ORIGINAL RESEARCH PAPER

Respiratory Medicine

FREQUENT EXACERBATION OF COPD: THE CLINICAL EVALUATION OF THE RISK FACTOR

KEY WORDS: ABG, AE (acute exacerbation), Bronchio-alveolar lavage (BAL), PRISM, sequelae of COPD, SOB (Shortness of breath).

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ABSTRACT

Introduction: COPD is the number 3rd killer of mankind from noninfectious disease. In the majority, the etiological factor is smoke and dust. The environmental factor, seasonal variation and usually the bacterial or viral infection leads to the AECOPD have been identified with increase of SOB and sputum volume with its purulence. The variant or sequelae of COPD is not uncommon. **Result:** The advance age of ≥50, male sex (93%), bacterial infection i.e. 14% each with P aeruginosa and Klebsiella followed by S pneumonae 5% etc remained the main cause of AECOPD. The associated co morbidity of TB (24%), DM (22%), asthma (22%) and HTN (18%) etc with low serum sodium in 41% and an increased CRP in all the cases were also observed. The 8% mortality recorded especially among the mechanical ventilator patient and with longer hospital stay. **Discussion:** There are certain variables in defining acute exacerbation of COPD. A simplified and clinical utility definition: Type 1 exacerbation's by the presence of increased SOB, sputum volume and sputum purulence; type 2 by the presence of two of these symptoms, and type 3 by the presence of one of these symptoms is more practical. The increase of SOB alone is a strong predictor that can be monitored with spirometry. AECOPD is a complex phenomenon that how the body system interact with V/Q mismatch. and haemodynamic instability hence a close monitoring of vitals, ABG, inflammatory markers with pharma and non pharmacological management is crucial. **Conclusion:** The damaged lung in COPD is largely irreversible but likely to

INTRODUCTION:

Chronic obstructive pulmonary disease (COPD) is the number 3rd leading cause of death world wide [1]. It is an obstructive disease among the group of diseases like emphysema, chronic bronchitis, bronchiectasis, cystic fibrosis, bronchial asthma etc. It is mainly concern with the airways abnormalities that leads to difficult air flow, however it does not mean that the vascular and other part of the lung are spared. The sequelae of COPD has a wide range of systemic inflammation which involve other organ system for example the corpulmonale, cardiovascular system, musculo skeletal etc. The hypoxia, hypercapnia and respiratory failures lead adifferent forms of COPD i.e. Blue bloater or Pink puffer, combined pulmonary fibrosis and emphysema (CPFE), asthma COPD overlap syndrome (ACOS), eosinophillic phenotype and bullous COPD. Similarly the co-existing other diseases (comorbidities) are also closely interrelated and are to worsen each other like PTB, CAD, DM, systemic HT, PAH, bronchial asthma, HIV etc. The COPD starts with a prolong environmental insults i.e. occupational/ tobacco smoking or other etiological factors (i.e. genetic) and with a continued trigger it attains a full blown stage usually by the age of 50 yrs. The most common presentation of COPD is dyspnea and decrease of lung functional capacity which can be assessed with pulmonary function test and other reproducible monitoring tools. However the enthusiastic smoking beginners may be detected quite earlier with PRISM (preserved ratio impaired spirometry) during their usual/ seasonal lower respiratory tract infection [2]. A stable COPD gradually progress to become a full blown disease and continue to be symptomatic through out the year.

The environmental, occupational, air pollution, seasonal variation, and certain other trigger factors and most of the time viral or bacterial infection can precipitate acute exacerbation of COPD (AE-COPD). It is defined (GOLD 2026) as "a heterogeneous lung condition characterized by chronic respiratory symptoms (dyspnea, cough, sputum production and/or exacerbations) due to abnormalities of the airways (bronchitis, bronchiolitis) and/or alveoli (emphysema) that cause persistent, often progressive, airflow obstruction" [1]. Each episode of AECOPD is well recognized with increased in breathlessness and sputum volume with its purulence. However the frequency and magnitude of these episodes can also be quantified with available tools because every episode of AECOPD is likely to impact the progression of disease. Hence it become important to recognize or identify and prevent the episodes of acute exacerbation. The present study is plan to understand and explore the possible reasons (trigger) in order to decrease repeated exacerbation, that enable prompt therapeutic intervention/ strategy to achieve goals of management.

