Section: Pulmonary Medicine

Original Research Article

CLINICO-BACTERIOLOGICAL PROFILE AND HEMATOLOGICAL INDICES IN ACUTE EXACERBATION OF CHRONIC OBSTRUCTIVE PULMONARY DISEASE IN A TERTIARY CARE HOSPITAL

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ABSTRACT

Background: Chronic Obstructive Pulmonary Disease (COPD) is a leading cause of morbidity and mortality worldwide and is characterized by persistent airflow limitation with recurrent acute exacerbations. Acute exacerbations significantly increase hospitalization, disease progression, and healthcare burden. Bacterial infections are one of the major causes of exacerbations, and understanding the bacteriological profile along with inflammatory markers is essential for proper management and treatment. The aim is to identify the common bacterial pathogens causing acute exacerbation of COPD, to study the antibiotic sensitivity and resistance patterns, and to assess the association between inflammatory markers and severity of exacerbation. **Materials and Methods:** This hospital-based cross-sectional observational study was conducted over 18 months from August 2024 to February 2026. Ninety-eight consecutive patients with COPD and acute exacerbation, diagnosed according to GOLD criteria, were included. Clinical assessment, spirometry, complete blood count, inflammatory markers (NLR, ESR, and CRP), sputum culture, and antimicrobial susceptibility testing were performed. Exacerbations were graded as mild, moderate, or severe according to GOLD 2024 criteria. Data collected were analysed to determine bacteriological patterns and their correlation with disease severity. **Results:** The mean age was 60.27±9.04 years, and 64.3% of patients were males; moderate (41.8%) and severe (40.8%) AECOPD predominated. Sputum cultures were positive in 53.1% of cases, with gram-negative organisms (71.2%) predominating, led by Klebsiella pneumoniae (26.9%). Imipenem (80.8%) and Meropenem (76.9%) demonstrated the highest antimicrobial sensitivity. NLR, PDW, ESR, and CRP increased significantly with increasing AECOPD severity, whereas FEV_1 declined significantly (all $p<0.0001$). Smoking status, sputum culture positivity, and inflammatory markers showed significant positive associations with disease severity, while FEV_1 exhibited a strong negative correlation ($\rho = -0.924$). **Conclusion:** Identification of the bacteriological profile and antibiotic sensitivity pattern in COPD exacerbations helps in selecting appropriate antibiotic therapy. Assessment of inflammatory markers can serve as useful indicators in determining disease severity and guiding clinical management, thereby improving patient outcomes.

INTRODUCTION

Acute exacerbations of COPD (AECOPD) are defined as episodes of worsening respiratory symptoms beyond normal day-to-day variation that necessitate a change in regular medication, and they play a pivotal role in determining disease outcomes, healthcare utilization, and economic burden. These exacerbations are associated with increased inflammation, physiological deterioration, and heightened risk of hospitalization and mortality, making them a central focus of COPD management strategies.[1,2,3]

Acute exacerbations represent a critical turning point in the natural history of COPD, as repeated episodes are associated with accelerated decline in lung function, increased symptom burden, and poorer long-term prognosis. Hospitalized exacerbations, in particular, are associated with substantial short- and long-term mortality, with a significant proportion of patients requiring intensive care support, non-invasive ventilation, or invasive mechanical ventilation. Early identification of high-risk patients during acute exacerbations is therefore essential for optimizing management and improving outcomes.[4,5]
Infective aetiologies play a central role in precipitating acute exacerbations of COPD, with bacterial infections accounting for a substantial proportion of hospitalized cases. Common bacterial pathogens implicated in AECOPD include gram-negative bacilli and gram-positive cocci, with variation observed based on disease severity, prior antibiotic exposure, hospitalization history, and regional microbiological patterns. Identification of the bacteriological profile during exacerbations is crucial for initiating appropriate antimicrobial therapy, preventing treatment failure, and reducing the emergence of antibiotic resistance. Therefore, studying the bacteriological spectrum and antibiotic sensitivity patterns in patients admitted with AECOPD is of considerable clinical importance, particularly in tertiary care settings where multidrug-resistant organisms are increasingly encountered.[6,7]
Regional variations in environmental exposures, smoking patterns, microbiological flora, and healthcare infrastructure necessitate localized research to better understand disease characteristics and outcomes. Tertiary care hospitals serve as referral centres for severe and complicated cases of AECOPD, providing an ideal setting to study the interplay between clinical presentation, bacteriological aetiology, and haematological inflammatory markers. A comprehensive evaluation of these parameters can aid in early risk stratification, guide rational antibiotic use, and improve prognostication.[8]

