Original Article

Integrating Etiotypes and Phenotypes in COPD Exacerbations: Clinical Insights from a North Indian Cohort

Brij Bihari Patel[1], Kapil Sharma[2], Vijay Pal[3], Vipin Goyal[3], Suraj Chawla[4], Shahanas C[3], Sachin Batra[3], Yuthika Agrawal[5]

[1]Assistant Professor, School of Excellence in Pulmonary Medicine, Netaji Subhash Chandra Bose Medical College, Jabalpur, MP, India. [2]Associate Professor, Department of Respiratory Medicine, Shaheed Hasan Khan Mewati Government Medical College (SHKM GMC), Nalhar, Nuh, Haryana, India.
[3]Department of Respiratory Medicine, Shaheed Hasan Khan Mewati Government Medical College (SHKM GMC), Nalhar, Nuh, Haryana, India.
[4]Department of Community Medicine, Shaheed Hasan Khan Mewati Government Medical College (SHKM GMC), Nalhar, Nuh, Haryana, India.
[5]Department of Biochemistry, Shaheed Hasan Khan Mewati Government Medical College (SHKM GMC), Nalhar, Nuh, Haryana, India

Abstract

Background: Chronic obstructive pulmonary disease (COPD) is a heterogeneous syndrome with diverse etiological and clinical presentations. While recent frameworks classify COPD patients by both etiotype (environmental, infection-related, asthma-related, or other) and phenotype (emphysema, chronic bronchitis, asthma–COPD overlap, or non-exacerbator), such dual classification has rarely been explored in low- and middle-income countries (LMICs), where unique exposures and infection burdens are prevalent. This study aims to assess the distribution and overlap of etiotypes and phenotypes among patients hospitalised with acute exacerbations of COPD (AECOPD) in North India. **Material and Methods:** We conducted a prospective observational study at a tertiary care hospital in Haryana, India, enrolling 60 adult patients admitted with AECOPD between April 2023 and March 2024. Each patient was classified by etiotype (GOLD 2023) and phenotype (GesEPOC). We compared clinical, spirometric, and radiological profiles across groups, including symptom scores (mMRC, CAT), lung function, HRCT findings, and outcomes. **Results:** Environmental exposures accounted for the most common etiotype (46.6%), followed by infection-related (25.0%), asthma-related (16.7%), and mixed/other causes (11.7%). The predominant phenotypes were chronic bronchitis (43.4%) and emphysema (40.0%), with fewer non-exacerbators (10.0%) or asthma–COPD overlap cases (3.4%). Environmental COPD was strongly correlated with emphysematous changes, while infection-related COPD was linked to chronic bronchitis. HRCT and spirometric patterns varied consistently among subgroups. Independent predictors of severe outcomes included smoking exposure, recurrent infections, high CAT scores, and exacerbator phenotype. **Conclusion:** Integrating etiotype and phenotype frameworks improves our grasp of COPD heterogeneity and supports individualised management strategies. This dual classification is particularly relevant in high-exposure settings such as India and may facilitate precision medicine for COPD in resource-limited contexts.

Keywords: COPD, etiotypes, phenotypes, exacerbation, asthma–COPD overlap, HRCT.

Received: 26 September 2025 **Revised: 25 October 2025** **Accepted: 10 November 2025** **Published: 01 December 2025**

Introduction

Chronic obstructive pulmonary disease (COPD) is a major and growing cause of morbidity and mortality worldwide, currently ranked as the third leading cause of death globally.[14,15,22] According to the Global Burden of Disease (GBD) study, COPD is responsible for an estimated 3.23 million deaths annually, with nearly 90% of these occurring in low- and middle-income countries (LMICs).[14,22] Apart from mortality, COPD imposes a heavy burden in terms of disability-adjusted life years (DALYs), increased healthcare expenditure, frequent hospitalisations, and loss of productivity.[14,15] Despite this, COPD has long been under-recognized and underfunded compared to other major health concerns.[6,7]

