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CLINICAL PROFILE AND OUTCOME OF PATIENTS WITH ACUTE EXACERBATION OF CHRONIC OBSTRUCTIVE PULMONARY DISEASE: A PROSPECTIVE OBSERVATIONAL STUDY

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ABSTRACT

Background: Acute exacerbation of chronic obstructive pulmonary disease is a common cause of hospitalization and poor clinical outcome among patients with chronic respiratory illness.

Aim: To evaluate the clinical profile, associated risk factors, precipitating factors, comorbidities, and outcome of patients admitted with acute exacerbation of chronic obstructive pulmonary disease.

Materials and Methods: A prospective observational study was conducted among 132 patients admitted with acute exacerbation of COPD. Clinical history, smoking status, risk factors, comorbidities, radiological findings, oxygen status, ventilatory support, and outcome were analyzed using SPSS version 16.

Results: Most patients were elderly males. Smoking was the most common risk factor, and respiratory tract infection was the most common precipitating factor. Breathlessness and productive cough were the predominant symptoms. Hypoxemia, prolonged hospital stay, multiple comorbidities, and long-term smoking were significantly associated with poor outcome.

Conclusion: Acute exacerbation of COPD commonly affects elderly smokers and is frequently precipitated by respiratory infection. Early recognition, prompt treatment, oxygen monitoring, and smoking cessation are essential to improve outcome.

Keywords

Acute Exacerbation, Chronic Obstructive Pulmonary Disease, Clinical Profile, General Medicine, Respiratory Infection, Smoking.

INTRODUCTION

Chronic obstructive pulmonary disease is a progressive respiratory disorder characterized by persistent airflow limitation that is usually not fully reversible. It is associated with chronic inflammatory response of the airways and lung parenchyma to harmful particles or gases. COPD is an important cause of morbidity and mortality worldwide and contributes significantly to hospital admissions, loss of productivity, reduced quality of life, and healthcare expenditure. The disease commonly develops gradually over several years and often becomes clinically apparent in middle-aged and elderly individuals.[1]

Acute exacerbation of chronic obstructive pulmonary disease refers to an acute worsening of respiratory symptoms beyond normal day-to-day variation that requires additional treatment. Exacerbations are clinically important because they accelerate decline in lung function, worsen health status, increase risk of hospitalization, and contribute to mortality. Patients with frequent exacerbations often have poorer quality of life and greater future risk of repeated admissions.[2]

Smoking remains the most important risk factor for development and progression of COPD. Cigarette smoke causes chronic airway inflammation, mucus hypersecretion, ciliary dysfunction, small airway narrowing, and destruction of alveolar walls. The risk increases with duration and intensity of smoking exposure. However, COPD may also occur in

non-smokers due to biomass fuel exposure, occupational dust exposure, outdoor pollution, recurrent childhood respiratory infections, and genetic susceptibility.[3]

Patients with acute exacerbation commonly present with worsening breathlessness, increased cough, increased sputum volume, change in sputum purulence, wheezing, chest tightness, fever, and reduced exercise tolerance. In severe cases, patients may develop hypoxemia, hypercapnia, altered sensorium, respiratory failure, or need for ventilatory support. Clinical assessment should focus on severity of dyspnea, respiratory rate, use of accessory muscles, cyanosis, oxygen saturation, hemodynamic status, and presence of comorbidities.[4]

Respiratory tract infection is one of the most common precipitating factors for exacerbation. Both bacterial and viral infections can increase airway inflammation, mucus production, bronchospasm, and gas exchange abnormality. Environmental pollution, poor medication compliance, cold climate, and exposure to irritants may also precipitate exacerbation. Identifying the precipitating factor is important because management may include bronchodilators, corticosteroids, antibiotics when indicated, oxygen therapy, and ventilatory support in severe cases.[5]

Outcome in AECOPD depends on baseline lung function, age, smoking history, hypoxemia, hypercapnia, comorbid illnesses, severity of exacerbation, delayed presentation, and need for ventilatory support. Early identification of severe exacerbation and prompt management are important to reduce morbidity and mortality. The present study was conducted to evaluate the clinical profile, associated risk factors, precipitating factors, comorbidities, and outcome of patients admitted with acute exacerbation of chronic obstructive pulmonary disease.[6]

MATERIALS AND METHODS

This prospective observational study was conducted in the Department of General Medicine of a tertiary care teaching hospital. A total of 132 patients admitted with acute exacerbation of chronic obstructive pulmonary disease were included in the study.