MATERIALS AND METHODS

This hospital based cross-sectional study was conducted in the Department of Pulmonary Medicine at Al Azhar Medical College, Thodupuzha over a period of 18 months from the date of approval obtained from the Institutional Ethics Committee (06-08-2024 to 6-02-2026). A total of 98 patients fulfilling the inclusion criteria were selected for the study using a non-probability consecutive sampling.

Inclusion & Exclusion Criteria

All confirmed cases of chronic obstructive pulmonary disease presenting with acute exacerbation as defined by GOLD guidelines were included.
Patients with a history of tuberculosis, HIV infection, autoimmune diseases, chronic kidney disease, or any respiratory disease other than COPD, recent history of angina or myocardial infarction, malignancy, cerebrovascular accident, uncontrolled hypertension, heart failure, or any psychiatric illness, those on systemic corticosteroid therapy for the past three months, unwilling to provide written informed consent for participation in the study were excluded.

Procedure

Patients were explained in detail about the study objectives and procedures, and written informed consent was obtained prior to enrolment. Each enrolled subject was a confirmed case of COPD presenting with acute exacerbation.
A detailed clinical evaluation was performed, including documentation of presenting symptoms, number of exacerbations in the previous one year, smoking history, prior hospital admissions, and antibiotic exposure in the last three months. Anthropometric measurements such as height, weight, and body mass index were recorded. History of comorbid conditions including diabetes mellitus, hypertension, coronary artery disease, cerebrovascular accident, and other relevant illnesses was noted.
Clinical assessment included evaluation of dyspnoea using the Modified Medical Research Council (mMRC) Dyspnoea Scale, symptom burden using the COPD Assessment Test (CAT), and breathlessness severity using the Visual Analogue Scale (VAS). Vital parameters, including respiratory rate, heart rate, and resting peripheral oxygen saturation (SpO_2), were recorded at presentation. Arterial blood gas (ABG) analysis was performed when clinically indicated.
Venous blood samples were collected within one hour of hospital admission or before initiation of treatment, whichever occurred earlier. Approximately 5 mL of venous blood was obtained under aseptic precautions out of which 2 mL was collected in EDTA vacutainers for complete blood count, neutrophil-to-lymphocyte ratio (NLR), platelet indices, and erythrocyte sedimentation rate (ESR). The remaining 3 mL was collected in a plain vacutainer, centrifuged to separate serum, and stored at −70°C until estimation of C-reactive protein (CRP).
Sputum samples were collected in sterile containers and were transported to the microbiology laboratory within two hours. Specimen adequacy was assessed by macroscopic and microscopic examination, and Gram-stained smears were evaluated using Bartlett's grading system. Acceptable specimens were processed by inoculation onto blood agar, chocolate agar, and MacConkey agar media. Bacterial isolates were identified using standard microbiological methods, and antimicrobial susceptibility testing was performed using the Kirby–Bauer disk diffusion technique on Mueller–Hinton agar in accordance with standard laboratory guidelines.
Pulmonary function testing was performed using a Winspiro PRO spirometer according to the American Thoracic Society (ATS) recommendations. Post-

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bronchodilator spirometry was conducted 20 minutes after administration of inhaled salbutamol. The severity of acute exacerbation was assessed, and patients were categorized as having mild, moderate, or severe exacerbation based on the Global Initiative for Chronic Obstructive Lung Disease (GOLD) 2024 criteria.

All clinical, laboratory, microbiological, and spirometric findings were recorded in a predesigned case record form and entered into the study database for statistical analysis. Statistical analysis was performed using Statistical Package for the Social Sciences (SPSS) version 30. Quantitative variables were expressed as mean and standard deviation, while categorical variables were expressed as frequency and percentage. A p value of less than 0.05 was considered statistically significant.

