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Pulmonary Medicine

Identification of Causative Bacteria and Sensitivity Pattern to Antibiotics in AE-COPD

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Original Research Article

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Abstract: COPD is a common preventable and treatable disease characterised by persistent airfloe limitation that is usually progressive and associated with an enhanced inflammatory response. Acute Exacerbation of chronic obstructive pulmonary disease is defined as an acute change in a patient's baseline dyspnea, cough and /or sputum beyond day-to-day variability sufficient to warrant change in therapy. Exacerbation may be triggered by infection with bacteria or viruses or by environmental pollutants. Typically infections cause 75% of exacerbations of which 25%are due to bacteria, 25%are due to virus and 25% are due to both virus and bacteria. The aim of this study was to identify the bacteria and their sensitivity pattern to antibiotics in AE-COPD.This was a cross sectional study conducted on 50 patients with acute exacerbations of COPD who got admitted in government hospital .the study group and comprised of both sexes with their age ranging from 40 to 86 years .all patients who got admitted because of acute exacerbation of COPD were included excluding those with known PTB ,Bronchial Asthma, Lung Cancer, Pneumonia, Bronchiectasis, Lung Abscess and Ischemic heart disease and those patients who already received antibiotics before admission were also excluded. Early morning samples of sputum were collected and cultured on 5%SHEEP BLOOD AGAR as enriched medium, Macconkey's medium as differential medium and Levinthal's medium. After overnight incubation, sensitivity testing was done for various antibiotics. In the study population 26 (56%) patients had Gram negative organisms 14(28%) patients had Gram positive organisms and 10 (20%) had normal flora. Most of the Gram negative bacteria showed susceptibility to cephalosporins, aminoglycosides, and quinolones. Streptococcus pneumonia and Staph aureus showed susceptibility to ceftriaxone, cefoperazone-sulbactam, piperacillin-tazobactam. Bacterial pathogens particularly Gram negative organisms are the chief etiological agents in AE-COPD. Empirical antibiotic therapy should be initiated early based on sensitivity pattern of that particular region to reduce antibiotic resistance, healthcare costs, morbidity and mortality.

Keywords: COPD Acute Exacerbation, Gram positive Gram Negative bacteria, Culture, Blood Agar,Macconkey's medium, Antibiotics, Cephalosporins, Quinolones, Aminoglycosides, Piperacillin-Tazobactam.

INTRODUCTION

Chronic obstructive pulmonary disease [COPD] a common preventable and treatable disease is characterised by persistent airflow limitation that is usually progressive and associated with an enhanced chronic inflammatory response in the airways and the lungs to noxious particles or gases[1].

COPD is a major cause of chronic morbidity and mortality throughout the world; many people suffer from this disease for years, and die prematurely from it or its complications. Globally, the COPD burden is projected to increase in coming decades because of continued exposure to COPD risk factors and aging of the population [2].

Cigarette smoking forms the single mostimportant risk factor for development of COPD. The clinical manifestations of COPD include dyspnea, cough, sputum production and impaired exercise tolerance. The clinical course of COPD is generally one of gradual progressive impairment, which may eventually lead to respiratory failure. Periods of relative clinical stability are interrupted by recurrent exacerbations [3].

Acute exacerbation of chronic obstructive pulmonary disease (AECOPD) is defined as an acute

change in a patient's baseline dyspnea, cough and/or sputum beyond day-to-day variability sufficient to warrant a change in therapy [4].

Exacerbation may be triggered by an infection with bacteria or viruses or by environmental pollutants. Typically, infections cause 75% or more of the exacerbations; bacteria can roughly be found in 25% of cases, viruses in another 25%, and both viruses and bacteria in another 25%[4].

The relative contributions of these different classes of pathogens may change depending on the severity of the underlying obstructive airway disease. Such changes may also happen within a class, especially for bacterial pathogens[5].

AIMS AND OBJECTIVES

- To identify the Etiological Agent in patients with Acute exacerbation of COPD by sputum culture

To determine the sensitivity pattern to antibiotics for isolated bacterial pathogens.

