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Common Bacterial Isolates in Sputum of AE-COPD Patients in National Institute of Diseases of the Chest & Hospital (NIDCH)

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Abstract **Original Research Article**

Introduction: Chronic Obstructive Pulmonary Disease (COPD) is a chronic inflammatory disabling disease. It is a spectrum of airway disease characterized by the chronic bronchitis (airway obstruction) & emphysema (parenchymal destruction).The major cause for morbidity & mortality in worldwide is COPD &it is the fourth leading cause of death in world. ***Objective of the Study***: To identify the most common bacterial organism isolated from sputum of COPD patients from Out Patient in National Institute of Diseases of the Chest & Hospital (NIDCH). ***Material & Methods:*** Patients who were >40 years old attended Medical OPD at National Institute of Diseases of the Chest & Hospital (NIDCH), Mohakhali, Dhaka, Bangladesh from January 2018 to December 2018, diagnosed with COPD and having symptoms of acute exacerbation were screened for participation. Samples were collected in sterile vial & sent within 2 hours to the central laboratory. Sputum sample to be deemed acceptable for analysis; Microbiological study done by using a low magnification lens (x100) reveals <10 epithelial cells & >25 leucocytes per field. Selected sputa were processed microbiologically for quantitative study following accepted laboratory method. ***Results:*** Total of 70 patients, fulfilling the inclusion & exclusion criteria, 40 male & 30 female with 57% & 43%. Out of 70 patients, 19 showed the overgrowth of the normal commensal in sputum, so only 51 patients (72%) showed growth with culture positive. In terms of age, the study group belongs to a wide range from 40 years to > 80 years with maximum patients (35%) belonging to the age group of 50 -60 years followed by 60-70 years (25%) and 40-50 years (21.88%), Streptococcus pneumonia (14 cases, 27%), Hemophilus influenza(10 cases, 20%), Pseudomonas aeruginosa (7 cases, 14%), Moraxella catarrhalis (6 cases, 12%), Eschericia coli (4 cases, 8%), Staphlococcus aureus (3 cases, 6%), Citrobacter freundii (3 cases, 6%), Klebsiella pneumonia (2 cases, 4%), Acinetobacter baumannii (1 case, 2%), Proteus mirabilis (1 case, 2%). The usual organisms which used to be considered responsible for AECOPD like S. Pneumonia, H. influenza were sensitive to commonly used antibiotics like cephalosporins aminoglycosides, fluoroquinolones. ***Conclusion:*** AE-COPD had serious negative impact on patient pulmonary function, quality of life as well as socioeconomic status. COPD can be triggered or exaggerated commonly by bacterial and viral infections with variable profile depending on the geographical areas. COPD has many aetiology, smoking and environmental pollution plays the major role in it.

Keywords: AECOPD, bacteria, sputum, bacterial culture.

INTRODUCTION

Chronic Obstructive Pulmonary Disease (COPD) is a chronic inflammatory disabling disease [1]. It is a spectrum of airway disease characterized by the chronic bronchitis (airway obstruction) & emphysema (parenchymal destruction) [2]. The major cause for morbidity & mortality in worldwide is COPD &it is the fourth leading cause of death in world [3-5]. In US alone, 24 million people suffer from COPD & is the third leading cause of death [6]. COPD is an inflammatory disease with significant airflow limitation that is not fully reversible [7], & is usually progressive [3], with remissions & exacerbations as Acute Exacerbations of Chronic Obstructive Pulmonary Disease (AE-COPD). Airflow limitation is caused by both airway obstruction & parenchymal destruction. An exacerbation of COPD is acute in onset, which is the worsening of the patient's condition from the stable state characterized by change in the patient's baseline cough, dyspnoea & sputum production [3]. Exacerbation is the amplification of inflammatory response in COPD patients & is triggered by bacterial, viral infections or by environmental pollutants [8]. In