Consent and ethical consideraton

The study was carried out after obtaining approval from the Institutional Human Ethics Committee. An informed, written consent was obtained from all the patients. Patients who had given written consent was enrolled in study. All patients had given freedom of opting out of study at any point of time during study.

Confidentiality of data

Confidentiality was ensured and maintained throughout the study. Study result was only being used for scientific purposes and publications. All information about patient's illness and results of the tests was kept confidential and retained in the institute as required under rules. Results may be presented at conferences and published without any disclosure of patient identity directly or indirectly.

RESULTS

The study included 98 patients with a mean age of 60.27 ± 9.04 years, with the majority (32.7%) belonging to the 61–70 years age group. Males predominated (64.3%), and moderate (41.8%) and severe (40.8%) AECOPD were the most common presentations. Nearly half of the participants were current smokers (49.0%), while hypertension (40.8%) and diabetes mellitus (33.7%) were the most frequent comorbidities. Most patients had a history of previous hospitalization (73.5%), and 62.2% experienced at least one exacerbation during the preceding year. Antibiotic use within the previous three months was reported in 43.9% of patients. The mean BMI was 22.14 ± 4.28 kg/m², with over half (54.1%) having a normal BMI.

Clinico-bacteriological Profile

Bacterial growth was identified in 53.1% of sputum samples. Among culture-positive isolates, Klebsiella pneumoniae (26.9%) was the predominant pathogen, followed by Pseudomonas aeruginosa (23.1%) and Acinetobacter baumannii (19.2%). Gram-negative organisms accounted for majority of isolates (71.2%). Purulent and mucoid sputum were each observed in 33.7% of patients, while 32.7% had mucopurulent sputum. This is shown in [Table1 and Figure 1].

Table 1: Clinico-bacteriological Profile of Study Participants (n = 98)

Variable	Category	n (%)
Sputum Culture Result	Positive	52 (53.1)
	Negative	46 (46.9)
Bacterial Isolates (n = 52)	Klebsiella pneumoniae	14 (26.9)
	Pseudomonas aeruginosa	12 (23.1)
	Acinetobacter baumannii	10 (19.2)
	Staphylococcus aureus	8 (15.4)
	Streptococcus pneumoniae	7 (13.5)
	Escherichia coli	1 (1.9)
Gram Stain Characteristics (n = 52)	Gram-negative organisms	37 (71.2)
	Gram-positive organisms	15 (28.8)
Sputum Consistency	Mucoid	33 (33.7)
	Mucopurulent	32 (32.7)
	Purulent	33 (33.7)

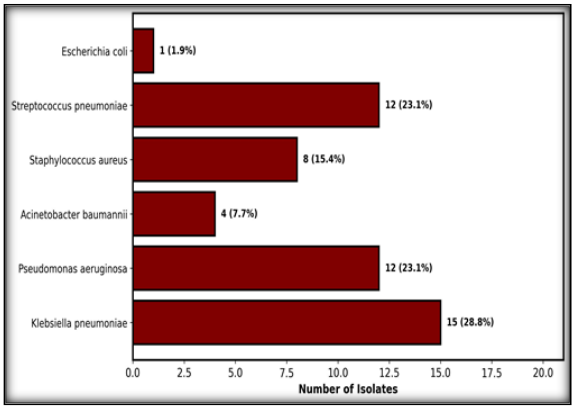

Figure 1: Distribution of bacterial isolates among culture positive cases (n=52)

Haematological, inflammatory and Spirometry parameters

The study population demonstrated elevated inflammatory and haematological indices, with mean NLR, ESR, and CRP values of 7.10 ± 3.64, 48.52 ± 20.95 mm/hr, and 40.02 ± 21.13 mg/L, respectively. The mean PDW, MPV, and platelet count were 14.16 ± 2.27 fL, 9.01 ± 1.21 fL, and 263.47 ± 51.31 $\times 10^3/\mu L$, respectively. This is shown in [Table 2 and Figure 2]. Spirometric evaluation revealed marked airflow obstruction, with a mean FEV_1 of 43.56 ± 14.20% predicted and a mean FEV_1 /FVC ratio of 46.52 ± 12.74%, consistent with moderate-to-severe airflow limitation among patients presenting with AECOPD.

