FARMER	160	71	27.734375	NIL	NAD	PRESENT	14	WNL	94	120/70	95	NVBS	COPD CHANGES	2	E	69.52	102.24	0.68	56	59	288	13	1	A	2	14	3.6	54	5.2	NIL	PRESENT	ABSENT								
44	BHARAMGOUDA	83	M	LABOURER	169	70	24.50894577	NIL	NAD	PRESENT	12	WNL	76	110/70	96	NVBS	NORMAL	2	E	77.44	113.88	0.7	77	80	338	12	1	A	2	13.6	5.6	56	5	NIL	PRESENT	ABSENT								
45	MAHADEVI	58	F	HOUSE WIFE	160	56	21.875	NIL	NAD	NIL	4	WNL	88	110/70	96	NVBS	NORMAL	3	E	42.26	69.44	0.61	52	42	401	10	1	B	0	11.8	4.2	63	4.8	NIL	PRESENT	ABSENT								
46	FAKKIRGOUDA	70	M	FARMER	147	55	25.45235781	HTN,DM	NAD	PRESENT	9	WNL	86	110/70	96	NVBS	NORMAL	3	E	39.6	56.57	0.7	22	27	356	17	2	B	2	12.8	6.4	60	4.2	NOG	PRESENT	ABSENT								
47	VAHIA	55	M	HOUSE WIFE	157	60	24.34175829	HTN,DM,IHD	NAD	PRESENT	4	WNL	84	130/80	95	NVBS	NORMAL	2	E	54.56	77.94	0.7	52	43	375	12	2	B	1	11.2	8.9	70	5.2	NIL	PRESENT	ABSENT								
48	APPANNA	65	M	BANK MANAGER	168	50	17.7154195	NIL	NAD	NIL	8	WNL	82	140/80	98	NVBS	NORMAL	2	CB	56.32	80.46	0.7	47	88	358	14	0	A	2	15.7	15.2	93	2.8	KLEBSIELLA PNEUMONIA	PRESENT	ABSENT								
49	RAMESH	59	M	COOK	170	74	25.60553633	NIL	NAD	PRESENT	6	WNL	99	120/70	96	NVBS	NORMAL	2	E	70.4	100.57	0.7	42	66	349	10	0	A	1	12.5	5.4	50	4.3	NIL	PRESENT	ABSENT								
50	PRAKASH	60	M	LABOURER	158	70	28.04037814	NIL	NAD	PRESENT	4	WNL	94	110/70	94	NVBS	NORMAL	3	E	43	62	0.68	24	36	278	18	2	B	3	14	8.6	60	2.6	NIL	PRESENT	ABSENT								
51	BASAPPA RAJAPURI	66	M	SUGAR FACTORY	169	70	24.50894577	T2DM,HTN,IHD	NAD	PRESENT	8	WNL	98	110/70	94	NVBS	COPD CHANGES	2	E	50.16	84.48	0.59	32	72	309	20	2	B	2	9.8	15.6	86	5.6	NOG	PRESENT	PRESENT	CYSTIC	36	RIGHT LOWER LOBE	UNILATERAL	DISTAL	NIL	NIL	CARDIOMEGALY
52	HEMANTH	56	M	FARMER	160	47	18.359375	NIL	NAD	PRESENT	4	WNL	84	130/80	95	NVBS	COPD CHANGES	2	CB	51.04	84.48	0.6	62	66	289	12	2	B	1	10	3.2	63	2.8	NIL	PRESENT	ABSENT								
53	SIDDAPPA	85	M	FARMER	155	63	26.2226847	HTN,IHD	NAD	PRESENT	12	WNL	86	130/80	64	NVBS	COPD CHANGES	2	E	65.12	93.03	0.7	42	40	346	10	1	A	2	15.2	5.4	56	5.2	NOG	PRESENT	ABSENT								
54	VENKAPPA	94	M	FARMER	163	65	24.4646016	NIL	NAD	NIL	20	WNL	86	120/70	95	NVBS	NORMAL	2	E	69.52	99.31	0.7	56	59	343	12	1	A	2	11.2	5	86	2.5	NIL	PRESENT	ABSENT								
55	CHAMBAPPA	64	M	COOK	168	72	25.51020408	T2DM, HTH	NAD	PRESENT	9	WNL	64	130/80	94	NVBS	NORMAL	2	E	72	88	0.7	50	46	392	8	0	A	1	10	4.8	70	15.5	COMMENSALS	PRESENT	ABSENT								
56	VIMAL	68	F	HOUSE WIFE	153	73	31.18458712	T2DM	NAD	NIL	7	WNL	58	110/70	94	NVBS	NORMAL	3	E	48	66	0.72	66	40	452	18	2	B	2	11.6	6.2	50	5.4	NIL	PRESENT	ABSENT								
57	SADASHIVAGOUDA	63	M	FARMER	160	60	23.4375	NIL	NAD	PRESENT	6	WNL	94	120/70	95	NVBS	NORMAL	2	E	70.4	100.57	0.7	20	22	389	10	0	A	1	16.7	5.4	54	5.2	NIL	PRESENT	ABSENT								