COPD is not a single disease but a syndrome with multiple pathological pathways, shaped by genetics, environmental exposures, and comorbidities.[2,6,7] It is defined by persistent respiratory symptoms such as dyspnoea, cough, and sputum production, as well as airflow limitation that is not fully reversible and usually progressive.[2,7] Historically, attention was focused on smoking as the primary risk factor, especially in high-income countries. However, the epidemiology in LMICs has shifted, with biomass fuel exposure, ambient air pollution, and recurrent respiratory infections now recognized as major contributors alongside tobacco use.[6,12]

COPD in the Indian Context

India carries a disproportionate share of the global COPD burden, accounting for nearly 18% of global cases and almost 20% of COPD-related deaths.[5,6] The prevalence among adults aged 40 years or older is estimated at 7–10%, but this figure masks significant regional variation.[5,6] Rural states such as Haryana,

Address for correspondence: Dr. Kapil Sharma, Associate Professor, Department of Respiratory Medicine, Shaheed Hasan Khan Mewati Government Medical College, Nalhar, Nuh, Haryana, India.
E-mail: kapshaims@gmail.com

DOI:
10.21276/amit.2025.v12.i3.211

How to cite this article: Patel BB, Sharma K, Pal V, Goyal V, Chawla S, Shahanas C, Batra S, Aggarwal Y. Integrating Etiotypes and Phenotypes in COPD Exacerbations: Clinical Insights from a North Indian Cohort. Acta Med Int. 2025;12(3):969-974.

Punjab, and Uttar Pradesh, where the use of biomass fuels and tobacco is common, report especially high prevalence rates.[5,12]

Several unique risk factors underpin the COPD epidemic in India:

- Nearly 700 million people, especially women, are exposed to household air pollution from solid fuels (such as wood, dung, and crop residues), which increases the risk of COPD even among non-smokers.[6,12]
- India is the world's second-largest consumer of tobacco, with both smoking and smokeless forms contributing.[6]
- Recurrent respiratory infections, including childhood and adult tuberculosis, predispose to chronic airway changes that may overlap with or mimic COPD.[6,13]
- Agricultural, mining, and industrial workers are at high risk for dust and chemical exposures.[6]
- Northern states, including Haryana and Delhi, consistently rank among the most polluted areas globally, compounding respiratory risks.[6]

These overlapping risk factors mean that COPD in India is not merely a "smoker's disease" but a complex syndrome resulting from a web of environmental and infectious exposures.[6,12] This diversity calls for more nuanced approaches to classification and management.

The Concept of COPD Etiotypes

Traditional COPD classification has relied on spirometric severity (GOLD stages based on FEV_1).[2] However, this approach does not capture the underlying etiological diversity. The 2023 GOLD strategy proposes an aetiotype-based framework, recognising four principal categories: asthma-related (COPD-A), infection-related (COPD-I), environmental (COPD-E), and other/genetic (COPD-O).[2,16] Each aetiotype reflects distinct pathobiological pathways—persistent asthma, infections (including post-tuberculosis), environmental exposures (tobacco, biomass, pollutants), and genetic or mixed causes.[2,8,12,13] Recognising aetiotypes can guide targeted prevention and management strategies.[2,16]

Phenotypic Heterogeneity in COPD

Parallel to aetiotype classification, phenotyping COPD based on clinical and radiological features has gained prominence [3,4]. The GesEPOC guidelines provide a widely used framework, categorising patients as non-exacerbators, exacerbators with emphysema or chronic bronchitis, or those with asthma–COPD overlap (ACO).[3,4,8] These phenotypes, although not mutually exclusive, help clinicians stratify risk and tailor therapy. For example, chronic bronchitis may benefit from mucolytics and macrolides, while emphysema may require rehabilitation or surgical intervention.[3,4,11]

Rationale for Combining Etiotypes and Phenotypes

Integrating both frameworks offers a comprehensive view of COPD heterogeneity. Etiotypes explain why the disease develops, while phenotypes describe how it presents and progresses.[2-4,11] For example, environmental exposures (COPD-E) often lead to emphysema, recurrent infections (COPD-I) to chronic bronchitis, and long-standing asthma (COPD-A) can evolve into ACO.[2-4,9,11,12] This dual classification supports precision medicine, enabling clinicians to identify high-risk subgroups, tailor interventions, and refine prognosis.[3,4,9,11]