Patients above 40 years of age diagnosed with COPD and presenting with acute worsening of respiratory symptoms were included. Patients with bronchial asthma, pulmonary tuberculosis, interstitial lung disease, bronchiectasis as the primary diagnosis, lung malignancy, and incomplete clinical records were excluded. These exclusion criteria were applied to maintain a uniform study population of acute COPD exacerbation and to avoid conditions that could independently produce similar respiratory symptoms.

A detailed clinical history was recorded for all patients. Information regarding age, gender, smoking status, duration of illness, occupational dust exposure, biomass fuel exposure, previous respiratory infection, previous hospitalization, medication compliance, and associated comorbidities was obtained. Smoking history was specifically documented, including duration of smoking and long-term exposure.

Clinical evaluation included general physical examination and detailed respiratory system examination. Presenting symptoms such as breathlessness, productive cough, wheezing, fever, and chest pain were recorded. Oxygen saturation was measured by pulse oximetry. Chest radiography was performed to assess hyperinflation, infection, consolidation, cardiomegaly, or other associated radiological findings. Arterial blood gas analysis was performed wherever clinically indicated, especially in patients with hypoxemia, severe respiratory distress, or suspected respiratory failure.

Precipitating factors for exacerbation were categorized as respiratory tract infection, environmental pollution, noncompliance to medication, and unknown cause. Associated comorbidities including hypertension, diabetes mellitus, ischemic heart disease, and cor pulmonale were documented.

Treatment outcome was assessed in terms of improvement and discharge, requirement of ventilatory support, and mortality. Poor outcome was analyzed in relation to hypoxemia, hospital stay more than seven days, multiple comorbidities, and smoking history more than 20 years.

Data were entered into a structured data sheet and analyzed using SPSS software version 16. Categorical variables were expressed as frequencies and percentages, while continuous variables were represented as mean ± standard deviation wherever applicable. Chi-square test and Student’s t-test were used for statistical analysis. A p-value less than 0.05 was considered statistically significant.

Institutional ethical committee approval was obtained before commencement of the study. Informed consent was obtained from all participants before enrolment, and confidentiality of patient information was maintained throughout the study.

RESULTS

A total of 132 patients with acute exacerbation of chronic obstructive pulmonary disease were evaluated.

Table 1: Demographic Characteristics of Study Participants

Variable	Frequency (%)
Male	94 (71.2)
Female	38 (28.8)
Age 41–50 years	24 (18.2)
Age 51–60 years	48 (36.4)
Age >60 years	60 (45.4)

Male predominance was observed, with 94 (71.2%) male patients and 38 (28.8%) female patients. The majority of participants belonged to the age group above 60 years, accounting for 60 (45.4%) cases. This indicates that acute exacerbation of COPD was more common among elderly male patients in the present study.

Table 2: Risk Factors Among Study Participants

Risk Factor	Frequency (%)
Smoking	96 (72.7)
Biomass fuel exposure	34 (25.8)
Occupational dust exposure	28 (21.2)
Previous respiratory infection	52 (39.4)

Smoking was the most common associated risk factor, observed in 96 (72.7%) patients. Previous respiratory infection was present in 52 (39.4%) cases. Biomass fuel exposure and occupational dust exposure were also important contributing factors.