58	BASAPPA	68	M	FARMER	160	50	19.53125	NIL	NAD	PRESENT	5	WNL	110	140/80	95	NVBS	NORMAL	2	CB	65.12	93.03	0.7	58	73	441	16	1	B	2	11.2	4.8	53	4.8	NIL	PRESENT	ABSENT								
59	ADIVEPPA PATTANSHETTI	86	M	FARMER	147	55	25.45235781	T2DM,HTN,IHD	NAD	PRESENT	14	WNL	112	130/80	98	NVBS	NORMAL	3	E	43.12	61.6	0.7	24	36	278	25	3	E	3	10	16.5	92	3.4	STAPH AUREUS	PRESENT	PRESENT	CYLINDRICAL	30	LEFT UPPER LOBE	UNILATERAL	PROXIMAL	PRESENT	LEFT THIN RIM	NIL
60	MALLAVVA	75	F	HOUSE WIFE	157	65	26.37023814	NIL	NAD	NIL	12	WNL	94	120/70	94	NVBS	NORMAL	3	E	48	42	0.7	60	49	286	12	2	A	2	13.1	5	60	3.8	NIL	PRESENT	ABSENT								
61	REVAPPA METAGATTI	62	M	FARMER	158	48	19.22768787	T2DM, HTN, CVA	NAD	PRESENT	16	WNL	96	130/80	96	NVBS	COPD CHANGES	2	CB	54.56	77.94	0.7	54	47	342	10	3	E	1	16	3.8	54	3.6	NIL	PRESENT	PRESENT	TRACTION	40	RIGHT UPPER LOBE, LEFT UPPER LOBE AND LEFT LOWER LOBE	BILATERAL	DISTAL	PRESENT	RT SIDED MILD EFFUSION	NIL
62	LAXMIBAI	74	F	HOUSE WIFE	169	68	23.80869017	NIL	NAD	NIL	9	WNL	84	140/80	96	NVBS	NORMAL	2	E	59.84	85.49	0.7	62	58	278	10	1	A	1	12.6	4	50	4.6	NIL	PRESENT	ABSENT								
63	NAGESH	77	M	CARPENTER	164	58	21.56454491	HTN	NAD	PRESENT	11	WNL	74	130/80	94	NVBS	NORMAL	2	CB	65.12	93.03	0.7	50	40	357	8	0	A	0	15	6.8	85	3.6	NIL	PRESENT	ABSENT								
64	JALALSAB	80	M	FARMER	167	68	24.38237298	HTN	NAD	PRESENT	15	WNL	64	130/80	96	CREPTS	NORMAL	2	E	69.52	99.31	0.7	56	59	285	8	0	A	0	12	7.6	54	3.5	NOG	PRESENT	ABSENT								
65	ADAPPA	68	M	FARMER	157	65	26.37023814	NIL	NAD	PRESENT	12	WNL	58	120/70	95	NVBS	NORMAL	2	E	65.12	93.03	0.7	40	59	410	14	1	B	1	11.7	5.6	60	3.6	NIL	PRESENT	ABSENT								
66	VISHNU PATIL	59	M	FARMER	167	72	25.81663021	T2DM,HTN,IHD	NAD	PRESENT	4	WNL	110	110/70	96	SCATTERED CREPTS	COPD CHANGES	2	E	70	78	0.7	41	48	372	12	3	E	1	14	14.2	93	2.6	ENTEROCOCCUS FAECALIS	PRESENT	PRESENT	CYSTIC	38	RIGHT MIDDLE LOBE, RIGHT LOWER LOBE, LEFT LOWER LOBE	BILATERAL	DISTAL	NIL	NIL	NIL
67	JANUKA	54	F	HOUSE WIFE	170	78	26.98961938	NIL	NAD	NIL	3	WNL	112	110/70	96	NVBS	COPD CHANGES	2	E	58.08	100.8	0.58	72	40	267	18	2	B	2	9.8	6.4	40	4.2	NIL	PRESENT	ABSENT								
68	SHREEPATI	67	M	FARMER	154	44	18.5528757	NIL	NAD	PRESENT	4	WNL	114	140/80	95	NVBS	NORMAL	2	E	60.72	95.2	0.64	38	30	327	16	1	B	2	12	8.6	60	1.8	NOG	PRESENT	ABSENT								
69	Z B DHAMNEKAR	78	F	HOUSE WIFE	154	55	23.19109462	NIL	NAD	NIL	3	WNL	86	130/80	95	NVBS	NORMAL	2	E	70	99	0.7	72	74	244	17	2	B	2	12.1	11.2	86	2.6	COMMENSALS	PRESENT	ABSENT								
70	SANGAPPA	61	M	FARMER	160	66	25.78125	T2DM,HTN	NAD	PRESENT	2	WNL	98	130/80	94	NVBS	COPD CHANGES	2	E	65.12	93.03	0.7	42	40	320	12	1	B	1	9.8	8.6	70	3.6	NIL	PRESENT	ABSENT								
