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Study of Chronic Obstructive Pulmonary Disease (COPD) Taxonomical Classification in a Tertiary Care Hospital

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ABSTRACT

Background: Chronic Obstructive Pulmonary Disease (COPD) is a major global health concern and the third leading cause of death worldwide. While traditionally associated with cigarette smoking, emerging evidence recognizes a broad range of etiological factors, including indoor air pollution, occupational exposure, infections, and impaired lung development. This study aimed to classify COPD patients into etiologic subtypes according to the updated GOLD 2023 taxonomy and to assess their prevalence in a tertiary care hospital.

Methods: A six-month observational study was conducted among patients with diagnosed COPD at the Oxford Medical College Hospital and Research Centre, Bangalore. Participants with an FEV_1/FVC ratio <0.7 were enrolled after obtaining informed consent. Detailed personal, environmental, and occupational histories were collected to classify each patient according to GOLD 2023 etiotypes.

Results: Among 110 participants, 79.09% were males and 30.90% females. The most common subtype was COPD-c and COPD-p (36.67%), followed by COPD-c alone (30%). Other contributing etiotypes included infection-related (14.17%), pollution-related (8.33%), and developmental (3.33%) factors. Moderate COPD severity was most frequent (36.36%), while severe cases were often associated with occupational exposure or recurrent childhood infections.

Conclusion: The findings highlight that COPD etiology in the studied population extends beyond smoking, with indoor pollution, infections, and early-life respiratory factors contributing significantly. Recognizing these diverse etiotypes allows for better patient stratification and tailored management strategies. Expanding COPD taxonomy beyond smoking-related causes is essential for effective prevention, diagnosis, and individualized treatment planning.

Key-words: COPD, Taxonomy, Etio-types, Smoking, Occupational Exposures, Indoor Pollution

INTRODUCTION

Chronic obstructive pulmonary disease (COPD) is a progressive respiratory disorder that remains a leading cause of morbidity and mortality worldwide. It currently ranks as the third most common cause of death globally and accounts for nearly 5% of all deaths annually [1]. COPD is characterized by persistent respiratory symptoms such as cough, sputum production, and dyspnea, along with irreversible or partially reversible airflow limitation due to abnormalities in the airways and alveoli [2]. Traditionally, the disease has been linked primarily to cigarette smoking, but growing evidence now recognizes several other risk factors, including genetic predisposition, repeated respiratory infections, environmental and

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occupational exposures, nutritional deficiencies, and impaired lung development [3,4].

Despite being historically viewed as a single disease entity, it is now understood that COPD represents a heterogeneous group of disorders arising from multiple interacting mechanisms. The 2023 report of the Global Initiative for Chronic Obstructive Lung Disease (GOLD) redefined COPD as a multifactorial, heterogeneous lung condition caused by lifelong cumulative interactions among genes, environment, and development [5]. These interactions contribute to chronic inflammation, structural airway remodeling, and parenchymal destruction, ultimately resulting in airflow limitation. The variability in causes and presentations demands that COPD be classified more broadly to accommodate etiological and phenotypic diversity rather than being limited to a smoking-related disease model [6].

Recent epidemiological and clinical research has revealed multiple etiological forms of COPD, which can occur even in lifelong non-smokers. The GOLD 2023 taxonomy introduced a new classification system based on the underlying causative mechanism. The first category, genetically determined COPD (COPD-g), includes conditions resulting from inherited disorders such as alpha-1 antitrypsin deficiency, which causes early-onset emphysema primarily affecting the lower lobes [7,8]. The second category, developmental COPD (COPD-D), originates from impaired lung growth during prenatal life or early childhood. Lung maturation continues until early adulthood—around 16–18 years in females and 20–25 years in males—and any insult during this period, including poor nutrition, recurrent infections, or exposure to pollutants, may lead to reduced maximal lung function in adulthood [9–11]. Studies have shown that while some adults develop COPD due to accelerated decline in lung function, others begin with low peak lung function early in life and develop the disease without rapid decline, indicating two distinct trajectories—(COPD-s) and (COPD-d)—each with different prognoses and biological mechanisms [12–14].