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Table 2: Baseline Hematological, Inflammatory and Spirometric Parameters

Parameter	Mean ± SD	Median	Range
Neutrophil-to-Lymphocyte Ratio (NLR)	7.10 ± 3.64	6.20	2.1–14.9
Platelet Distribution Width (PDW), fL	14.16 ± 2.27	14.00	10.0–17.9
Mean Platelet Volume (MPV), fL	9.01 ± 1.21	9.00	6.1–11.9
Platelet Count (×10³/µL)	263.47 ± 51.31	261	151–391
Erythrocyte Sedimentation Rate (ESR), mm/hr	48.52 ± 20.95	48	16–89
C-Reactive Protein (CRP), mg/L	40.02 ± 21.13	35.10	5.2–79.4
FEV_1 (% predicted)	43.56 ± 14.20	—	23.1–77.8
FEV_1 /FVC (%)	46.52 ± 12.74	—	25.2–68.9

Antimicrobial susceptibility testing demonstrated the highest sensitivity to imipenem (80.8%), followed by meropenem (76.9%), piperacillin–tazobactam (73.1%), and amikacin (69.2%). In contrast, the highest resistance rates were observed for cotrimoxazole (69.2%), amoxicillin–clavulanate (65.4%), and azithromycin (61.5%) as shown in [Table 3 and Figure 3]. These findings indicate superior in vitro efficacy of carbapenems against bacterial isolates from patients with AECOPD, whereas commonly prescribed oral antibiotics exhibited comparatively higher resistance.

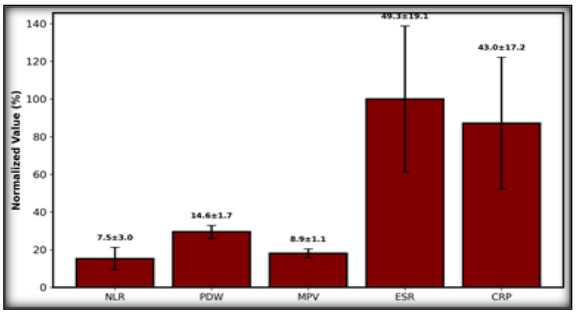

Figure 2: Haematological and inflammatory parameters

Table 3: Antibiotic Sensitivity Pattern (n=52)

Antibiotic	Sensitive n (%)	Resistant n (%)
Imipenem	42 (80.8%)	10 (19.2%)
Meropenem	40 (76.9%)	12 (23.1%)
Piperacillin-Tazobactam	38 (73.1%)	14 (26.9%)
Amikacin	36 (69.2%)	16 (30.8%)
Cefoperazone - Sulbactam	34 (65.4%)	18 (34.6%)
Levofloxacin	30 (57.7%)	22 (42.3%)
Gentamicin	28 (53.8%)	24 (46.2%)
Ciprofloxacin	26 (50.0%)	26 (50.0%)
Ceftriaxone	22 (42.3%)	30 (57.7%)
Azithromycin	20 (38.5%)	32 (61.5%)
Amoxicillin-Clavulanate	18 (34.6%)	34 (65.4%)
Cotrimoxazole	16 (30.8%)	36 (69.2%)

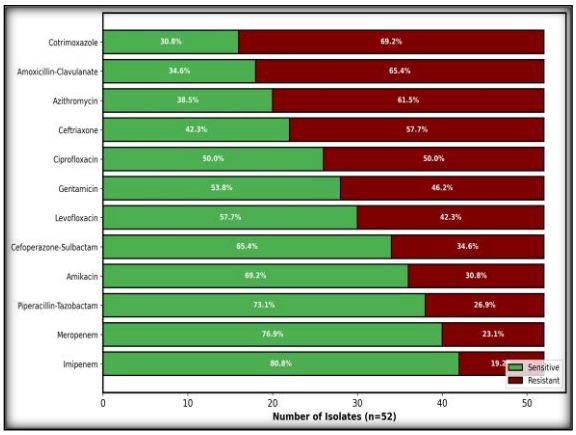

Figure 3: Antibiotic sensitivity pattern (n=52)