71	REVAPPA	62	M	FARMER	165	55	20.2020202	HTN	NAD	PRESENT	2	WNL	98	130/80	95	NVBS	NORMAL	2	CB	69.52	99.31	0.7	56	59	267	18	1	B	2	12.7	4.2	50	3.5	NIL	PRESENT	ABSENT								
72	SANNAPPA	80	M	FARMER	156	50	20.54569362	NIL	NAD	PRESENT	6	WNL	76	130/80	95	NVBS	NORMAL	2	CB	59.84	85.49	0.7	60	77	220	16	1	B	2	13.4	3.9	60	3.6	NIL	PRESENT	ABSENT								
73	MAHADEV KHANPET	64	M	LAND LOARD	155	52	21.64412071	T2DM	NAD	NIL	7	WNL	86	140/80	96	NVBS	COPD CHANGES	3	CB	45	59	0.69	34	28	342	18	2	B	2	10	12.5	78	3.1	KLEBSIELLA PNEUMONIA	PRESENT	PRESENT	TRACTION	36	RIGHT MIDDLE LOBE	UNILATERAL	PROXIMAL	NIL	NIL	CARDIOMEGALY

74	HANMANTAPPA	69	M	FARMER	163	65	24.4646016	HTN	NAD	PRESENT	6	WNL	96	130/80	98	NVBS	NORMAL	2	E	54.56	80.24	0.68	52	43	363	12	0	A	1	16	5.6	60	4.5	NIL	PRESENT	ABSENT								
75	AJIT PATIL	74	M	FARMER	154	80	33.73250126	NIL	COPD	PRESENT	8	WNL	82	120/70	96		COPD CHANGES	2	E	60	64	0.78	47	88	347	16	2	B	2	15	6.5	50	4.8	NOG	PRESENT	ABSENT								
76	BHAVAKKANNA PATIL	71	M	FARMER	159	66	26.10656224	NIL	NAD	PRESENT	7	WNL	92	110/70	96	NVBS	NORMAL	2	E	68	92	0.7	53	27	365	14	1	A	2	12.8	5.4	60	5.1	NIL	PRESENT	ABSENT								
77	KASTURI	65	F	HOUSE WIFE	160	47	18.359375	T2DM,HTN,IHD	NAD	NIL	6	WNL	84	110/70	94	NVBS	COPD CHANGES	2	CB	66.88	95.54	0.7	38	52	342	18	2	B	2	12.5	5.1	60	2.8	NIL	PRESENT	ABSENT								
78	BASSAPPA	70	M	SUGAR FACTORY	160	78	30.46875	NIL	NAD	PRESENT	8	WNL	76	130/80	94	NVBS	COPD CHANGES	1	E	82	95	0.8	50	30	335	8	0	A	0	16.8	5.2	56	2.9	NIL	PRESENT	ABSENT								
79	SHIVAMMA	68	F	HOUSE WIFE	168	72	25.51020408	HTN,IHD	NAD	NIL	4	WNL	98	130/80	94	NVBS	NORMAL	2	E	64.25	100.28	0.64	50	78	676	10	1	B	1	12.4	6.8	55	3.6	NAD	PRESENT	ABSENT								
80	NAMDEV	70	M	LABOURER	157	60	24.34175829	NIL	NAD	NIL	10	WNL	92	140/80	94	NVBS	NORMAL	2	E	69.34	109.15	0.64	56	77	540	24	1	B	3	11.2	11.2	70	3.8	COMMENSALS	PRESENT	ABSENT								
81	MAINUDDIN	64	M	FARMER	156	66	27.12031558	T2DM, HTH	NAD	PRESENT	7	WNL	90	110/70	96	NVBS	NORMAL	2	E	70.93	106.82	0.66	62	55	597	16	1	A	2	17	10.8	86	5.1	COMMENSALS	PRESENT	ABSENT								
82	MAHADEV KURBAR	61	M	FARMER	166	62	22.4996371	T2DM	NAD	PRESENT	6	WNL	80	130/80	95	NVBS	COPD CHANGES	2	E	66.23	97.9	0.68	46	60	724	14	1	B	1	10	3.8	40	4.5	NIL	PRESENT	ABSENT								
83	MARNAYAK	58	M	FARMER	163	56	21.07719523	NIL	NAD	NIL	4	WNL	74	120/70	95	NVBS	NORMAL	2	CB	65.74	99.98	0.66	60	82	686	16	2	B	2	12.6	6.4	56	4.8	NIL	PRESENT	ABSENT								
84	ASHOK	81	M	FARMER	162	52	19.81405274	HTN	NAD	PRESENT	9	WNL	70	110/60	95	NVBS	COPD CHANGES	2	CB	62.82	96.97	0.65	66	49	310	24	3	E	3	15	8.6	77	2.9	COMMENSALS	PRESENT	PRESENT	CYSTIC	30	RIGHT MIDDLE LOBE, RIGHT LOWER LOBE, LEFT MIDDLE LOBE, LEFT LOWER LOBE	BILATERAL	DISTAL	NIL	NIL	NIL