Environmental COPD, including smoking-related COPD (COPD-c) and pollution-related COPD (COPD-p), accounts for the majority of global cases. COPD-c results from chronic exposure to cigarette smoke and other forms of tobacco combustion, leading to airway inflammation, parenchymal damage, and emphysema [15,16]. However, not all smokers develop COPD, suggesting that genetic and environmental modifiers influence disease susceptibility. COPD-p, on the other hand, occurs predominantly in developing countries where exposure to biomass fuel smoke, indoor air pollution, and occupational dusts and chemicals are common. It is particularly prevalent among women who cook with solid fuels in poorly ventilated homes [17,18]. Patients with COPD-p typically show less emphysema and more airway-predominant disease, with a slower rate of lung function decline compared to smoking-related COPD [19].

Infection-related COPD (COPD-i) represents another major etiological subgroup. It develops as a consequence of repeated respiratory infections in infancy or early childhood, or following pulmonary tuberculosis, both of which are prevalent in developing countries [20,21]. The pathophysiological mechanism is thought to involve chronic airway inflammation and abnormal tissue repair, leading to fibrosis, bronchiectasis, and airflow obstruction. Another significant phenotype, asthma-associated COPD (COPD-a), arises in patients with severe, uncontrolled asthma who develop persistent airflow limitation independent of smoking or other risk factors [22,23]. These patients often show overlapping features of asthma and COPD, such as eosinophilic inflammation and reduced lung elastic recoil. They may exhibit emphysematous changes similar to those seen in chronic smokers [24–26].

Additionally, idiopathic COPD (COPD-u) refers to cases in which patients develop airflow obstruction without identifiable risk factors. These individuals often present with mild to moderate disease that progresses slowly over time. Epidemiological studies indicate that COPD-u may result from complex interactions involving genetic, epigenetic, and socioeconomic factors, including occupation, sex, education, and income level [27–29]. Although this form is relatively rare, it underscores the need for further research into unidentified causes and their contribution to the global COPD burden.

Recognizing COPD as a spectrum of diseases with diverse etiologies has important implications for both research and clinical practice. The updated GOLD classification emphasises assessing individual risk factors—such as smoking history, indoor air quality, occupational exposure, childhood respiratory infections, and genetic background—to establish an etiological diagnosis. This approach helps clinicians design targeted preventive strategies and personalized therapeutic plans rather than

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relying on a one-size-fits-all model. Moreover, categorizing COPD into etiotypes can improve understanding of disease mechanisms, allow for more accurate prognostic assessment, and optimize resource utilization in healthcare systems. The GOLD 2024 update further reiterates the need for etiological identification in routine clinical evaluation to tailor management and improve patient outcomes.

MATERIALS AND METHODS

Study Design and Setting- A hospital-based observational study was conducted over a period of six months in the Department of Respiratory Medicine, The Oxford Medical College Hospital and Research Centre, Bangalore.

Study Population- All patients clinically diagnosed with COPD based on spirometry (post-bronchodilator FEV_1/FVC ratio <0.7) and showing poor reversibility to bronchodilators were included in the study.

Inclusion Criteria

- Diagnosed cases of COPD as per GOLD 2023 guidelines.
- Patients providing written informed consent for participation.

Exclusion Criteria

- Patients unwilling to participate in the study.
- Patients with alternative diagnoses or overlapping pulmonary conditions.

Data Collection- Detailed demographic data, personal and occupational history, smoking status, indoor air pollution exposure, childhood respiratory infections, and comorbidities were recorded using a pre-designed questionnaire. Each patient was classified into etiological subtypes (COPD-c, COPD-p, COPD-i, COPD-d, COPD-a, COPD-g, COPD-u) according to the latest GOLD 2023 taxonomy.

Statistical Analysis- All data were entered into Microsoft Excel and analysed using descriptive statistics. Frequencies and percentages were calculated for categorical variables, and continuous variables were expressed as mean ± standard deviation (SD). The association between etiological types and disease severity was assessed using cross-tabulation.

Ethical Considerations- The Institutional Ethics Committee approved the study. Written informed consent was obtained from all participants, and confidentiality was maintained throughout the study.

RESULTS

Table 1 shows that the majority of patients belonged to the combined COPD-C and COPD-P group, indicating that both smoking and pollution exposure are the leading causes of COPD in the study population. COPD-C alone was the next most frequent type, followed by infection-related and mixed etiologies. Only a few cases were linked to developmental or asthma-associated factors. Overall, the findings suggest that chronic exposure to smoke and environmental pollutants remains the dominant contributor to COPD in this setting.