Evidence Gap and Study Rationale: Despite these advances, few Indian studies have explored the intersection of etiotype and phenotype.[5,13] Most research focuses on prevalence and risk factors, without dual classification, even in high-burden regions like Haryana.[5,13] Clinicians thus often rely on generic treatment algorithms that may not address underlying risks or clinical diversity. This study systematically classifies AECOPD patients by both etiotype (GOLD 2023) and phenotype (GesEPOC), examines their clinical and radiological features, and explores the relationship between these frameworks in a North Indian setting.

Materials and Methods

Study Design and Setting: This was a prospective, observational study conducted at the Department of Respiratory Medicine, SHKM Government Medical College, Nalhar, Haryana, India. The institution is a tertiary care teaching hospital, serving rural and semi-urban populations in a region with high biomass fuel use, tobacco consumption, and ambient air pollution. The study was conducted over 12 months, from April 2023 to March 2024.

Study Population: We enrolled adult patients (≥40 years) admitted with AECOPD, defined as a sustained worsening of baseline respiratory symptoms (dyspnoea, cough, sputum) necessitating intensified therapy and hospitalisation. Inclusion criteria were: (1) prior COPD diagnosis confirmed by spirometry (post-bronchodilator $FEV_1/FVC < 0.70$); (2) admission for AECOPD; (3) informed consent. Exclusion criteria were: (1) active tuberculosis or post-TB sequelae without COPD; (2) bronchiectasis unrelated to COPD; (3) primary interstitial lung disease, lung cancer, pneumothorax, or pleural effusion; (4) severe cardiovascular or neurological disease likely to affect outcomes; (5) refusal/inability to consent. A total of 60 consecutive eligible patients were recruited, based on admission trends and feasibility for comprehensive assessment.

Ethical Considerations: The study was approved by the Institutional Ethics Committee of SHKM Government Medical College (Approval No: SHKM/IEC/2023/24). Written informed consent was obtained from all participants. Data confidentiality was maintained in accordance with the Declaration of Helsinki.

Clinical Assessment: Demographic data (age, sex, residence), exposure history (smoking status, biomass exposure, occupational history), comorbidities, and exacerbation details were recorded using a structured pro forma. Symptom burden was assessed by the modified Medical Research Council (mMRC) dyspnoea scale and the COPD Assessment Test (CAT).

Classification of Patients

- **Etiotypes (GOLD 2023):** Asthma-related (COPD-A), infection-related (COPD-I), environmental (COPD-E), and other/genetic (COPD-O).[2,16] Classification was based on asthma history, recurrent infections (including TB), exposure histories, and clinical judgement.
- **Phenotypes (GesEPOC):** Non-exacerbator (<2 exacerbations/year), exacerbator with emphysema (≥2 exacerbations/year plus HRCT emphysema), exacerbator with chronic bronchitis (≥2 exacerbations/year plus chronic productive cough), asthma–COPD overlap (ACO; features of asthma and fixed obstruction).[3,4,8]

Radiological and Spirometric Assessment: All patients underwent chest X-ray and high-resolution computed tomography (HRCT) of the thorax using a 128-slice multidetector scanner (1-mm slices, inspiratory/expiratory phases). HRCT findings were independently reviewed by two radiologists blinded to clinical data; discrepancies were resolved by consensus. Recorded features included centrilobular/panlobular emphysema, bronchiectasis, airway wall thickening, bullae, fibrosis, and mosaic attenuation.

Spirometry was performed in accordance with ATS/ERS standards, measuring post-bronchodilator FEV_1, FVC, and FEV_1/FVC ratio. Severity was staged per GOLD: Stage 1 ($FEV_1 \geq 80\%$), Stage 2 (50–79%), Stage 3 (30–49%), Stage 4 (<30%).