85	RAJENDRA	62	M	FARMER	147	45	20.82465639	T2DM,HTN,IHD	NAD	NIL	7	WNL	60	140/80	96	NVBS	NORMAL	2	CB	68.52	101.82	0.67	50	75	595	16	1	B	2	12	5.6	46	3.2	NIL	PRESENT	ABSENT								
86	SANGANGOUDA	72	M	FARMER	168	56	19.84126984	NIL	NAD	PRESENT	5	WNL	62	130/80	94	NVBS	COPD CHANGES	2	CB	65.25	98.24	0.66	61	68	484	22	2	B	3	14.5	8.5	60	2.6	NIL	PRESENT	ABSENT								
87	BHIVAJI	69	M	LABOURER	168	55	19.48696145	HTN	NAD	PRESENT	2	WNL	88	130/80	99	NVBS	COPD CHANGES	2	CB	69.16	103.92	0.67	46	78	489	20	1	A	2	16	7.5	40	3.6	NIL	PRESENT	ABSENT								
88	LALSAB	68	M	SHOPKEEPER	157	40	16.22783886	NIL	NAD	PRESENT	6	WNL	86	130/80	96	NVBS	COPD CHANGES	2	CB	68.19	103.54	0.66	38	60	541	18	3	E	2	8.8	6.5	68	1.8	NIL	PRESENT	ABSENT								
89	GULABAHEMAD	75	M	BUISNESS	166	66	23.95122659	NIL	NAD	PRESENT	5	WNL	88	110/70	96	NVBS	NORMAL	2	E	71.68	100	0.72	67	70	514	18	1	A	2	13.2	6.4	64	2.6	NIL	PRESENT	ABSENT								
90	SOMLINGAPPA	77	M	FARMER	159	58	22.94213045	NIL	NAD	PRESENT	4	WNL	88	120/70	98	NVBS	NORMAL	2	E	65.1	92	0.7	62	70	645	10	1	B	0	10	5	60	3.8	NIL	PRESENT	ABSENT								
91	BIBIJAN	65	F	HOUSE WIFE	156	52	21.36752137	HTN,T2DM	NAD	NIL	6	WNL	98	130/80	94	NVBS	COPD CHANGES	2	E	55.43	65	0.67	58	66	244	25	2	B	3	10.2	5.7	66	3.6	NIL	PRESENT	PRESENT	CYSTIC	34	RIGHT LOWER LOBE	UNILATERAL	PROXIMAL	NIL	NIL	NIL
92	KEMPANNA GUNTAKAL	73	M	FARMER	170	70	24.22145329	NIL	NAD	PRESENT	4	WNL	88	140/80	96	NVBS	COPD CHANGES	1	E	81.48	115.85	0.7	58	66	512	20	0	A	2	10.2	7.5	54	4.4	NIL	PRESENT	ABSENT								
93	SIDRAM	75	M	FARMER	162	60	22.86236854	T2DM,HTN,IHD	NAD	PRESENT	5	WNL	86	130/80	98	NVBS	NORMAL	2	E	63.33	107.92	0.59	50	66	544	18	0	B	2	16.5	7.4	55	4.6	NIL	PRESENT	ABSENT								
94	NANJAPPA	65	M	SUGAR FACTORY	165	60	22.03856749	NIL	NAD	NIL	10	WNL	112	130/80	99	NVBS	NORMAL	2	E	70.64	122.41	0.58	47	80	533	20	0	B	3	12.2	10.2	60	4.8	NIL	PRESENT	ABSENT								
95	VIMAL	65	F	HOUSE WIFE	157	76	30.83289383	HTN,IHD	NAD	NIL	12	WNL	86	120/70	100	NVBS	NORMAL	2	E	79.72	100	0.7	60	57	649	16	0	A	2	11.7	10.8	45	5.4	ENTEROCOCCUS FAECALIS	PRESENT	ABSENT								
96	GUNDAPPA BASAPPA	63	M	FARMER	164	68	25.2825699	NIL	NAD	PRESENT	9	WNL	76	140/80	94	NVBS	NORMAL	2	E	78.32	99	0.7	55	67	479	13	2	A	2	10	12.6	78	2.8	PSEUDOMONAS AUREGINOSA	PRESENT	PRESENT	CYLINDRICAL	36	LEFT LOWER LOBE	UNILATERAL	DISTAL	NIL	RT MILD EFFUSION	NIL
97	RAMU	58	M	LABOURER	160	78	30.46875	T2DM, HTH	COPD	PRESENT	4	WNL	96	130/80	99	NVBS	COPD CHANGES	2	E	53.65	53	0.67	62	70	531	8	1	B	0	9.8	11.6	70	3.4	NIL	PRESENT	ABSENT								
98	SHIVAGOUDA	80	M	FARMER	163	52	19.57168128	T2DM	NAD	PRESENT	15	WNL	74	130/80	90	NVBS	COPD CHANGES	2	CB	65.12	93.03	0.7	55	62	414	12	1	A	2	10.2	6.5	55	3.4	ENTEROCOCCUS FAECALIS	PRESENT	ABSENT								