Table 1: Etiological Classification among the study population

COPD Classification	Number of Subjects	Percentage (%)
COPD-c + COPD-p	44	36.66
COPD-c only	36	30
COPD-i + COPD-c + COPD-p	17	14.16
COPD-p only	10	8.33
COPD-d + COPD-c	4	3.33
Unclassified	4	3.33
COPD-i + COPD-c	2	1.66
COPD-i + COPD-c + COPD-p + COPD-a	1	0.83
COPD-d + COPD-c + COPD-p	1	0.83
COPD-d + COPD-p	1	0.83

Fig. 1 depicts the distribution of COPD severity according to the GOLD grading system in the study population. The majority of patients had moderate COPD, followed by severe, mild, and very severe forms. This pattern indicates that most patients present at an intermediate stage of the disease, while prolonged exposure to occupational or environmental risk factors tends to be associated with greater severity.

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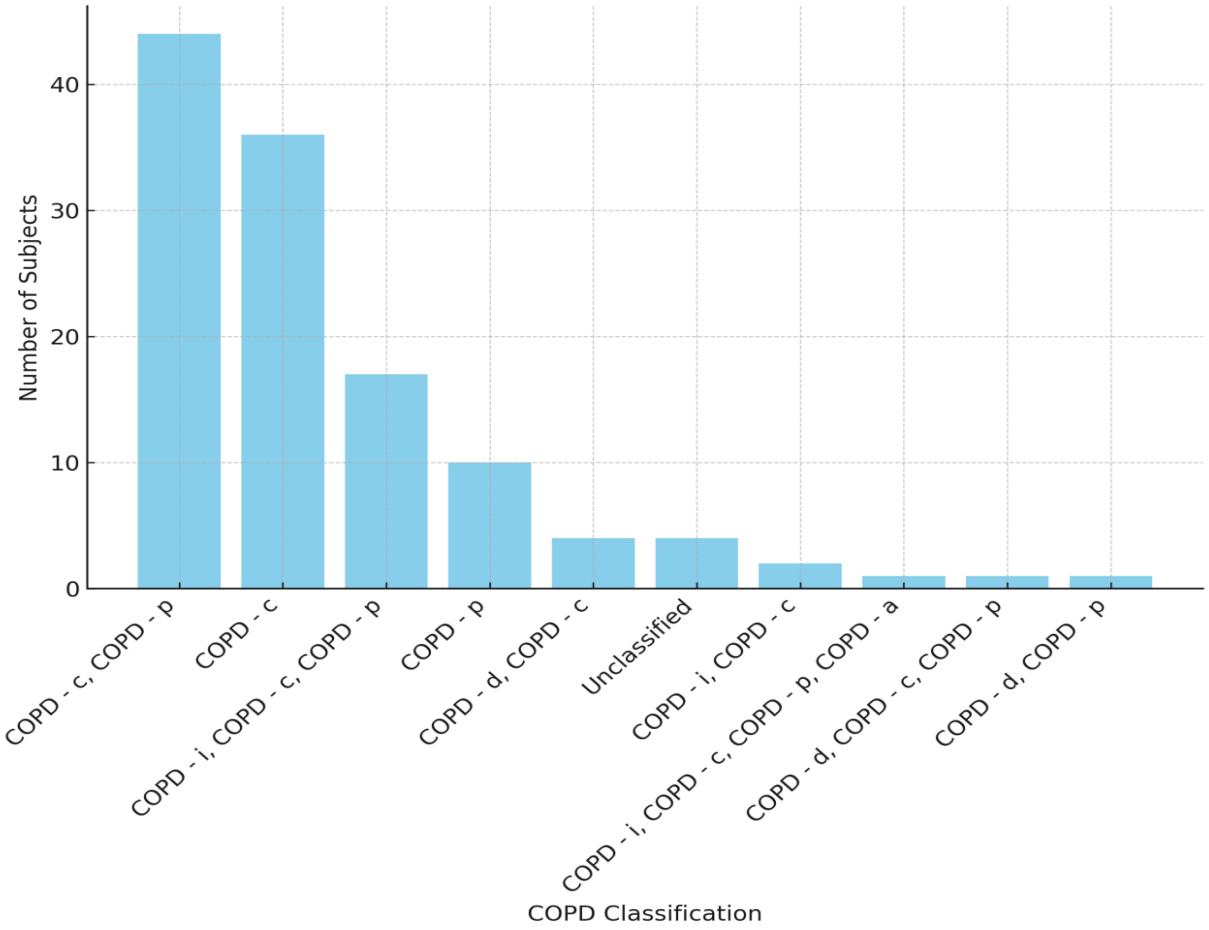