Table 3: Clinical Presentation Among Study Participants

Symptom	Frequency (%)
Breathlessness	126 (95.5)
Productive cough	104 (78.8)
Wheezing	88 (66.7)
Fever	42 (31.8)
Chest pain	18 (13.6)

Breathlessness was the most common presenting symptom, observed in 126 (95.5%) patients. Productive cough was present in 104 (78.8%) patients, followed by wheezing in 88 (66.7%) cases. Fever was present in nearly one-third of patients, suggesting infective exacerbation in a considerable proportion.

Table 4: Precipitating Factors for Exacerbation

Precipitating Factor	Frequency (%)
Respiratory tract infection	74 (56.1)
Environmental pollution	22 (16.7)
Noncompliance to medication	18 (13.6)
Unknown cause	18 (13.6)

Respiratory tract infection was the most common precipitating factor, observed in 74 (56.1%) patients. Environmental pollution and noncompliance to medication were also identified as important triggers. In 18 (13.6%) patients, no definite precipitating factor was identified.

Table 5: Associated Comorbidities Among Study Participants

Comorbidity	Frequency (%)
Hypertension	38 (28.8)
Diabetes mellitus	26 (19.7)
Ischemic heart disease	20 (15.2)
Cor pulmonale	18 (13.6)

Hypertension was the most common associated comorbidity, observed in 38 (28.8%) patients, followed by diabetes mellitus in 26 (19.7%) cases. Ischemic heart disease and cor pulmonale were also noted and are clinically important because they may worsen respiratory and overall outcome.

Table 6: Treatment Outcome Among Study Participants

Outcome	Frequency (%)
Improved and discharged	108 (81.8)

Required ventilatory support	18 (13.6)
Mortality	6 (4.6)

Most patients improved with treatment and were discharged successfully. Ventilatory support was required in 18 (13.6%) cases, indicating severe exacerbation with respiratory compromise. Mortality was observed in 6 (4.6%) patients.

Table 7: Factors Associated with Poor Outcome

Variable	Poor Outcome n (%)	p-value
Hypoxemia	16 (66.7)	0.002
Hospital stay >7 days	14 (58.3)	0.006
Multiple comorbidities	12 (50.0)	0.011
Smoking history >20 years	18 (75.0)	0.004

Hypoxemia, prolonged hospital stay, multiple comorbidities, and long-term smoking demonstrated significant association with poor outcome. Smoking history of more than 20 years showed the highest association with poor outcome in the present study.

DISCUSSION

The present prospective observational study evaluated the clinical profile, risk factors, precipitating factors, comorbidities, and treatment outcome among 132 patients admitted with acute exacerbation of COPD. The study showed that AECOPD was more common among elderly males, smoking was the predominant risk factor, respiratory tract infection was the most common precipitating factor, and hypoxemia was significantly associated with poor outcome.

Male predominance was observed in the present study. Mannino reported that COPD prevalence is strongly influenced by smoking patterns, occupational exposure, and age distribution.[7] In the present study, males constituted 71.2% of cases. This finding may be explained by higher smoking exposure among males and greater occupational exposure to dust, fumes, and outdoor pollutants.

The majority of patients were above 60 years of age. COPD is a chronic progressive disease, and clinical severity usually increases with advancing age and longer exposure to risk factors. The present study showed that 45.4% of patients were older than 60 years. Elderly patients are more likely to have reduced pulmonary reserve, frequent exacerbations, associated comorbidities, and increased need for hospitalization.

Smoking was the most common risk factor. Barnes described cigarette smoke as a major cause of chronic airway inflammation, mucus hypersecretion, protease-antiprotease imbalance, oxidative stress, and progressive airflow limitation.[8] In the present study, 72.7% of patients had a history of smoking, supporting the strong association between tobacco exposure and COPD exacerbation burden.

Biomass fuel exposure was present in 25.8% of patients. This is clinically important in Indian settings, where indoor air pollution from biomass fuel may contribute to chronic respiratory disease, especially among women. Occupational dust exposure was also present in 21.2% patients. These findings indicate that COPD prevention should not focus only on smoking cessation but also on reducing environmental and occupational exposures.