99	SUNIL PATIL	48	M	SHOPKEEPER	155	57	23.72528616	NIL	NAD	PRESENT	2	WNL	90	140/80	96	NVBS	NORMAL	1	E	81.57	98	0.7	55	62	576	14	2	B	2	12.5	6.4	50	3.4	NIL	PRESENT	ABSENT								
100	NINGAVVAWWA	62	F	COTTON FACTORY WORKER	160	61	23.828125	NIL	NAD	NIL	4	WNL	86	110/70	98	NVBS	NORMAL	2	E	62	71	0.7	49	52	565	14	1	A	1	11.2	6.6	56	4.6	NIL	PRESENT	ABSENT								
101	UMESH BHAJANTRI	47	M	BANK EMPLOYEE	146	73	34.24657534	T2DM,HTN,IHD	NAD	PRESENT	6	WNL	80	110/70	94	NVBS	NORMAL	3	E	45	59	0.69	34	28	260	18	4	E	3	11.2	12.5	80	3	COMMESNSALS	PRESENT	PRESENT	CYSTIC	32	RIGHT UPPER LOBE, RIGHT MIDDLE LOBE, RIGHT LOWER LOBE, LEFT UPPER LOBE AND LOWER LOBES	BILATERAL	DISTAL	NIL	NIL	NIL
102	SOMAPPA	68	M	FARMER	155	66	27.47138398	NIL	NAD	PRESENT	4	WNL	72	120/70	96	NVBS	COPD CHANGES	3	E	40	66.7	0.66	32	40	230	16	2	A	3	11.2	16.5	86	4.3	NIL	PRESENT	ABSENT								
103	MADIWALAPPA	80	M	FARMER	160	71	27.734375	HTN	NAD	PRESENT	5	WNL	86	130/80	94	NVBS	COPD CHANGES	3	E	45.76	65.37	0.7	24	28	318	14	0	A	2	12.5	12.5	70	3.1	NIL	PRESENT	ABSENT								
104	SUSHILA KAMBLE	59	F	HOUSE WIFE	169	70	24.50894577	NIL	NAD	NIL	4	WNL	84	140/80	94	NVBS	COPD CHANGES	2	E	65.12	93.03	0.7	52	40	321	16	1	A	2	12.4	5.6	56	3.6	NIL	PRESENT	ABSENT								
105	BALAKKA	68	F	HOUSE WIFE	168	50	17.7154195	HTN,T2DM	NAD	NIL	6	WNL	66	130/80	98	NVBS	COPD CHANGES	3	CB	44	70.4	0.63	33	40	441	18	2	A	2	11.8	6.2	60	3.6	NOG	PRESENT	ABSENT								
106	RAMACHANDRA PATIL	70	M	FARMER	170	62	21.4532872	HTN	NAD	PRESENT	12	WNL	68	130/80	99	NVBS	COPD CHANGES	3	E	49.28	70.4	0.7	25	27	320	25	4	E	3	12.5	7.8	76	3.2	MRSA	PRESENT	PRESENT	TRACTION	34	RIGHT LOWER LOBE	UNILATERAL	DISTAL	NIL	LEFT THIN RIM	NIL
107	SHRISHAIL	65	M	FARMER	158	70	28.04037814	HTN	NAD	PRESENT	7	WNL	76	140/80	97	NVBS	NORMAL	2	E	52.8	89.6	0.59	39	52	300	20	0	A	2	11.2	6.2	54	3.6	COMMENSALS	PRESENT	ABSENT								
108	MAHADEVI KURBAR	68	F	HOUSE WIFE	169	70	24.50894577	HTN	NAD	NIL	4	WNL	56	120/70	99	NVBS	NORMAL	2	E	63.36	100.8	0.63	50	30	321	18	2	A	2	10	4.5	41	4.2	NIL	PRESENT	ABSENT								
109	GOVIND	55	M	LABOURER	163	65	24.4646016	NIL	NAD	PRESENT	6	WNL	68	110/70	98	NVBS	NORMAL	2	E	52.8	75.43	0.5	28	32	330	16	1	A	2	14	6.8	46	4.6	NIL	PRESENT	ABSENT								
110	APPANNA PATIL	76	M	FARMER	164	52	19.33372992	T2DM	NAD	PRESENT	9	WNL	92	140/80	94	NVBS	NORMAL	2	CB	70.4	100.57	0.7	20	22	322	14	3	B	2	12.8	3.4	66	4	NIL	PRESENT	PRESENT	TRACTION	38	RIGHT MIDDLE LOBE, RIGHT LOWER LOBE, LEFT LOWER LOBES	BILATERAL	DISTAL	NIL	NIL	NIL
111	VENKAT RAO	65	M	LABOURER	147	55	25.45235781	T2DM,HTN,IHD	NAD	NIL	4	WNL	52	110/70	96	NVBS	NORMAL	2	E	66.23	97.9	0.68	46	60	724	16	0	A	2	11.2	5.3	41	3.5	NOG	PRESENT	ABSENT								

112	BHIMU	61	M	LABOURER	157	65	26.37023814	NIL	NAD	NIL	7	WNL	86	140/80	96	NVBS	COPD CHANGES	2	E	65.74	99.98	0.66	60	82	686	16	2	B	2	10.9	6.3	46	3.6	NOG	PRESENT	ABSENT								