Fig. 1: Frequency distribution of COPD classifications

The results indicate that the majority of cases fall under the COPD-c, COPD-p category (36.67%), followed by COPD-c alone (30%). These findings suggest that chronic exposure to certain risk factors plays a dominant role in COPD pathogenesis among the subjects. COPD-c, COPD-p (36.67%): This is the most common classification, indicating that a significant proportion of patients may have COPD due to a combination of cigarette smoking, passive smoking and indoor pollution, including occupational exposures. The second largest group consists of patients classified solely under COPD-c, signifying that cigarette smoking is a primary risk factor for their disease. COPD-i, COPD-c, COPD-p (14.17%): This category suggests that infection-related factors may have played a role alongside smoking and indoor pollution, including occupational exposures. It highlights the significance of early-life infections or recurrent respiratory infections in the development of COPD.COPD-p (8.33%): Patients in this category are primarily affected by indoor pollution, including occupational exposures, which underscores the importance of public health measures aimed at reducing exposures to smoke/biomass fuels and occupational exposures. COPD-d, COPD-c (3.33%): The presence of developmental factors alongside chronic exposure suggests that low birth weight or premature birth may contribute to compromised lung function, increasing susceptibility to COPD (Fig. 2).

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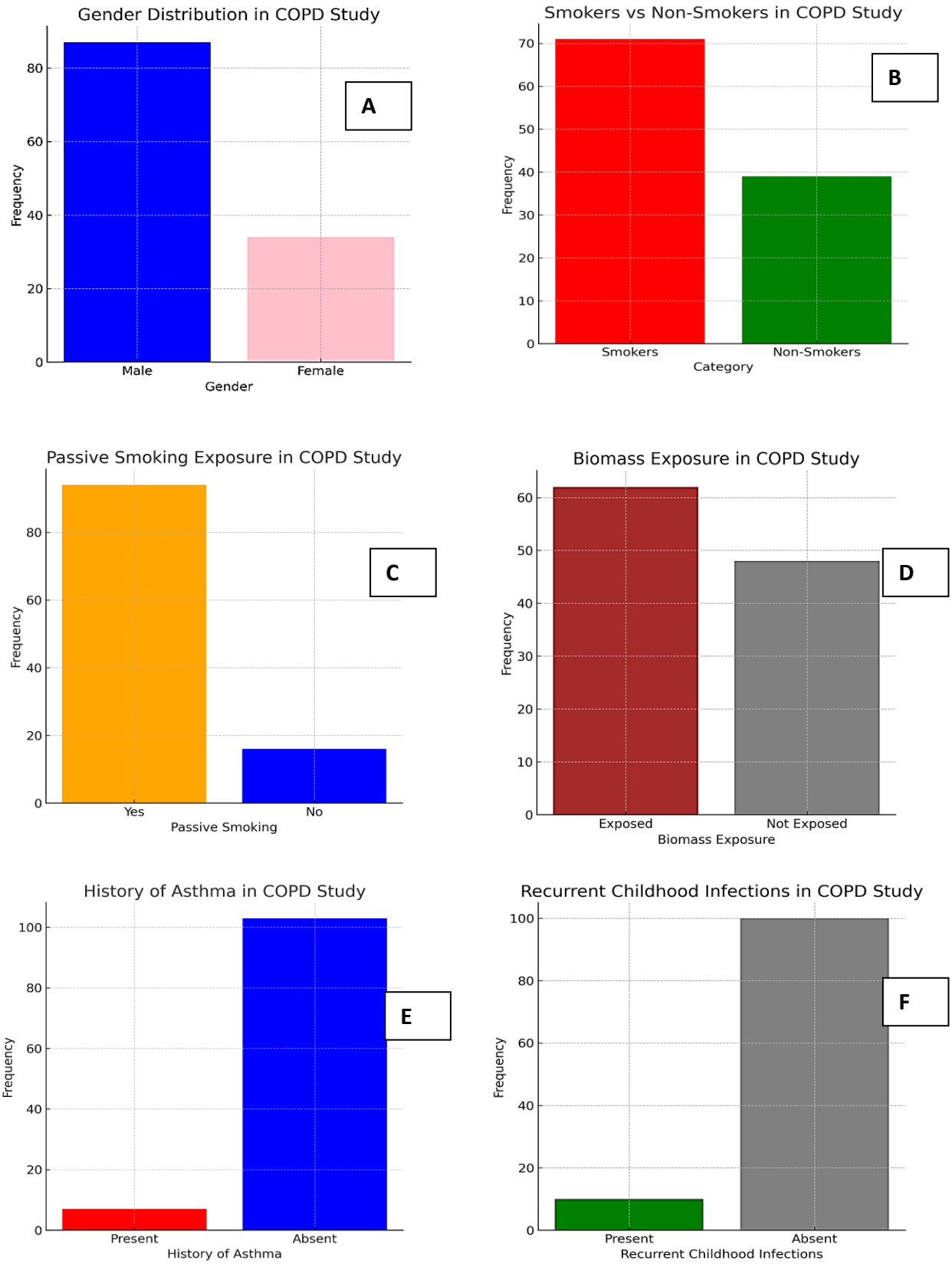