Table 1: Profile of Patients N=100

Indicators	%	Inference
AGE Wise		A steep rise after
51- 60	26	may have different
<= 50 Yrs	17	50 yr of age. Younger sub set
61-70	44	etiologic factors.
>70 Yrs	13	
SEX Wise		Male predilection

Male	93	accepted world wide
Female	07	
Rural	88	Maximum were of
Urban	12	rural background
Schooling		55% even not
Nil	55	completed primary education
Primary	44	
Higher	01	
Occupation		Triggers: seasonal
Farmer	67	changes, humidity, pollen, dust, mold..
Other	33	
Smoker	73	73% were present
Alcohol	44	smoker and 10% were past.
Tobacco		
Chewer	29	
Medication Discontinue Regular	70 30	More in defaulters 48/70=69% 12/30= 40%

MATERIAL AND METHOD

An observational, prospective study was carried out in patients admitted with AECOPD in the Department of Respiratory Medicine (C.R. Gardi Hospital), for a period of 12 months

during the year 2023-24. Data were statistically described in terms of mean, standard deviation, or frequencies (number of cases) and percentages when appropriate. The statistical calculations were done using computer program SPSS (Statistical Package for the Social Science; SPSS Inc., Chicago, IL, USA) version 21 for Microsoft Windows; and For comparing categorical data, Chi square test was performed. P values less than 0.05 was considered statistically significant.

Results

A total of 438 patients during the period were admitted to the RICU and among them 186 (43.8%) were of AE-COPD. However a total of 100 recurrent cases were finally selected for the analysis and the patients profile is as under (Table 1). The vitals and signs symptoms are in (Table-2). The COPD is more prevalent among advance aged (mean 62.9 ± 8.079 years in our study) and the male sex and in current smokers as is unanimously accepted worldwide.

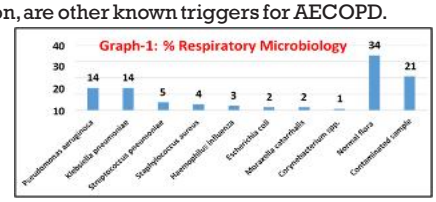
Table 2:Vitals & Signs

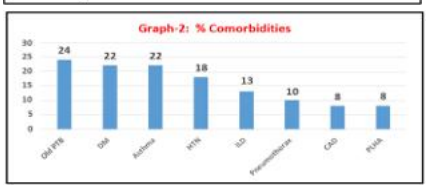
Indicators	%	Inference
Pulse > 100	 85	To be correlate with other signs.
BP <90 > 140	 03 23	To be correlate with other indicators
Respi Rate 20-25 25-30 >30	 20 77 03	All had shortness of breath, leads to increase work of breath and fatigue.
SpO2 91-95 80-90 <80	 26 51 23	All had hypoxia and in need of O2 therapy with ABG status
Respi Failure Type 1 Type 2	 26 74	Hypercapnic Respi failure pronounced
PN Drip	37	Suggestive of URI
Barrel chest	82	Emphysema
Febal breath	78	Low VT
BB Sound	03	Pneumonia
Crepts	38	Infection
Rhonchi	82	Broncho-spasm
Pleural rub	02	pleuritis
Hb gm % <10 gm >16.9 gm	 06 02	Corrective action may require.
TLC>12 K	56	Anti microbial
PLT <1.5 Lakh >4,5 Lakh	 09 07	Coagulation and fever profile may require.
Creatinine > 1.5	 10	Renal profile required
Serum Na < 135	 41	Correction needed
Serum K < 3.5 > 5	 12 19	Corrective action is required
FBS >140	28	DM detected
CRP >10	100	Raised in all cases
CKmb>20	0	ECG, 2D ECHO,
HsTropI>15	0	may require.
D-dimer 500-1000 >1000 ng/ml	 20 80	All were without significant clinical evidences.

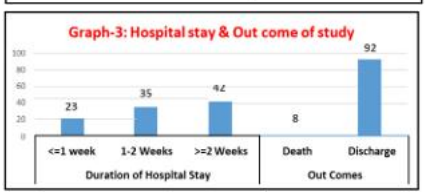
Those who defaulted on their regular prescribed medication (70%) were more vulnerable to frequent attack of AE 69% (48/70), as against only 40% (12/30) among the 30 regular treatment patients. The old treated PTB sequelae cases were 24%, DM and asthma each were 22% and HT (18%) etc were more prone to get AE. The microbiology examination detected P aeruginosa and Klebsiella in (14%) followed by S pneumonae (5%), Staphylococcus aureus (4%), Haemophilus influenza (3%) etc however 55% samples were of normal or contaminated flora (Graph-1). An in hospital mortality among AECOPD remained 18/186 (9.7%). Mortality increases with a patient on ventilator, longer hospital stay and associated co-morbidity (Graph-3).

DISCUSSION:

The industrial and vehicle pollution is predominantly found in the cities but in our presentation most of the patients belonged to the (88%) rural based farming job with exposure to dusty and unhygienic environment. Tobacco smoking is universally accepted an etiologic factor found in 73% of cases in our study and an addition use of bio fuel and house dust in the rural area is most likely to further 'fuel' the COPD. The farming job, humidity, exposure to pollen, mold and seasonal variation, are other known triggers for AECOPD.