AECOPD-CAUSES

Causes of exacerbation can be both infectious and noninfectious

Infectious causes

- Bacteria (*Haemophilus influenzae*, *Streptococcus pneumoniae*, *Moraxella catarrhalis*, *Enterobacteriaceae* spp., *Pseudomonas* spp.).
- Viral (*Rhinovirus*spp.,*influenza)*

Non-infectious causes 6, 7

- Smoking
- Environmental conditions (low temperas).
- Air pollution exposure.

Table 1: Factors that potentially modify the risk of

Intrinsic factors	Extrinsic factors
Impairment of lung function	Type of bacteria
Active smoking	Lower environmental temperature
Bronchial hyper responsiveness	Air pollution
Chronic mucous secretion	Treatment of stable and exacerbated COPD
Impairment of defensive mechanisms	
Nonspecific : age , co morbid conditions	

AECOPD-BACTERIAL INFECTION

Bacterial infections are one of the most common causes of acute exacerbation of COPD. They account for 25% exacerbations alone.

Six potential pathways by which bacteria could contribute to the course and pathogenesis of AECOPD were identified:

Childhood lower respiratory tract infection impairs lung growth, reflected in smaller lung volumes in adulthood.

Bacteria cause a substantial proportion of acute exacerbations of chronic bronchitis which cause considerable morbidity and mortality.

Chronic colonization of the lower respiratory tract by bacterial pathogens amplifies the chronic inflammatory response present in COPD and leads to progressive airway obstruction (vicious circle hypothesis).

A defect in the macrophage phagocytosis may result in defective clearance of infectious agents from the lower respiratory tract [8].

Bacterial antigens in the lower airway induce hypersensitivity that enhances airway hyper reactivity and induces eosinophilic inflammation.

Bacterial pathogens invade and persist in respiratory tissue, alter the host response to cigarette smoke or induce a chronic inflammatory response and thus contribute to the pathogenesis of COPD.

Bacterial load is also very important in acute exacerbations. The bacterial load is proportional to the proinflammatory mediators irrespective of the pathogen present although some bacteria associated with greater response than others. Bacterial pathogens as a cause of AECOPD

The three predominant bacterial species isolate are Nontypable Haemophilus influenza, Moraxella catarrahalis, Streptococcus pneumoniae.

Other isolated pathogens are *Staphylococcus aureus, Pseudomonas aeruginosa*, *Klebsiella pneumoniae*, *Haemophilus parainfluneza*e, Members of the family Enterobacteriaceae

Non typeable Haemophilus

MATERIALS AND METHODS

This is a cross sectional study comprising of 50 patients with acute exacerbation of COPD who got admitted in government hospilal. The study group comprising both sexes with age group ranging from 40 to 86 years and 90 % were smokers.

METHODS

Inclusion Criteria

It is a hospital based study done on patients admitted with acute exacerbation of COPD.

Exclusion Criteria

Known case of pulmonary tuberculosis.

All cases who had evidence of pneumonia or bronchiectasis, bronchialasthma, lung abscess, lung cancer.

Pts who were already taking antibiotics before selection, Patients with Ischemic heart disease.

ANTIBIOTICS TESTED

Ceftrioxone,
Cefaperazone-sulbactum
Ciprofloxacin
Levofloxacin
Amikacin
Gentamycin
Piperacillin –tazobactum
Amoxycillin

RESULTS

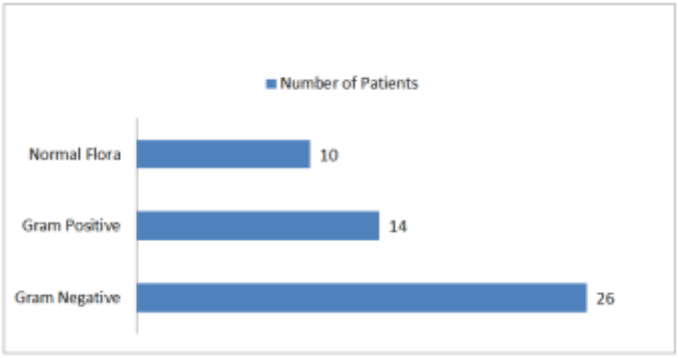
A Total of 50 patients clinically diagnosed as acute exacerbation of chronic obstructive pulmonary disease admitted were studied. Bacterial infections of AECOPD were analyzed. The individual bacterial isolates and their culture & sensitivity patterns to various antibiotics were also recorded. In the study population of 50 who were clinically diagnosed as Acute exacerbation of chronic obstructive pulmonary disease 40(80%) were males and 10(20%) were females.