Outcomes: Primary outcomes were the distribution and overlap of etiotypes and phenotypes. Secondary outcomes included symptom burden (mMRC, CAT), lung function, HRCT findings, hospitalisation rate, need for ventilatory support (NIV/IMV), and predictors of severe outcomes.

Data Management and Statistical Analysis: Data were double-entered and checked for accuracy. Analyses were done with SPSS v19 (IBM, Chicago, USA). Continuous variables are summarised as mean ± SD; categorical variables as frequencies and percentages. Associations were tested using the chi-square test for categorical data and an independent t-test or ANOVA for continuous variables. Logistic regression identified predictors of severe outcomes. A p-value <0.05 was considered statistically significant.

Results

A total of 85 patients were screened for eligibility during the study period. Of these, 25 patients were excluded (12 due to incomplete data and 13 due to alternative diagnoses). The final cohort included 60 patients who met all inclusion criteria and were systematically classified by both etiotype and phenotype [Figure 1].

Baseline Demographic and Clinical Characteristics

The study cohort (n=60) had a mean age of 64.8 ± 9.2 years (range 46–81); 70% were male and 30% female. The higher proportion of men reflected greater smoking prevalence, while most women had significant biomass exposure. The majority lived in rural areas (63.3%). Smoking exposure was reported by 76.7% (current: 43.3%; former: 33.3%; never: 23.4%), with a mean of 18.7 ± 6.3 pack-years among smokers. Biomass exposure was present in 31.7% of patients, mostly among women. Hypertension (35%) and diabetes mellitus (23.3%) were the most frequent comorbidities.

The most common symptoms were dyspnoea (78.3%), cough (61.7%), sputum (55.0%), and wheeze (40.0%). The mean mMRC score was 2.8 ± 0.9; the mean CAT score was 24.3 ± 6.5, indicating moderate-to-severe symptom burden. Spirometry revealed a mean FEV_1% % predicted of 38.6 ± 12.4, with most patients in GOLD Stage 2 (30%), Stage 3 (48.3%), or Stage 4 (21.7%).

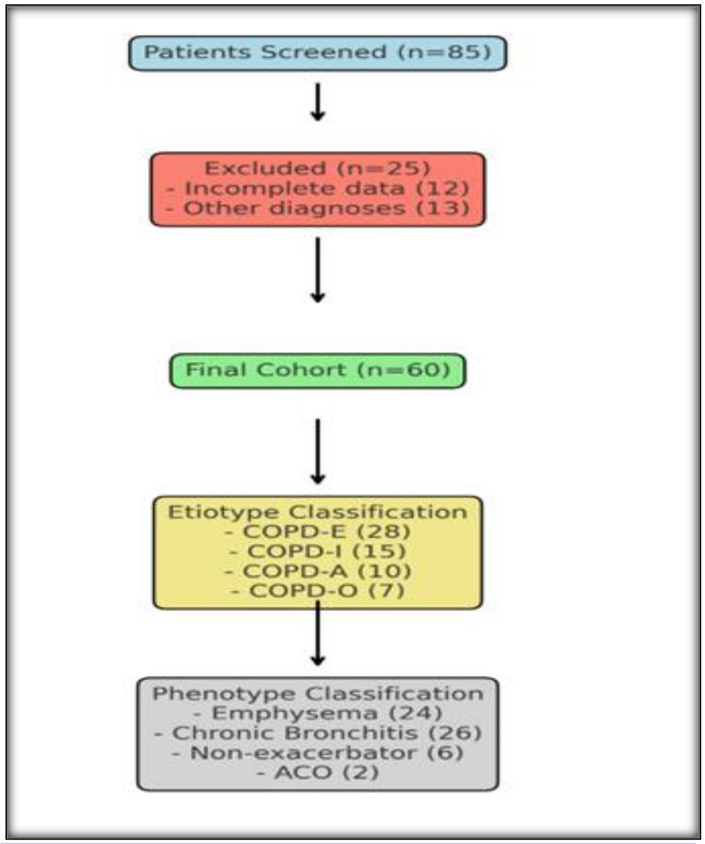

Figure 1: Patient Recruitment Flowchart.