There are certain variables in defining acute exacerbation of COPD. B. WAGECK et al. had mentioned that two consecutive days with an increase in at least two major signs and/or symptoms; or the presence of one major symptom together with a minor sign and/or symptom is defined as AECOPD [3]. The recovery from an AECOPD has been defined as two

consecutive days without symptoms; or, a return of symptoms to their base line level. However Sunil K. Chhabra has mentioned a simplified and clinical utility definition: Type 1 exacerbation's by the presence of increased SOB, sputum volume and sputum purulence; type 2 by the presence of two of these symptoms, and type 3 by the presence of one of these symptoms [4]. The increase of SOB alone is a strong predictor that can be monitored with spirometry. Kshatriya RM et al, [5] mentioned difference in FEV1 of 43.98% and 29.28% in ward and among ICU patients with AECOPD, similarly a difference of 93% and 88.62% respectively wasobserved in oxygen saturation.

The culture and sensitivity test for the sputum/ BAL and blood must be included as protocol and our study too had detected 45 % cases with bacterial or viral infection. A higher yield of 78.94% & 73.65% with BAL and sputum sample was reported by Anand A et al. [6] and M.F. Aleemullah et al. [7], but it is not always feasible to collect protected lower respiratory sample with FOB during acute phase and that could be the reason for lower yield in the present study. A variable isolates were detected in different studies: Haemophilus influenzae 34.86% and Streptococcus pneumonia 22.94% (14 of RL-1), Klebsiella pneumoniae (38%) followed by Staphylococcus aureus (18%) reported by Singh et al. [8] and our study too had presented 14% each with P aeruginosa and klebsiella. The sensitivity report and past history of drug intake to be considered however; Sunil K Chhabra et al recommended precise use of antibiotics for type I exacerbation [4]. The seasonal common cold (viral) with or without secondary infection is the most common/ usual trigger/ cause of AECOPD and normally does not require antibiotic but a susceptible individual with recurrent exacerbation should be provided a prophylactic cover. The inflammatory indicators and eosiniphilic phenotype will determine the management of AECOPD hence an inhaled with or without systemic steroid may be used under the umbrella of antibiotics likely to prevent from the complication of secondary infection.

Our study observed substantial number with electrolytes imbalance and haematological anormalities at admission that include raised CRP in all the cases (Table-2). Kumar H, Choubey S. concluded that a low blood pH, high PaCO2, SGPT and random blood sugar and low serum sodium, at admission are independent predictors to increased mortality in AECOPD [9]. Similarly Cojocaru E et al [10] studied 104 patients in ICU setting and reported 65 (62.5%) death and emphasized the importance of CRP, neutrophil to lymphocyte ratio (NLR), monocyte to lymphocyte ratio (MLR) and systemic immune-inflammation index SII in assessing high risk of mortality.

SujathaG et al. insisted an early consideration of advanced age, history of smoking, diabetes, low FEV1 volume, high PaCO2 levels to reduce the morbidity and mortality [11]. The AECOPD is a complex phenomenon of how the body system interact with V/Q mismatch.and haemodynamic instability during AECOPD, the effect of mechanical ventilator with duration of hospital stay (Graph-3). The present study considered most of the above mentioned parameters and it was found that the mortality remained concise/ limited to 8% (Table-3 & Graph-3). The presence of co-morbidities further accelerate the acute episodes. Present study associated with old PTB (24%), DM (22%), asthma (22%) etc (Graph 2).

The 8% cases presented with pneumothorax as a complication of COPD, hence an appropriate regular supportive care plan which is followed by rehabilitation programme is thus insisted. The Sunil K Chhabra et al. also cautioned about certain acute conditions in the differential diagnosis for SOB as pneumothorax, left ventricular failure, pulmonary thrombus-embolism which may coincide the COPD [3]. Chakrabarty S et al. [12] had mentioned that AECOPD is a pro-inflammatory state that generates or reactivate cytokines which distort coagulation profile and can lead to intra vascular thrombosis and occlusion of blood flow with clinical worsening. However >1000 D-dimer it was observed that in 80% of our cases without any significant clinical evidence of coagulopathy.

Table 3:

Indicators	%	Inference
Spirometry		FEV1 Values N=45
Mild Gold 1	00	GOLD 1 (70 -79)
moderate	06	GOLD 2 (60-69)
Sever	21	GOLD 3 (40-59)
Very severe	18	GOLD 4 (< 40)
unable	55	Could not perform
DLCO	0	Pending
6MWT		Simple reliable and
<200 M	09	installationfree toolto assess
201-400M	40	functional capacity
>400 M	25	
HDU	31	High Dependency
RICU	69	Unit in RICU
Intubation	28	Optimize FiO2,
NIV support	47	PEEP: maintained
O2 support	25	PaO2 >60mm
Hospital stay		Effect the out come shorter is the best

CONCLUSION:

A complete evaluation of AECOPD with a thorough history, clinical, radiological, inflammatory bio-markers, micro-biology and stratification of the episode with ABG analysis, spirometry etc is necessary to detect the trigger as well as etiological factors. The ultimate aim should be to prevent further episode and improve the quality of life

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