113	SHIVAJI PATIL	78	M	FARMER	166	54	19.59645812	HTN,IHD	NAD	PRESENT	12	WNL	86	110/70	96	CREPTS	COPD CHANGES	2	CB	62.82	96.97	0.65	66	49	402	20	3	B	2	14	7.2	74	3	NOG	PRESENT	PRESENT	CYSTIC	36	RIGHT UPPER LOBE, LEFT UPPER LOBE AND LEFT LOWER LOBE, LINGULAR LOBE	UNILATERA L	PROXIMAL	NIL	RT MILD EFFUSION	NIL
114	SOMNATH DHAMNEKAR	58	M	BANK EMPLOYEE	157	65	26.37023814	NIL	COPD	NIL	2	WNL	88	120/70	96	NVBS	COPD CHANGES	2	E	70.4	100.57	0.7	20	22	322	13	1	A	1	13.4	6.2	42	3.3	NIL	PRESENT	ABSENT								
115	SATISH KUMBAR	65	M	SUGAR FACTORY	170	78	26.98961938	T2DM, HTH	NAD	PRESENT	3	WNL	86	140/80	96	NVBS	NORMAL	2	E	66.23	97.9	0.68	46	60	724	10	2	B	0	16	5.2	55	3.8	PSEUDOMON AS AUREGINOSA	PRESENT	ABSENT								
116	BASAVARAJ PATTANSHETTI	84	M	FARMER	154	44	18.5528757	T2DM	NAD	PRESENT	9	WNL	86	110/70	94	NVBS	NORMAL	2	CB	65.74	99.98	0.66	60	82	686	14	1	A	2	11.8	3.9	41	3.2	NIL	PRESENT	ABSENT								
117	RAMA D CHOUGULE	82	M	FARMER	160	54	21.09375	NIL	NAD	PRESENT	7	WNL	88	140/80	96	NVBS	NORMAL	2	E	62.82	96.97	0.65	66	49	402	14	1	A	2	12.4	3.5	41	2.8	NIL	PRESENT	ABSENT								
118	BHIMAPPA PATIL	56	M	LABOURER	164	52	19.33372992	NIL	NAD	NIL	8	WNL	96	110/70	98	NVBS	NORMAL	2	CB	65.12	93.03	0.7	52	40	321	16	3	E	2	12.6	5.6	66	3.4	COMMENSAL S	PRESENT	PRESENT	CYSTIC	37	RIGHT MIDDLE LOBE, RIGHT UPPER LOBE, LEFT UPPER LOBE	BILATERAL	PROXIMAL	NIL	NIL	NIL
119	MARUTI PATIL	68	M	FARMER	158	80	32.04614645	T2DM,HTN,IHD	NAD	PRESENT	5	BARREL CHEST	94	130/80	98	NVBS	COPD CHANGES	2	E	62	71	0.7	49	52	565	24	5	E	3	16.8	13.2	80	5.4	KLEBSIELLA PNEUMONIA	PRESENT	PRESENT	TRACTION	30	RIGHT LOWER LOBE	UNILATERA L	DISTAL	PRESENT	NIL	CARDIOMEG ALY
120	SHARANAYYA HIREMATH	83	M	SHOPKEEPER	166	47	17.05617651	T2DM,HTN	NAD	PRESENT	15	WNL	82	140/80	98	B/L CREPTS	COPD CHANGES	2	CB	66.23	97.9	0.68	46	60	208	16	3	E	2	12.6	7.6	74	2.8	NIL	PRESENT	PRESENT	TRACTION	34	RIGHT MIDDLE LOBE, RIGHT UPPER LOBE	UNILATERA L	DISTAL	NIL	NIL	NIL
121	GOUDAPPA KALLASANAVAR	81	M	FARMER	160	57	22.265625	HTN	NAD	PRESENT	10	WNL	74	110/70	96	NVBS	COPD CHANGES	2	E	65.74	99.98	0.66	60	82	230	14	1	A	2	12.4	6.5	55	2.4	NOG	PRESENT	ABSENT								
122	VIRUPAXI KOUJALGI	65	M	FACTORY WORKER	160	77	30.078125	T2DM	NAD	NIL	11	WNL	76	120/70	96	NVBS	COPD CHANGES	2	E	52.8	75.43	0.5	28	32	300	13	2	A	2	11.8	6.4	50	3.4	NOG	PRESENT	ABSENT								
123	MAHADEVAPPA RAYR	63	M	LABOURER	168	70	24.8015873	NIL	NAD	NIL	6	WNL	84	140/80	96	NVBS	NORMAL	2	E	70.4	100.57	0.7	20	22	310	8	0	A	0	15.4	6.7	51	4.5	NIL	PRESENT	ABSENT								
124	KRISHNAJI	65	M	LABOURER	157	60	24.34175829	T2DM	NAD	PRESENT	9	WNL	92	130/80	94	NVBS	NORMAL	2	E	66.23	97.9	0.68	46	60	290	12	0	A	1	14.2	6.8	51	5	NIL	PRESENT	ABSENT								