Fig. 2: Representative images showing different etiological types of COPD (A–F): genetically determined (G), developmental (D), smoking-related (C), pollution-related (P), infection-related (I), and asthma-associated (A) forms.

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Among the study population of 110 COPD patients enrolled over a period of six months-79.09% were males, whereas 30.90% were females. 64.54 % were smokers, while 85.45% had exposure to passive smoking. 56.36% were exposed to biomass, 8.18% had occupational exposures, 6.36% had a history of asthma, and 9.09% had recurrent childhood infections (Table 2).

Table 2: Distribution of COPD severity based on GOLD grading

Gold Grade	Frequency	Percentage
Mild-FEV1 >80	22	20
Moderate-FEV1- 50-80%	40	36.36
Severe-FEV1 -30- 50 %	35	31.81
Very Severe <30 %	13	11.81

Patients with a history of recurrent childhood infections were more commonly found in the severe COPD category, followed by moderate and mild stages. This indicates that early-life respiratory infections may have long-term effects on lung development, predisposing individuals to more severe forms of COPD later in life (Table 3).

Table 3: Correlation of COPD severity with recurrent childhood infections

COPD Severity (GOLD Grade)	No Infection History	Yes (Recurrent Infections)	Total
Mild (FEV_1 > 80%)	20	2	22
Moderate (FEV_1 50–80%)	38	3	41
Severe (FEV_1 30–50%)	31	5	36
Very Severe (<30%)	11	0	11
Total	100	10	110

Patients exposed to occupational irritants such as dust, fumes, or chemicals predominantly showed moderate to severe COPD. The higher frequency of severe cases among exposed individuals suggests that prolonged occupational exposure contributes significantly to disease progression and increased severity (Table 4).

Table 4: Correlation of COPD severity with occupational exposures

COPD Severity (GOLD Grade)	No Occupational Exposure	Yes (Occupational Exposure)	Total
Mild (FEV_1 > 80%)	21	1	22
Moderate (FEV_1 50–80%)	39	2	41
Severe (FEV_1 30–50%)	31	5	36
Very Severe (<30%)	10	1	11
Total	101	9	110

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DISCUSSION

In our study, 79.09% of participants were male, and 30.90% were female. This male predominance aligns with global trends, where COPD has historically been more prevalent in men. However, recent studies indicate a rising prevalence of COPD among women, attributed to increased smoking rates and exposure to biomass fuels. For instance, a study highlighted by the American Lung Association notes that while smoking is a primary risk factor, long-term exposure to environmental pollutants also significantly contributes to COPD development [29].

Our finding that 64.54% of participants were smokers is consistent with existing literature identifying smoking as the leading cause of COPD. The high rate of passive smoking exposure (85.45%) in our study underscores its significant risk. The Mayo Clinic emphasizes that the risk of COPD increases with the duration and intensity of smoking [30]. The World Health Organization acknowledges that exposure to second-hand smoke is a considerable risk factor for COPD [31]. Our observation that 56.36% of participants were exposed to biomass fuels aligns with studies from developing countries, where indoor air pollution from biomass combustion is a major COPD risk factor. The American Lung Association reports that ongoing exposure to chemicals, dust, and fumes is are environmental risk for COPD [29].