Breathlessness was the most common presenting symptom, followed by productive cough and wheezing. Seemungal et al. demonstrated that COPD exacerbations significantly affect symptoms and health-related quality of life.[9] The present study similarly found breathlessness in 95.5% of patients and productive cough in 78.8%. These symptoms reflect increased airway obstruction, mucus production, and impaired gas exchange during exacerbation.

Respiratory tract infection was the most common precipitating factor. Sethi highlighted that infectious agents play a major role in acute exacerbations of COPD, with bacteria and viruses contributing to airway inflammation and symptom worsening.[10] In the present study, respiratory infection was identified in 56.1% of cases. Fever, purulent sputum, and radiological changes may support infective exacerbation and guide antibiotic use where appropriate.

Noncompliance to medication was observed in 13.6% patients. Poor adherence to inhaled bronchodilators, corticosteroids when indicated, smoking cessation advice, and follow-up care may increase exacerbation risk. Patient education regarding inhaler technique, medication adherence, vaccination, early recognition of worsening symptoms, and timely hospital visit is essential in long-term COPD management.

Hypertension was the most common comorbidity, followed by diabetes mellitus, ischemic heart disease, and cor pulmonale. COPD frequently coexists with systemic comorbidities due to shared risk factors such as age, smoking, chronic inflammation, and reduced physical activity. Comorbidities may increase hospitalization duration, complicate treatment, and worsen prognosis.

Hypoxemia was significantly associated with poor outcome. Connors et al. reported that severe COPD exacerbation requiring hospitalization is associated with poor prognosis, particularly in patients with respiratory failure and significant physiological derangement.[11] In the present study, hypoxemia was present in 66.7% of poor outcome cases. Oxygen saturation monitoring and arterial blood gas analysis are therefore important in acute assessment.

Ventilatory support was required in 13.6% of patients. Non-invasive ventilation has an important role in selected patients with acute exacerbation and respiratory acidosis. Brochard et al. showed that non-invasive ventilation can reduce intubation requirement and improve outcomes in acute exacerbations of COPD.[12] Patients with severe dyspnea, hypercapnia, acidosis, or respiratory muscle fatigue require close monitoring and timely ventilatory support.

Multiple comorbidities were significantly associated with poor outcome. Patients with COPD and cardiovascular disease may have overlapping symptoms such as dyspnea and chest discomfort. Ischemic heart disease and cor pulmonale increase the risk of decompensation during exacerbation. In such patients, treatment should include careful fluid management, oxygen therapy, bronchodilator use, infection control, and monitoring for cardiac complications.

Prolonged hospital stay was significantly associated with poor outcome. Patients requiring hospitalization for more than seven days likely had severe exacerbation, persistent hypoxemia, infection, comorbidities, or delayed recovery. Wedzicha described exacerbations as important events that increase healthcare burden and contribute to disease progression.[13] The present study supports this observation, as prolonged hospital stay was associated with poor outcome.

Long-term smoking history of more than 20 years showed significant association with poor outcome. Anthonisen et al. reported that smoking cessation has a major beneficial effect on lung function decline and COPD progression.[14] The present finding emphasizes the importance of smoking cessation counselling at every hospital visit. Even after COPD diagnosis, stopping smoking can reduce further decline and exacerbation risk.

Mortality was observed in 4.6% patients. Mortality in AECOPD is often related to severe hypoxemia, hypercapnia, respiratory failure, pneumonia, sepsis, cardiac comorbidities, and delayed presentation. Early recognition of danger signs such as altered sensorium, cyanosis, severe breathlessness, hypotension, silent chest, and low oxygen saturation is essential for escalation of care.

Management of AECOPD requires a comprehensive approach. Short-acting bronchodilators, systemic corticosteroids, controlled oxygen therapy, antibiotics when indicated, hydration, physiotherapy, and ventilatory support are important treatment components. Careful oxygen titration is necessary because excessive oxygen administration may worsen hypercapnia in susceptible patients.