125	RATNAPPA KHATEDAR	69	M	FARMER	166	56	20.32225287	T2DM	NAD	PRESENT	8	WNL	90	110/70	94	NVBS	NORMAL	2	E	65.74	99.98	0.66	60	82	280	14	3	B	2	10.2	6.5	70	3.1	NIL	PRESENT	PRESENT	CYSTIC	38	RIGHT MIDDLE LOBE, RIGHT LOWER LOBE, LEFT LOWER LOBE	BILATERAL	DISTAL	NIL	NIL	NIL
126	BASAVARAJ PUJAR	56	M	SUGAR FACTORY	170	60	20.76124567	NIL	NAD	NIL	4	WNL	88	120/70	95	NVBS	NORMAL	3	E	45.76	65.37	0.7	24	28	318	20	2	B	3	12.6	6.8	55	4.3	NOG	PRESENT	ABSENT								
127	BHIMANGOUDA PATIL	71	M	FARMER	157	57	23.12467037	HTN	COPD	PRESENT	12	WNL	88	140/80	96	NVBS	NORMAL	2	E	65.12	93.03	0.7	52	40	321	18	2	B	2	9.8	6.6	51	3.4	NIL	PRESENT	ABSENT								
128	BASVANTAPPA PATIL	78	M	FARMER	158	67	26.83864765	HTN, DM, IHD	NAD	PRESENT	17	WNL	86	130/80	96	NVBS	COPD CHANGES	3	E	44	70.4	0.63	33	40	310	24	4	E	3	12.8	8.2	77	3.1	NIL	PRESENT	PRESENT	TRACTION	30	RIGHT UPPER LOBE, LEFT UPPER LOBE AND LEFT LOWER LOBE	BILATERAL	DISTAL	PRESENT	NIL	NIL
129	RAMANGOUDAPATIL	71	M	BANK EMPLOYEE	147	45	20.82465639	HTN	NAD	PRESENT	11	WNL	74	140/80	98	NVBS	NORMAL	3	E	49.28	70.4	0.7	25	27	400	18	2	B	2	12.8	6.4	41	3.6	NIL	PRESENT	ABSENT								
130	SHIVAGOUDA GALI	67	M	LABOURER	165	70	25.71166208	T2DM,HTN	NAD	PRESENT	6	WNL	72	130/80	94	NVBS	COPD CHANGES	2	E	52.8	89.6	0.59	39	52	300	18	2	B	2	12.6	6.3	42	4.2	NIL	PRESENT	ABSENT								
131	BABURAO LAKHE	56	M	FARMER	167	56	20.07960128	HTN	NAD	PRESENT	4	WNL	78	120/70	96	NVBS	NORMAL	2	E	63.36	100.8	0.63	50	30	321	20	2	B	2	11.2	4.5	41	3.6	NIL	PRESENT	ABSENT						ABSENT	ABSENT	NAD
132	BASAVARAJ SANNVEERAPPANA VAR	58	M	FARMER	168	74	26.21882086	HTN, DM, IHD	NAD	PRESENT	3	WNL	80	130/90	94	NVBS	NORMAL	2	E	52.8	75.43	0.5	28	32	330	24	1	A	2	14	4.6	55	4.1	ENTEROCOC CUS FAECALIS	PRESENT	ABSENT							PRESENT - B/L	CARDIOMEG ALY
133	BAGAPPA ARABALLI	70	M	EX-MILITARY	164	84	31.23140988	HTN	NAD	PRESENT	9	WNL	90	120/90	96	NVBS	COPD CHANGES	2	E	70.4	100.57	0.7	20	22	322	16	2	B	1	9.7	15.6	86	2.9	PSEUDOMON AS AUREGINOSA	PRESENT	PRESENT	CYSTIC	44	RIGHT MIDDLE LOBE, RIGHT LOWER LOBE, LEFT LOWER LOBE, LINGULAR LOBE	BILATERAL	PROXIMAL	PRESENT	ABSENT	MUCUS PLUGGING IN RT UL
134	BIVAJI NANDVADEKAR	69	M	FARMER	164	74	27.51338489	HTN	NAD	PRESENT	4	WNL	90	110/70	96	NVBS	COPD CHANGES WITH E/O PLEURAL EFFUSION	4	E	29.04	41.49	0.7	15	6	209	24	1	A	3	12.6	8.6	56	3.6	NIL	PRESENT	ABSENT						NIL	PRESENT B/L	NIL
135	CHANAMALLAPPA NYAMAGOUDA	67	M	LABOURER	158	52	20.82999519	NIL	NAD	PRESENT	4	WNL	94	120/80	96	NVBS	COPD CHANGES	3	CB	45.76	65.37	0.7	38	40	330	18	3	B	2	11.5	10.5	70	2.9	KLEBSIELLA PNEUMONIA	PRESENT	PRESENT	TUBULAR	38	RIGHT MIDDLE LOBE, RIGHT LOWER LOBE	BILATERAL	PROXIMAL	NIL	NIL	NIL