The 8.18% of participants with occupational exposures in this study is slightly lower than the 10–20% range reported in other studies. The 6.36% prevalence of asthma history among our participants is noteworthy, as the Mayo Clinic identifies asthma as a potential risk factor for developing COPD, particularly when combined with smoking [30]. Our finding that 9.09% of participants had recurrent childhood infections aligns with evidence suggesting that such infections can impair lung development and increase COPD susceptibility later in life. The World Health Organization notes that childhood respiratory infections are among the risk factors for COPD [31].

This study reports that 36.36% of participants had moderate COPD, followed by severe, mild, and very severe cases. This distribution is comparable to other studies, which often find a higher prevalence of moderate and severe COPD stages. For example, research indicates that moderate COPD is commonly observed in clinical settings. Our observation that all aetiological groups predominantly exhibited moderate COPD, with those having occupational exposures tending towards more severe disease, is supported by existing literature. A study published in the European Respiratory Journal found that occupational exposures are associated not only with the development but also with the progression and increased severity of COPD [32].

The classification of COPD cases in our study reveals that the majority fall under the COPD-c, COPD-p category (36.67%), followed by COPD-c alone (30%). These findings align with existing literature emphasising the significant role of cigarette smoking, passive smoking, and indoor pollution, including biomass exposure, in COPD pathogenesis. A study published in the European Respiratory Journal highlights that active smoking remains the primary risk factor for COPD [33]. Our observation that 14.17% of cases were classified as COPD-i, COPD-c, and COPD-p suggests that infection-related factors, alongside smoking and indoor pollution, play a role in COPD development. This is supported by research indicating that respiratory infections can contribute to the onset and progression of COPD [34]. The 8.33% of patients classified under COPD-p underscores the impact of indoor pollution and occupational exposures. Studies have shown that exposure to indoor pollutants, such as biomass fuel used for cooking and heating, is a significant risk factor for COPD, especially in developing countries [35].

The presence of developmental factors alongside chronic exposure in 3.33% of cases (COPD-d, COPD-c) aligns with findings that early-life factors, including low birth weight and childhood respiratory infections, can increase susceptibility to COPD in adulthood [36].

It was also observed that patients across various etiological types predominantly exhibited moderate COPD severity. However, individuals with a history of occupational exposures and recurrent childhood infections tended to present with more severe forms of the disease. Prolonged exposure to occupational irritants such as dust, chemicals, and fumes has been linked to the development and progression of COPD. A case-control study highlighted that prior occupational exposure is not only associated with the onset of COPD but also correlates with greater disease severity. The study found that individuals exposed to vapors, gases, dust, or fumes had a higher risk of developing severe COPD compared to those without such exposures [37].

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Early-life respiratory infections can have long-term implications for lung health. The American Lung Association notes that a history of childhood respiratory infections is a significant risk factor for developing COPD in later life. These infections can impair lung development, leading to reduced lung function and increased susceptibility to COPD. Our findings align with this, as patients with recurrent childhood infections exhibited more severe COPD manifestations [29].

Limitations

The limitations of the study were that certain known risk factors such as premature birth, low birth weight, and alpha-1 antitrypsin deficiency, did not yield significant numbers, possibly due to recall bias and the lack of invasive diagnostic testing in this study. Despite these limitations, the study provides valuable insights into COPD classification and its correlation with various risk factors.

CONCLUSIONS

The present study highlights that COPD in the Indian population is predominantly related to combined exposure from cigarette smoking and environmental pollution, with additional contributions from infections, occupational hazards, and early-life respiratory factors. The findings emphasize the heterogeneous nature of COPD and support the expanded GOLD 2023 taxonomy that recognizes multiple etiological pathways. Identification of specific etiotypes enables more targeted interventions and improved patient management strategies tailored to individual risk profiles. Strengthening public health measures against indoor pollution, promoting smoking cessation, and early screening among high-risk groups are essential to reduce disease burden in resource-limited settings.

CONTRIBUTION OF AUTHORS

Research concept- Dr. Jyothi
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Supervision- Dr. Jyothi
Materials- Dr. E. Ananda Nandini Nimeela
Data collection- Dr. Keerthi N.
Data analysis and interpretation- Dr. Keerthi N., Dr. E. Ananda Nandini Nimeela
Literature search- Dr. Keerthi N.
Writing article- Dr. Keerthi N.
Critical review- Dr. Jyothi
Article editing- Dr. Jyothi
Final approval- Dr. Jyothi

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