BACTERIOLOGICAL PROFILE

In the total study population, 26 patients (52%) had gram negative organisms, 14 (14%) had gram positive pathogenic bacteria and 10 (20%) had normal flora isolated from sputum cultures.

Table-2: Bacteriological profile

Bacteriolgy	Number of Patients	% (n=50)
Gram Negative	26	52 %
Gram Positive	14	14 %
Normal Flora	10	20%

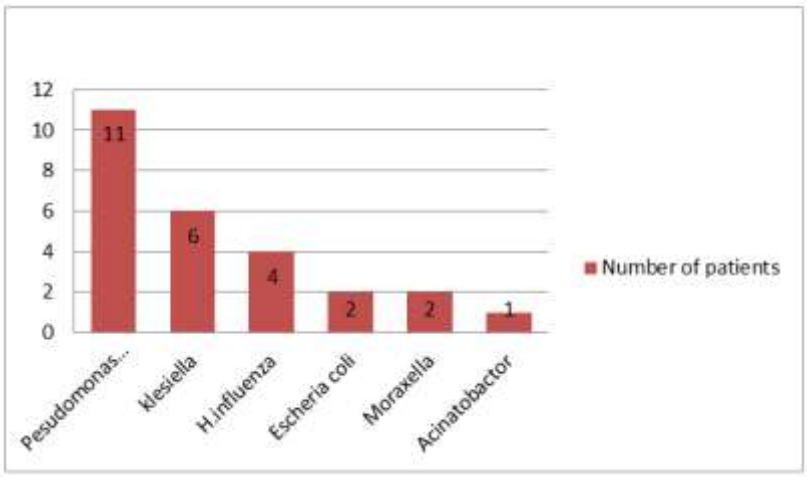


Graph-1: Bacteriolgical profile in study population

ORGANISMS IN GRAM NEGATIVE CULTURES

Table-3: Gram Negative organism distribution

Organism	Number of patients	% [n=26]
Pesudomonas auerginosa	11	42%
Klebsiella pneumonia	06	23 %
H.influenza	04	15.3 %
E. coli	02	7.6%
Moraxella cattralis	02	7.6 %
Acinetobactor	01	3.8 %



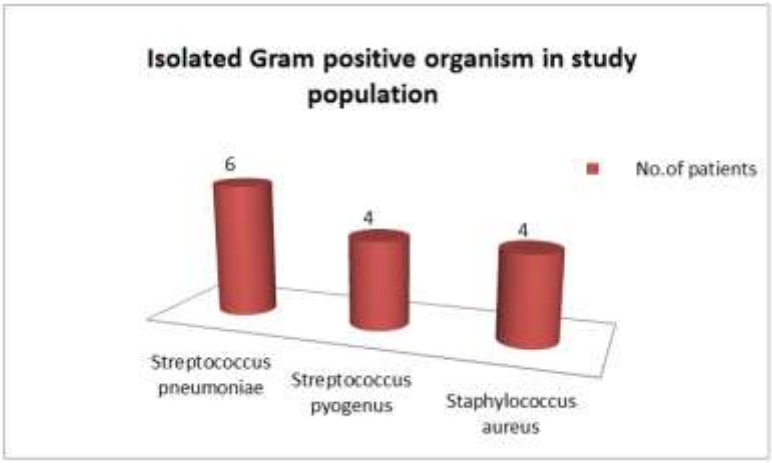
Graph-2: Gram negative organism distribution

Total number of gram negative organisms isolated was 26. The commonest gram negative organism yielded in cultures was Pseudomonas 11cases [42%] and the next commonest organism yielded was Klebsiella pneumonia in 6 cases[23%].