136	CHANDRAKANTH KALLAPPAGOUDAR	72	M	FARMER	160	42	16.40625	NIL	NAD	NIL	8	WNL	90	110/70	92	NVBS	COPD CHANGES	3	CB	39.6	56.57	0.7	22	27	280	16	3	E	3	14.2	14.6	98	1.8	COAGULASE NEGATIVE STREPTOCO CCI	PRESENT	PRESENT	CYLINDRICAL	34	RIGHT UPPER LOBE, RIGHT MIDDLE LOBE, LEFT UPPER LOBE	BILATERAL	DISTAL	NIL	NIL	NIL
137	DHARMSINGH KAKHABDAKI	48	M	BANK	158	48	19.22768787	NIL	NAD	PRESENT	6	WNL	94	110/70	92	NVBS	COPD CHANGES	2	CB	54.56	77.94	0.7	52	43	250	14	4	E	3	13.6	16.8	96	3.4	ECOLI	PRESENT	PRESENT	CYLINDRICAL	36	RIGHT MIDDLE LOBE	UNILATERA L	DISTAL	NIL	NIL	NIL
138	DUNDAPPA	71	M	FARMER	160	74	28.90625	NIL	NAD	PRESENT	9	WNL	92	130/80	94	NVBS	NORMAL	3	E	46	66	0.69	28	38	311	16	2	B	2	11	5.8	56	3.6	NIL	PRESENT	ABSENT						PRESENT	PRESENT	CARDIOMEG ALY
139	GANGAVVA LAGALI	48	F	HOUSE WIFE	148	52	23.73995617	NIL	NAD	NIL	8	WNL	90	110/70	96	NVBS WITH SCATTERE D RHONCHI	NORMAL	3	E	45	59	0.69	34	28	260	18	2	B	3	8.6	6.8	65	4.2	NIL	PRESENT	ABSENT						NIL	NIL	NIL

140	GANGAVVA MADIWALAR	49	F	HOUSE WIFE	162	52	19.81405274	NIL	NAD	NIL	4	WNL	74	120/70	94	NVBS	NORMAL	2	CB	65.12	93.03	0.7	42	40	303	16	3	E	3	8.2	14.2	77	3	PSEUDOMONAS AUREGINOSA	PRESENT	PRESENT	TUBULAR	34	LEFT LOWER LOBE	BILATERAL	PROXIMAL	NIL	NIL	NIL
141	HARAKCHAND JAIN	78	M	VENDER	168	77	27.28174603	HTN,DM,IHD	NAD	PRESENT	6	WNL	98	130/90	94	VBS	COPD CHANGES	3	E	39.6	56.57	0.7	22	27	280	10	2	A	1	9.6	12.6	66	3.2	NIL	PRESENT	ABSENT						PRESENT	NIL	NIL
142	HUSENSAB NADAF	65	M	VENDER	172	68	22.98539751	NIL	NAD	NIL	4	WNL	94	110/80	95	NVBS	COPD CHANGES	2	E	54.56	77.94	0.7	52	43	250	10	0	A	1	14.2	5.4	50	4.2	NIL	PRESENT	ABSENT						NIL	NIL	NIL
143	JINAPPA MARADI	76	M	FARMER	174	52	17.17532039	HTN, IHD	NAD	PRESENT	12	WNL	68	130/90	94	NVBS	COPD CHAGES	3	CB	46	66	0.69	28	38	311	24	3	B	3	9.8	9.6	86	3.1	COMMENSALS	PRESENT	PRESENT	CYSTIC	32	RIGHT UPPER LOBE, RIGHT MIDDLE LOBE	BILATERAL	DISTAL	PRESENT	NIL	NIL
144	RAJSHEKAR TORGAL	55	M	FARMER	154	44	18.5528757	HTN,CVA,CLD	NAD	NIL	3	WNL	110	120/70	96	NVBS	NORMAL	2	CB	52.8	89.6	0.59	39	52	300	18	4	E	2	7.4	14.9	87	1.8	NIL	PRESENT	PRESENT	TRACTION	38	RIGHT UPPERLOBE, RIGHT MIDDLE LOBE	BILATERAL	PROXIMAL	NIL	NIL	NIL
145	MUTTAPPA HEGADE	50	M	FARMER	162	51	19.43301326	T2DM	NAD	PRESENT	2	WNL	98	110/70	94	NVBS WITH SCATTERED RHONCHI	NORMAL	3	CB	44	70.4	0.63	33	40	270	24	3	E	3	10.2	17.6	78	2.8	NOG	PRESENT	PRESENT	CYSTIC	32	RIGHT UPPER LOBE, RIGHT MIDDLE LOBE, RIGHT LOWER LOBE, LEFT UPPER LOBE, LEFT LOWER LOBE	BILATERAL	DISTAL	NIL	NIL	NIL
146	MARUTI JOGANI	73	M	FARMER	158	52	20.82999519	HTN, DM, IHD	NAD	PRESENT	8	BARREL CHEST	78	120/70	94	NVBS	COPD CHAGES	2	CB	52.8	89.6	0.59	39	52	300	14	3	B	2	12.5	13.8	90	3.3	PSEUDOMONAS AUREGINOSA	PRESENT	PRESENT	CYLINDRICAL	40	RIGHT UPPER LOBE, RIGHT MIDDLE LOBE, LINGULAR LOBE	UNILATERAL	PROXIMAL	NIL	NIL	NIL