Table 1: Baseline Demographic and Clinical Characteristics of the Study Population (n = 60)

Variable	Value / Distribution
Mean Age (years ± SD)	64.8 ± 9.2 (range: 46–81)
Sex Distribution	Male: 42 (70%); Female: 18 (30%)
Residence	Rural: 38 (63.3%); Urban: 22 (36.7%)
Smoking Status	Current: 26 (43.3%); Former: 20 (33.3%); Never: 14 (23.4%)
Pack-years (mean ± SD)	18.7 ± 6.3 among smokers
Biomass Exposure	19 (31.7%)
Comorbidities	Hypertension: 21 (35%); Diabetes: 14 (23.3%); IHD: 8 (13.3%)
Most Common Symptoms	Dyspnoea: 47 (78.3%); Cough: 37 (61.7%); Sputum: 33 (55%); Wheeze: 24 (40%)
Mean mMRC Score	2.8 ± 0.9
Mean CAT Score	24.3 ± 6.5
Spirometry (FEV1 % predicted)	38.6 ± 12.4
GOLD Staging	Stage 2: 18 (30%); Stage 3: 29 (48.3%); Stage 4: 13 (21.7%)

Note- Values are presented as mean ± SD or number (%). FEV1 = forced expiratory volume in 1 s; GOLD = Global Initiative for Chronic Obstructive Lung Disease; mMRC = modified Medical Research Council dyspnea scale; CAT = COPD Assessment Test; IHD = ischemic heart disease.

Distribution of Etiotypes: According to GOLD 2023, environmental etiotype (COPD-E) was the most common

(46.6%, n=28), followed by infection-related (COPD-I, 25.0%, n=15), asthma-related (COPD-A, 16.7%, n=10), and other/mixed (11.7%, n=7). Among men, environmental etiotype predominated (64.3%), while infection-related COPD was more common among women (40%), largely reflecting post-TB or recurrent infections [Table 2, Figures 2 & 3].

Table 2: Distribution of COPD etiotypes according to GOLD 2023 framework (n = 60).

Etiotype	Number of Patients (%)	Key Characteristics Observed
COPD-E (Environmental)	28 (46.6%)	Smoking, biomass fuel, outdoor air pollution; emphysematous HRCT changes
COPD-I (Infection)	15 (25.0%)	History of TB, recurrent infections; bronchiectasis on HRCT
COPD-A (Asthma)	10 (16.7%)	Longstanding asthma; eosinophilia; reversibility
COPD-O (Other/Mixed)	7 (11.7%)	Genetic, mixed exposures, unclassified
COPD-E, environmental; COPD-I, infection-related; COPD-A, asthma-related; COPD-O, other or mixed causes.		

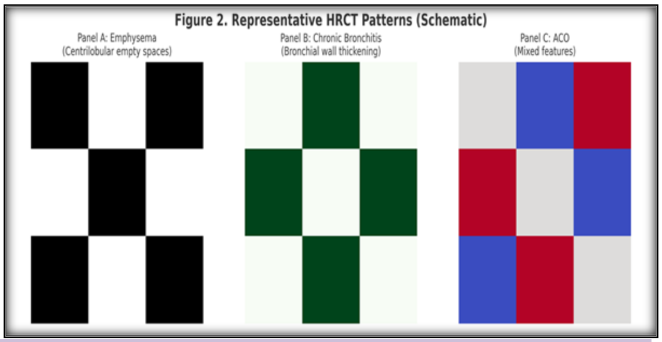

Figure 2: Representative HRCT schematics illustrating the major COPD patterns.

[Figure 2] Panel A: Centrilobular emphysema (environmental etiotype); Panel B: Chronic bronchitis with bronchial wall thickening (infection-related etiotype); Panel C: Asthma–COPD overlap (mixed features).
These schematics illustrate conceptual HRCT patterns rather than patient images.
[Figure 3] Bar chart showing the proportion of environmental, infection-related, asthma-related, and other/mixed etiotypes in the cohort. COPD-E was the most common etiotype (46.6% of patients).