Hematological, inflammatory and spirometry values across AECOPD severity

There was a significant progressive increase in the mean values of NLR, PDW, ESR, and CRP with increasing AECOPD severity (all $p < 0.0001$). In contrast, the mean FEV_1 (% predicted) decreased significantly from 67.94 ± 5.05% in mild exacerbations to 29.84 ± 3.70% in severe exacerbations ($p < 0.0001$), reflecting worsening airflow limitation. However, MPV and platelet count did not differ significantly across severity groups ($p = 0.8822$ and $p = 0.2272$, respectively) as shown in [Table 4]. These findings indicate that inflammatory markers and selected haematological indices are significantly associated with increasing AECOPD severity, whereas MPV and platelet count are not useful indicators of disease severity.

Association of Clinical and Haematological Markers with AECOPD Severity

Smoking status and sputum culture positivity were significantly associated with AECOPD severity ($\chi^2 = 26.521$, $p < 0.0001$ and $\chi^2 = 15.064$, $p = 0.0005$, respectively). Significant positive correlations were observed between AECOPD severity and CRP ($\rho = 0.792$), ESR ($\rho = 0.742$), NLR ($\rho = 0.729$), and PDW ($\rho = 0.629$) (all $p < 0.0001$), whereas FEV_1 (% predicted) showed a strong negative correlation ($\rho = -0.924$, $p < 0.0001$). This is shown in [Table 5]. No significant correlation was observed for MPV ($\rho = 0.025$, $p = 0.8064$) or platelet count ($\rho = -0.186$, $p = 0.0671$). These findings indicate that smoking, sputum culture positivity, and inflammatory biomarkers are significantly associated with increasing AECOPD severity, while declining lung function demonstrates a strong inverse relationship with disease severity.

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Table 4: Haematological, Inflammatory and Spirometric Markers Across AECOPD severity, data are presented as mean ± SD

Parameter	Mild (n = 17)	Moderate (n = 41)	Severe (n = 40)	Test Statistic (F)	p-value
NLR	3.76 ± 1.30	6.43 ± 1.40	9.49 ± 2.92	53.869	<0.0001*
PDW (fL)	11.72 ± 1.23	13.81 ± 1.25	15.49 ± 1.37	41.333	<0.0001*
MPV (fL)	8.91 ± 1.08	9.05 ± 1.27	9.00 ± 1.20	0.126	0.8822
Platelet Count (×10³/μL)	277.65 ± 51.56	267.88 ± 49.54	253.78 ± 51.33	1.505	0.2272
ESR (mm/hr)	24.00 ± 5.68	43.39 ± 10.05	64.55 ± 14.52	78.161	<0.0001*
CRP (mg/L)	16.46 ± 8.55	33.26 ± 9.02	56.77 ± 13.28	89.476	<0.0001*
FEV_1 (% predicted)	67.94 ± 5.05	45.39 ± 5.87	29.84 ± 3.70	391.574	<0.0001*

Table 5: Association of Clinical and Haematological Markers with AECOPD Severity

Variable	Measure	Statistic	p-value	Interpretation
Smoking status vs AECOPD severity	Chi-square test	$\chi^2 = 26.521$	<0.0001*	Significant association
Sputum culture positivity vs AECOPD severity	Chi-square test	$\chi^2 = 15.064$	0.0005*	Significant association
NLR	Spearman's ρ	+0.729	<0.0001*	Strong positive correlation
CRP	Spearman's ρ	+0.792	<0.0001*	Strong positive correlation
ESR	Spearman's ρ	+0.742	<0.0001*	Strong positive correlation
PDW	Spearman's ρ	+0.629	<0.0001*	Moderate positive correlation
FEV_1 (% predicted)	Spearman's ρ	−0.924	<0.0001*	Strong negative correlation
MPV	Spearman's ρ	+0.025	0.8064	Not significant
Platelet count	Spearman's ρ	−0.186	0.0671	Not significant

DISCUSSION

The present study evaluated the clinical profile, bacteriological pattern, and haematological inflammatory indices in patients hospitalized with acute exacerbation of chronic obstructive pulmonary disease (AECOPD), and their association with disease severity. Most patients were older adults (mean age 60.27 ± 9.04 years), with a predominance of males (64.3%) and substantial smoking exposure. Similar demographic patterns have been reported by Sharan et al. and Gai et al.[9,10] Hypertension and diabetes mellitus were the commonest comorbidities, while frequent previous hospitalizations and recurrent exacerbations reflected the chronic and progressive nature of COPD.[11]