ORGANISMS IN GRAM POSITIVE CULTURES

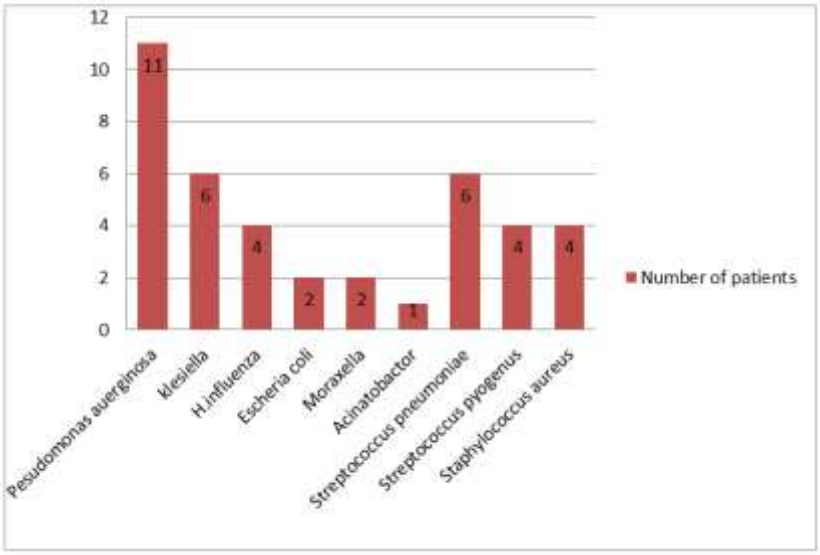
Table-4: Gram positive organism distribution

Organism isolated	No.of patients	Total % (n=14)
Streptococcus pneumoniae	06	42.8%
Streptococcus pyogenus	04	28.5%
Staphylococcus aureus	04	28.5 %



Graph-3: Gram positive organism distribution

Total number of Gram positive organism's isolatedwere 14. Most common was streptococcus pneumoniae 42.8 % (6).The next commonest organism isolate was streptococcus pyogenes (4) and Staph Aureus (4) 28.5 %.



Graph-4: Bacterial isolates

Commonest organisms isolated were Pseudomonas (11), *Klebsiella pneumonia* (06), *Streptococcus pneumonia* (06), *Streptococcus pyogenes* (04), *Staphylococcus aureus* (04),

H.influenza (04), Morxella (02) , E.coli (02), Acinetobacter (01).

The culture plate with antibiotic discs and the circles showing inhibition of growth of bacteria. The antibiotic codes on the discs can be read that designate a particular antibiotic.

In our study Pseudomonas was isolated in 11 patients. The culture and sensitivity shows that 91 % were susceptible to gentamycin, levofloxacin and piperaciliin-tazobactum. 81 % were susceptible to ciprofloxacin and Cefperazone-sulbactum. 73% were susceptible to ceftriaxone and 54 % susceptible to amikacin. Amoxicillin was resistant in all cultures.

Klebsiella was isolated in a total of 06 patients. The culture and sensitivity shows that 84 % were susceptible to Cefperazone –sulbactum, piperacillin – tazobactum and quinolones.66% was susceptible to ceftriaxone and aminoglycosides. Amoxicillin was resistant in all cultures.

Streptococcus pneumonia was isolated in a total of 06 patients. The culture and sensitivity shows that 100 % were susceptible to ceftriaxone, cefperazone-sulbactum and piperacillin-tazobactum. 84 % were susceptible to aminoglycosides and quinolones.66 % was susceptible to amoxicillin.

Streptococcus pyogenus was isolated in a total of 04 patients. The culture and sensitivity shows that 100 % were susceptible to ceftriaxone, cefperazone-sulbactum, piperacillin-tazobactumand levofloxacin. 75 % were susceptible to gentamycin, ciprofloxacin and amoxicillin.50 %were susceptible to amikacin.

H.influenza was isolated in a total of 04 patients. The culture and sensitivity shows that 100% were susceptible to cefperazone-sulbactum.75 % were susceptible to ceftriaxone, piperacillin–tazobactum and gentamycin.50%were susceptible to amikacin and quinolones. amoxcillin was resistant in all cultures.

Staph aureus was isolated in a total of 04 patients. The culture and sensitivity shows that all were susceptible to ceftriaxone, cefperazone-sulbactum, piperacillin-tazobactum and ciprofloxacin i.e100%.75 % were susceptible to levofloxacin. 50 %were susceptible to amoxicillin and aminoglycosides.

E.coli was isolated in a total of 02 patients. The culture and sensitivity shows that 100 % were susceptible to ceftriaxone, piperacillin-tazobactum and quinolones. 50% were susceptible to cefperazone-sulbactum and aminoglycosides amoxicillin was resistant in all cultures.