severe cases, the patient is unable to maintain the normal blood gases that lead to respiratory failure [9]. Typically 26% of the exacerbations are caused by bacteria, 25% by viruses, and 27% by combination of two & 22% with no ascertainable cause [10]. Therefore, the predominant cause of acute exacerbations in COPD are bacterial infections [10]. In healthy individuals, the lower airways are sterile, but in COPD patients, there is bacterial colonization of the lower airways with Hemophilus influenza, Streptococcus pneumonia & Branhamella catarrhalis [11, 12]. The most common risk factor for colonization in COPD is cigarette smoking 85% [13]. Vicious Circle Hypothesis states that once bacterial pathogens have entered the lower respiratory tract from impaired mucociliary clearance due to tobacco smoking, they persist by further mucociliary clearance. This is due to increased mucous secretion, disruption of normal ciliary activity and airway epithelial injury [14, 15]. Frequent exacerbations are associated with an accelerated decline of lung function, reduced physical activity, poorer quality of life & an increased risk of mortality [16, 17]. Patients with frequent exacerbations have higher levels of IL-6 & IL-8 in sputum with infrequent exacerbations, suggesting the higher incidence of bacterial colonisation [18]. The presence of bacteria depends on the severity of airway disease 18]. Bacterial pathogens alter the host response to cigarette smoke, induce the inflammatory change & hypersensitivity that enchances airway hypersensitivity [19]. Bacterial infection complicating COPD is diagnosed by the microbiological data [20]. Neutrophils in sputum gram stain indicates the bacteria inducing an inflammatory response rather than colonization [21]. Sputum cultures do not always correlate with clinical parameters & gram stain results [21, 22]. Antibiotics is the vital therapy for patients with severe exacerbations [23]. The choice of antibiotics depends on the sputum culture & sensitivity test. Mild to moderate exacerbations have a high spontaneous remission rate [23]. For diagnosing & grading the acute exacerbation of COPD, clinical guidelines WINNIPEG CRITERIA [24], used based on Increased Breathlessness, Sputum Volume & Purulence.

The Winnipeg Criteria

a) TYPE 1- all the 3 symptoms
b) TYPE 2-any 2 symptoms
c) TYPE 3-any 1 symptom plus at least 1 of the following ; URTI lasting > 5 days ,fever , increase in wheeze , increase in cough and increase in heart rate 20 %.

OBJECTIVE OF THE STUDY

To identify the most common bacterial organism isolated from sputum of COPD patients from Out Patient in National Institute of Diseases of the Chest & Hospital (NIDCH).

MATERIAL & METHODS

Patients who were >40 years old attended Medical OPD at National Institute of Diseases of the Chest & Hospital (NIDCH), Mohakhali, Dhaka, Bangladesh from January 2018 to December 2018, diagnosed with COPD and having symptoms of acute exacerbation were screened for participation. The Spanish Society of Pneumology and Thoracic Surgery 1996 definition was used to diagnose COPD [25], whereas the presence of at least two of the three following symptoms defined by Anthonisen *et al*., [26] was required to diagnose exacerbations:

- Increase in dyspnoea;
- Increase in the production of sputum; and
- Increase in purulence of sputum.

Inclusion Criteria

a) COPD patients diagnosed according to Spanish Society of Pneumology and Thoracic Surgery 1996 definition was used to diagnose COPD [25].
b) Acute exacerbation based on increased dyspnoea, increased sputum volume and purulence.
c) Patients requiring in patient ward admission.
d) Adequate sputum sample based on <10 squamous epithelial cells and >25 pus cells.

Exclusion Criteria

a) Patients having bronchiectasis, tuberculosis, asthma, malignancy, community acquired pneumonia.
b) Previous admission or antibiotic treatment in the last 21 days.
c) Patients managed in outpatient department, emergency or admission required in Intensive Care Unit.
d) Ischaemic heart disease patients.