Preventive strategies are equally important after discharge. Patients should receive smoking cessation advice, inhaler technique training, vaccination guidance, pulmonary rehabilitation advice, and follow-up planning. Those with frequent exacerbations may need optimization of maintenance therapy. Education regarding early symptoms of exacerbation can reduce delayed presentation.

CONCLUSION

Acute exacerbation of chronic obstructive pulmonary disease commonly affected elderly male smokers in the present study. Smoking was the most common associated risk factor, and respiratory tract infection was the most frequent precipitating factor. Breathlessness, productive cough, and wheezing were the predominant symptoms. Hypertension, diabetes mellitus, ischemic heart disease, and cor pulmonale were common comorbidities. Most patients improved and were discharged, but ventilatory support was required in a significant proportion, and mortality occurred in a small number of patients. Hypoxemia, prolonged hospital stay, multiple comorbidities, and long-term smoking were significantly associated with poor outcome. Early recognition, prompt treatment, oxygen monitoring, infection control, ventilatory support when indicated, and smoking cessation are essential for improving clinical outcomes in patients with AECOPD.

REFERENCES

1. American Thoracic Society. Standards for the diagnosis and care of patients with chronic obstructive pulmonary disease. Am J Respir Crit Care Med. 1995;152:S77-S121.
2. Rodriguez-Roisin R. Toward a consensus definition for COPD exacerbations. Chest. 2000;117:398S-401S.
3. Burge S, Wedzicha JA. COPD exacerbations: definitions and classifications. Eur Respir J Suppl. 2003;41:46S-53S.
4. Seemungal TA, Donaldson GC, Paul EA, Bestall JC, Jeffries DJ, Wedzicha JA. Effect of exacerbation on quality of life in patients with chronic obstructive pulmonary disease. Am J Respir Crit Care Med. 1998;157:1418-1422.
5. Sethi S. Infectious etiology of acute exacerbations of chronic bronchitis. Chest. 2000;117:380S-385S.

6. Pauwels RA, Buist AS, Calverley PMA, Jenkins CR, Hurd SS. Global strategy for the diagnosis, management, and prevention of chronic obstructive pulmonary disease. Am J Respir Crit Care Med. 2001;163:1256-1276.
7. Mannino DM. COPD: epidemiology, prevalence, morbidity and mortality, and disease heterogeneity. Chest. 2002;121(5 Suppl):121S-126S.
8. Barnes PJ. Chronic obstructive pulmonary disease. N Engl J Med. 2000;343:269-280.
9. Seemungal TA, Donaldson GC, Paul EA, Bestall JC, Jeffries DJ, Wedzicha JA. Effect of exacerbation on quality of life in patients with chronic obstructive pulmonary disease. Am J Respir Crit Care Med. 1998;157:1418-1422.
10. Sethi S. Infectious etiology of acute exacerbations of chronic bronchitis. Chest. 2000;117:380S-385S.
11. Connors AF Jr, Dawson NV, Thomas C, Harrell FE Jr, Desbiens N, Fulkerson WJ, et al. Outcomes following acute exacerbation of severe chronic obstructive lung disease. Am J Respir Crit Care Med. 1996;154:959-967.
12. Brochard L, Mancebo J, Wysocki M, Lofaso F, Conti G, Rauss A, et al. Noninvasive ventilation for acute exacerbations of chronic obstructive pulmonary disease. N Engl J Med. 1995;333:817-822.
13. Wedzicha JA. Exacerbations: etiology and pathophysiologic mechanisms. Chest. 2002;121(5 Suppl):136S-141S.
14. Anthonisen NR, Connett JE, Kiley JP, Altose MD, Bailey WC, Buist AS, et al. Effects of smoking intervention and inhaled bronchodilator on the rate of decline of FEV1. JAMA. 1994;272:1497-1505.