Phenotype Distribution: By GesEPOC classification, the majority of patients belonged to exacerbator phenotypes: exacerbator with chronic bronchitis (43.4%, n=26) and exacerbator with emphysema (40.0%, n=24). A smaller proportion were non-exacerbators (10.0%, n=6) or ACO (3.4%, n=2). Exacerbator phenotypes accounted for 83.4% of hospitalised patients, highlighting the high-risk nature of this cohort [Table 3].

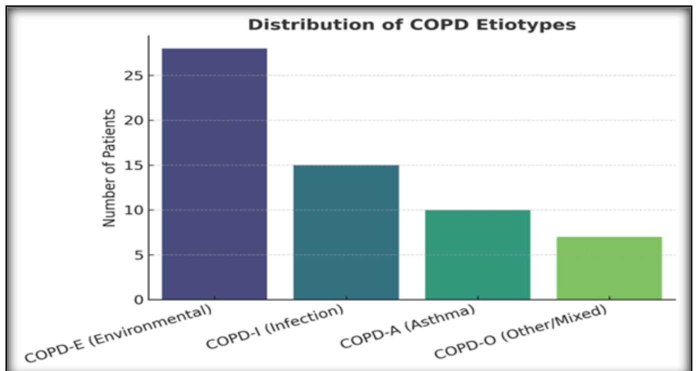

Figure 3. Distribution of COPD etiotypes (n = 60).

Table 3: Distribution of COPD phenotypes according to the GesEPOC classification (n = 60).

Phenotype	Number of Patients (%)	Radiological/Clinical Features
Exacerbator with Emphysema	24 (40.0%)	HRCT: centrilobular/panlobular emphysema; severe dyspnoea
Exacerbator with Chronic Bronchitis	26 (43.4%)	Productive cough; HRCT: bronchial wall thickening, bronchiectasis
Non-exacerbator	6 (10.0%)	Infrequent exacerbations; moderate obstruction
Asthma-COPD overlap (ACO)	2 (3.4%)	Asthma history; eosinophilia; mixed spirometry pattern
Note- Phenotypes included exacerbator with emphysema, exacerbator with chronic bronchitis, non-exacerbator, and asthma–COPD overlap (ACO).		

Etiotype–Phenotype Correlation: Crosstab analysis demonstrated strong associations between etiotypes and phenotypes. Environmental etiotype (COPD-E) was strongly associated with emphysema phenotype (62.5%, $p<0.05$), while infection-related (COPD-I) was linked predominantly to chronic bronchitis (60%, $p<0.05$). Asthma-related COPD (COPD-A) overlapped with ACO in most cases. The other/mixed group showed a varied distribution (Table 4, Figure 4).

Table 4: Cross-tabulation of Etiotypes and Phenotypes (n = 60)

Etiotype \ Phenotype	Emphysema (%)	Chronic Bronchitis (%)	Non-exacerbator (%)	ACO (%)
COPD-E (n=28)	18 (62.5)	7 (25.0)	2 (7.1)	1 (3.6)
COPD-I (n=15)	3 (20.0)	9 (60.0)	2 (13.3)	1 (6.7)
COPD-A (n=10)	1 (10.0)	1 (10.0)	2 (20.0)	6 (60.0)
COPD-O (n=7)	2 (28.6)	3 (42.8)	0 (0.0)	2 (28.6)
The numbers represent the patients in each subgroup. COPD-E was strongly associated with emphysema, whereas COPD-I was more frequent in patients with chronic bronchitis.				

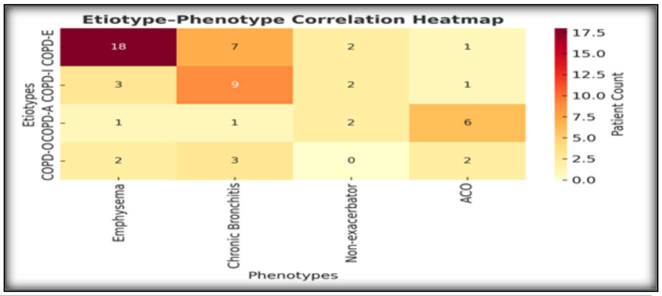

Figure 4: Etiotype–phenotype correlation heatmap.