Moraxella was isolated in a total of 02 patients. The culture and sensitivity shows that 100 % were susceptible to cefperazone-sulbactum, piperacillin-tazobactum, levofloxacin and gentamycin. 50% were susceptible to ceftriaxone, amikacin and ciprofloxacin. Amoxicillin was resistant in all cultures.

Acinatobacter was isolated in only 1 patient, shows susceptibility to cefperazone-sulbactum, piperacillin-tazobactum, ciprofloxacin and gentamycin; resistance to amoxicillin, amikacin and levofloxacin.

DISCUSSION

Inspite of the multifactorial etiopathogenic nature of acute exacerbation of chronic obstructive

pulmonary disease, infection plays a very important and unique role in acute exacerbation of COPD, thus impairing not only ventilator function of the lung but also restricting patient daily routine activities. The prevalence of pathogenic organism responsible for acute exacerbation is showing lot of geographical variation from place to place.

The present study confirms previous data reporting a role of bacterial infection in acute exacerbation of COPD during hospital admissions for acute exacerbations. Presence of bacterial pathogens was found in 80 % of all admitted patients. No significant differences in clinical characteristics could be demonstrated between patients with and without isolation of bacterial pathogens neither with the type of bacterial pathogens.

Remarkably, the incidence of positive sputum cultures in 80 % of our patient population was quite similar to previous data [9-11]. In a large study of 1016 in-patients with acute exacerbation respiratory infection was found in 47 % of sputum cultures [11]. Therefore, it seems a consistent finding that bacterial infection is present in 50% of exacerbations, in hospital as well as in outpatient populations.

In our study 20 % patients show normal flora in sputum culture & sensitivity. In these cases exacerbation may be due to viral infection, atypical organisms and air pollution.

Normal flora in sputum culture cannot rule out bacterial infection. It could be well diagnosed with newer sampling methods like fiber optic bronchoscopy, tracheo bronchial aspirated sample (TBAS), Broncho alveolar lavage fluid (BAL), and protected specimen brushing (PSB).

In our study 3 commonest organisms were isolated Pseudomonas aeruginosa in 11 cases, klebsiella pneumonia in 06 cases, and streptococcus pneumonia in 06 cases

SUMMARY

A total of 50 patients with AECOPD were included in our study. Out of 50 patients 40 were males (80%) and 10 were females (20%). Most of the males [69%] were smokers. The most common organism isolated was gram negative i.e in 26 patients and only 14 were gram positive. Patients with >20 pack years of smoking had predominantly isolated gram negative organism.

The most common gram negative organisms were pseudomonas aeruginosa in 42% of cases followed by klebsiella pneumonia in 23% of cases.

The most common gram positive organisms were Streptococcus pneumonia in 42.3% of cases followed by Streptococcus pathogenes and staph. Aureus in 28.5% of cases.

Most of the Gram negative organisms shows susceptibility to cephalosporins, aminoglycosides and quinolones; Resistance to Amoxicillin.

Streptococcus pneumoniae shows susceptibility to ceftriaxone, cefperazone-sulbactum, piperacillin-tazobactumand levofloxacini.e 100 %; 75 % to gentamycin, ciprofloxacin and amoxicillin;50 % to amikacin.

Streptococcus pyogenus shows susceptibility to ceftriaxone, cefperazone-sulbactum, piperacillin-tazobactumand levofloxacin i.e100 %; 75 % to gentamycin, ciprofloxacin and amoxicillin;50 % to amikacin.

Staph aureus shows susceptibility to ceftriaxone, cefperazone-sulbactum, piperacillin-tazobactum and ciprofloxacin i.e100%; 75 % to levofloxacin; 50 % to amoxicillin and aminoglycosides.

CONCLUSIONS

There have been significant advances in our understanding of aetiology of acute exacerbations of COPD. Frequent exacerbations appear to be associated with worsening health outcomes and effort should focus on prompt effective treatment of each episode.

Bacterial pathogens, mostly gram negative organisms are found to be chief etiological agents in AECOPD.

Empirical antibiotic therapy should be initiated early depending on the culture. Susceptibility patterns of that particular region in order to prevent antibiotic resistance and to decrease health costs.

However treatment options are limited and further research is needed to clarify the mechanisms that commence and sustain exacerbations and to identify new therapeutic agents.

Better and more specific approaches to boosting immune competence are currently under study.

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