Sputum cultures were positive in 53.1% of patients, supporting bacterial infection as an important contributor to hospitalized AECOPD. This was comparable to findings by Jog et al. and Neelamma et al, although lower than the positivity reported by Talatam et al, possibly due to prior antibiotic exposure.[12,13,14] Gram-negative organisms predominated (71.2%), with Klebsiella pneumoniae, Pseudomonas aeruginosa, and Acinetobacter baumannii being the major isolates, consistent with previous studies.[9,14,15] Culture positivity was significantly associated with greater exacerbation severity and purulent sputum, supporting the role of bacterial infection in severe exacerbations.

The study demonstrated elevated inflammatory indices, including NLR, ESR, and CRP, reflecting marked systemic inflammation during acute exacerbations. Spirometry revealed severe airflow limitation, with a mean FEV_1 of 43.56 ± 14.20% predicted, consistent with advanced COPD among hospitalized patients. Similar observations have been reported by Neelamma et al, who described advanced airflow limitation in hospitalized AECOPD, while Yadav et al. reported higher inflammatory marker levels among severe inflammatory phenotypes.[13,16] These findings support the concept that acute exacerbations are characterized by both significant pulmonary dysfunction and systemic inflammatory activation.

Carbapenems showed the highest antimicrobial sensitivity, with imipenem and meropenem demonstrating sensitivity rates of 80.8% and 76.9%, respectively, followed by piperacillin–tazobactam and amikacin. Lower sensitivity to ceftriaxone, azithromycin, amoxicillin–clavulanate, and cotrimoxazole indicated substantial resistance to commonly used antibiotics. Similar patterns have been reported by Bannaravuri et al. and Mood et al.[15,17] These findings emphasize the importance of culture-guided therapy and antibiotic stewardship.

NLR, PDW, ESR, and CRP increased significantly with worsening exacerbation severity, whereas FEV_1 declined. CRP showed the greatest increase and the strongest positive correlation with severity, while FEV_1 demonstrated a strong inverse relationship. These findings are consistent with previous studies by Neelamma et al. and Yadav et al.[13,16] In contrast, MPV and platelet count showed no significant association with severity, suggesting limited utility as severity markers.

Smoking status, sputum culture positivity, and purulent sputum were significantly associated with more severe exacerbations. Correlation analysis further demonstrated positive associations of CRP, ESR, NLR, and PDW with disease severity, while FEV_1 showed a negative correlation.[9,12,18] Overall, CRP, ESR, NLR, and PDW, together with spirometric assessment, may serve as useful adjuncts for assessing the severity of hospitalized AECOPD.

The present study is strengthened by its comprehensive evaluation of the clinical profile, bacteriological spectrum, antibiotic susceptibility, haematological inflammatory markers, and spirometry parameters in hospitalized AECOPD patients within a single framework, allowing robust assessment of their association with exacerbation

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severity. However, the findings should be interpreted in light of certain limitations, including the single-centre cross-sectional design, relatively modest sample size, inclusion of only hospitalized patients, potential influence of prior antibiotic use on culture yield, lack of evaluation of viral etiologies and long-term outcomes, and measurement of inflammatory markers at a single time point, which may limit the generalizability and temporal interpretation of the results.

CONCLUSION

Overall, the findings of this study emphasize that acute exacerbations of COPD are multifactorial events influenced by smoking exposure, bacterial infections, systemic inflammation, and declining lung function. The integration of clinical evaluation, sputum bacteriology, and haematological markers can provide valuable insight into disease severity and help guide early management decisions. Identification of patients with severe inflammation and high bacterial burden may allow timely intervention, reduce complications, and improve treatment outcomes. Strengthening preventive measures such as smoking cessation, regular follow-up, vaccination, and early treatment of infections may help reduce the frequency and severity of exacerbations. The study highlights the importance of individualized and evidence-based management strategies in reducing the burden of hospitalization and improving quality of life in patients with chronic obstructive pulmonary disease.

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