Microbiological Sputum Study

All the patient's sputum sample were obtained at the first visit to OPD or emergency department in National Institute of Diseases of the Chest & Hospital (NIDCH), Mohakhali, Dhaka, Bangladesh. Samples were collected in sterile vial & sent within 2 hours to the central laboratory. Sputum sample to be deemed acceptable for analysis; Microbiological study done by using a low magnification lens (x100) reveals <10 epithelial cells & >25 leucocytes per field. Selected sputa were processed microbiologically for quantitative study following accepted laboratory method. Using the microbiological loop, 0.01 ml sputa were seeded in the following culture media: blood agar, Maconkey agar, Chocolate agar, Sabouraud's agar plus chloramphenicol. Incubation was carried out at 35 +/- 2^0C in aerobic condition. In case of chocolate agar, the atmosphere contained 5-7% carbon dioxide. A first reading was taken after 24 hours, second final one was taken after 48 hours of culture [27].

The sensitivity of bacteria identified as potentially pathogenic micro organisms to antimicrobial agents was studied by the minimum inhibitory concentration technique. Classifications as sensitive, intermediate & resistant were made according to the criteria issued by the national committee for clinical laboratory study [28]. Antibiotic tested were the following: amoxicillin, amoxicillin/clavulanic acid, co-trimoxazole, cefixime, cefuroxamine, erythromycin and ciprofloxacin. All data analysis Windows SPSS Version 21.0.

RESULTS

Total of 70 patients, fulfilling the inclusion & exclusion criteria, 40 male & 30 female with 57% & 43%. Out of 70 patients, 19 showed the overgrowth of the normal commensal in sputum, so only 51 patients (72%) showed growth with culture positive. In terms of age, the study group belongs to a wide range from 40 years to > 80 yearswith maximum patients (35%) belonging to the age group of 50 -60 years followed by 60-70 years (25%) and 40 -50 years (21.88%).

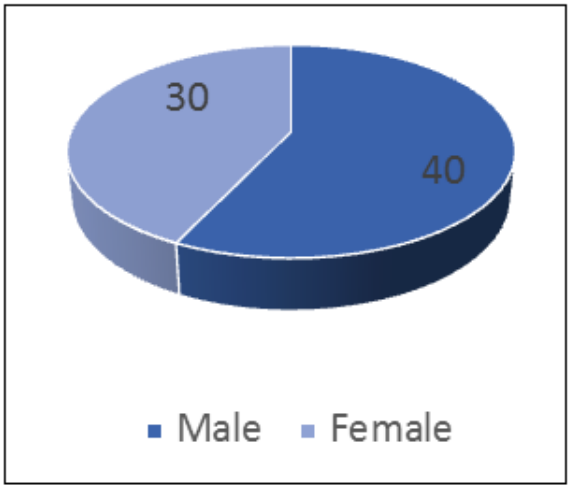

Fig-1: Distribution of male and female patients

Table-1: Organisms were isolated in significant concentration in sputum as noted COAD (N=70)

S. No	Organism Name	Number	Percent
1	Streptococcus pneumonia	14	27
2	Hemophilus influenza	10	20
3	Pseudomonas aeruginosa	7	14
4	Moraxella catarrhalis	6	12
5	Eschericia coli	4	8
6	Staphylococcus aureus	3	6
7	Citrobacter freundii	3	6
8	Klebsiella pneumoniae	2	4
9	Acinetobacter baumannii	1	2
10	Proteus mirabilis	1	2