[Figure 4] Matrix plot showing the patient counts across etiotype–phenotype overlaps. Strong clustering was observed between COPD-E and emphysema and between COPD-I and chronic bronchitis.

Radiological and Spirometric Findings: HRCT scans illustrated distinct patterns according to etiotype and phenotype: environmental COPD showed centrilobular and panlobular emphysema with vascular attenuation, while infection-related COPD revealed bronchial wall thickening and bronchiectasis. ACO cases showed mixed features. Spirometric severity was greatest in the emphysema group (mean FEV_1 32.5 ± 8.4%) compared to chronic bronchitis (mean FEV_1 41.2 ± 13.1%, $p<0.05$).

Discussion

This study provides a comprehensive assessment of dual etiotype–phenotype classification in hospitalised AECOPD patients in Haryana, North India. The findings underscore the heterogeneity of COPD in this region, shaped by a unique combination of environmental, infectious, and genetic factors.

Comparison with the Literature: In Western cohorts, COPD has been predominantly linked to smoking and emphysema-dominant phenotypes.[1,11,19] The ECLIPSE study identified significant variability in exacerbation frequency and radiological features and found that exacerbator subgroups (with emphysema or chronic bronchitis) were clinically meaningful.[1,19] Our current findings parallel the literature in showing that environmental exposures (tobacco, biomass) are strongly associated with emphysema, but diverge in the high prevalence of infection-driven COPD (25%), reflecting the unique Indian epidemiology.[12,13]

Indian and Asian studies emphasise the role of biomass smoke, particularly among women, and the persistent burden of post-tuberculosis airway disease.[5,12,13] Jindal et al. reported that over half of rural women with COPD had no smoking history but significant biomass exposure.[5] Lee et al. demonstrated that biomass smoke exposure independently predicted emphysema and lower lung function in Asian women.[12] Ravikumar et al. described post-tuberculosis COPD as a neglected but distinct subgroup with frequent exacerbations and chronic bronchitis features.[13] Our cohort aligns with these findings, with a significant female population and a notable proportion of infection-related cases.

Clinical Interpretation and Mechanistic Insights: The mapping of etiotypes to phenotypes has direct clinical implications. Environmental exposures induce diffuse alveolar destruction (emphysema) and vascular attenuation on HRCT, with corresponding spirometric impairment and increased symptom burden. Infection-driven COPD is characterised by airway remodelling, mucus hypersecretion, and bronchiectasis, often manifesting as chronic bronchitis with frequent exacerbations and ventilatory needs. Martinez-Garcia et al. described this as the "infective phenotype," in which bacterial colonisation predicts frequent exacerbations.[9]

Asthma-related COPD (COPD-A) overlapped substantially with ACO, with these patients experiencing the highest symptom burden (mean CAT ~32) and poorer short-term outcomes, supporting the concept that ACO is a high-risk phenotype requiring tailored inhaled corticosteroid (ICS) therapy.[8,16]

Strengths and Limitations: Key strengths of our study include the systematic dual classification using current GOLD and GesEPOC frameworks, objective imaging and spirometry, and context-specific data from a high-burden, under-represented setting. Limitations include a modest sample size, a single-centre design, and a cross-sectional approach (precluding long-term outcomes). Exposure assessment was based on self-report, and biomarker (eosinophil, microbiome, genetics) data were lacking.

Clinical and Policy Implications: The high-risk profile of the hospitalised cohort (over 80% exacerbator phenotypes) underscores the need for aggressive prevention, early identification, and tailored management. Environmental/emphysema patients require smoking cessation, rehabilitation, and potentially lung volume reduction; infection/chronic bronchitis patients need infection control, airway clearance, and vaccination; ACO cases are best managed with ICS-LABA regimens and close follow-up.[2-4,8,9,11]

From a policy perspective, COPD prevention in India must address biomass exposure and tobacco use, alongside strengthening infection control (including TB and other respiratory pathogens). Health systems must be prepared for recurrent admissions and ventilatory care.[5,6,12]

Future Research: Longitudinal studies are required to determine whether etiotype–phenotype mapping predicts long-term outcomes (mortality, lung function decline, quality of life). Biomarker research (eosinophils, microbiome, genetics) may further refine subgroups. Interventional trials of phenotype-specific therapies and inclusion of dual classification in national COPD guidelines are warranted.

Conclusion

COPD in India is a complex, heterogeneous condition shaped by diverse exposures and clinical phenotypes. Integrating etiotype and phenotype frameworks provides a pragmatic, clinically relevant approach that enhances understanding of disease patterns and informs personalised management, particularly in resource-limited, high-burden settings such as India.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

References

1. Agustí A, et al. Characterisation of COPD heterogeneity in the ECLIPSE cohort. Lancet Respir Med. 2010;375(9729):965–973.
2. Global Initiative for Chronic Obstructive Lung Disease (GOLD). Global strategy for diagnosis, management, and prevention of COPD, 2023 Report. https://goldcopd.org
3. Miravitlles M, et al. Clinical phenotypes of COPD: identification, definition, and implications for guidelines. Arch Bronconeumol. 2012;48(3):86–98.
4. Miravitlles M, et al. The Spanish COPD guidelines (GesEPOC). Arch Bronconeumol. 2017;53(6):331–341.
5. Jindal SK, et al. COPD prevalence in India: Results from the INSEARCH study. Indian J Chest Dis Allied Sci. 2019;61(3):147–154.
6. Salvi S, et al. The burden of chronic respiratory diseases in India. Lung India. 2019;36(1):1–10.
7. Burney P, et al. The global burden of chronic respiratory disease. Eur Respir J. 2020;55(1):1901215.
8. Sin DD, et al. Asthma-COPD overlap syndrome: what we know and do not know. Chest. 2016;149(1):7–8.
9. Martinez-Garcia MA, et al. Bacterial colonization and COPD exacerbations. Chest. 2012;142(6):1580–1588.
10. Seemugal TA, et al. Exacerbation patterns in COPD: the role of bacterial infection. Am J Respir Crit Care Med. 1998;157(5):1418–1422.
11. Han MK, et al. Radiologic phenotypes of COPD: CT emphysema and disease progression. Thorax. 2017;72(10):857–865.
12. Lee JJ, et al. Biomass fuel exposure and COPD in women: a multi-country Asian study. Respirology. 2023;28(4):331–340.
13. Ravikumar P, et al. Post-tuberculosis COPD in India: A neglected phenotype. Lung India. 2021;38(5):450–456.
14. Soriano JB, et al. Global, regional, and national COPD mortality, 1990–2019: results from GBD 2019. Lancet Respir Med. 2022;10(6):585–601.
15. World Health Organization (WHO). Global Health Observatory data: COPD mortality and disability-adjusted DALYs. Geneva: WHO; 2022.
16. Celli BR, et al. Updated GOLD strategy 2023: Implications for clinicians. Eur Respir J. 2023;61(1):2202246.
17. Divo M, et al. Comorbidities and COPD prognosis: a longitudinal study. Chest. 2012;142(1):112–119.
18. Kim V, et al. Chronic bronchitis in COPD: impact on prognosis and outcomes. Am J Respir Crit Care Med. 2011;183(9):1221–1228.
19. Vestbo J, et al. COPD as a heterogeneous syndrome: insights from ECLIPSE. Respir Med. 2014;108(1):30–38.
20. Papi A, et al. Exacerbations of COPD. N Engl J Med. 2020;383:1832–1848.
21. Chen R, et al. Hospitalization trends in COPD across Asia-Pacific. Int J Chron Obstruct Pulmon Dis. 2021;16:3153–3164.
22. Global Burden of Disease Collaborative Network (GBD). Global Burden of Disease Study 2019. Seattle: IHME; 2020.