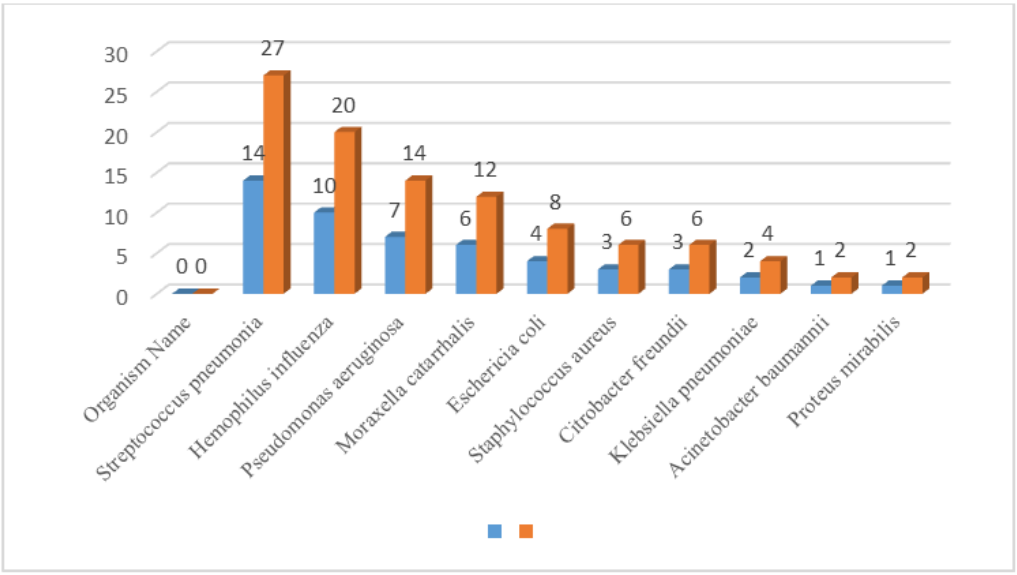

Fig-2: Organisms were isolated in significant concentration in sputum as noted Patients

The following organisms were isolated in significant concenteration in sputum as noted in (Table-1 & Figure-2): Streptococcus pneumonia (14 cases, 27%), Hemophilus influenza(10 cases, 20%), Pseudomonas aeruginosa (7 cases, 14%), Moraxella catarrhalis (6 cases, 12%), Eschericia coli (4 cases, 8%), Staphlococcus aureus (3 cases, 6%), Citrobacter freundii (3 cases, 6%) , Klebsiella pneumonia (2 cases,

4%), Acinetobacter baumannii (1 case, 2%), Proteus mirabilis (1 case, 2%). The usual organisms which used to be considered responsible for AECOPD like S. Pneumonia, H. influenza were sensitive to commonly used antibiotics like cephalosporins aminoglycosides, fluoroquinolones .Gram Negative Bacteria were sensitive to colistin & polymyxin compared to above antibiotics. Organism like E.coli were mostly resistant to second and third generation cephalosporins.

DISCUSSION

COPD considered as leading cause of mortality & morbidity & exacerbation of COPD brings burden to both patient & hospital. AECOPD had serious negative impact on patient pulmonary function, quality of life as well as socioeconomic status. COPD can be triggered or exaggerated commonly by bacterial and viral infections with variable profile depending on the geographical areas. COPD has many aetiology, smoking and environmental pollution plays the major role in it. AECOPD can be triggered by bacterial infection since many patients had infection in lower airway or it can be non-infectious can be triggered by allergens which needs to be confirmed by laboratory investigation. The common trigger for acute exacerbation of COPD are infections due to virus or bacteria of the trachea-bronchial tree & air pollution [29]. In terms of age, the study group belongs to a wide range from 40 years to >80 years with maximum patients (35%) belonging to the age group of 50 -60 years followed by 60-70 years (25%) and 40 -50 years (21.88%). Our study has maximum number of cases in 50-60 years age group which correlate with the study in American lung association [30]. In this study Streptococcus pneumonia (14 cases, 27%), Hemophilus influenza (10 cases, 20%) , Pseudomonas aeruginosa (7 cases, 14%), Moraxella catarrhalis (6 cases, 12%), Eschericia coli (4 cases, 8%), Staphlococcus aureus (3 cases, 6%), Citrobacter freundii (3 cases, 6%), Klebsiella pneumonia (2 cases, 4%), Acinetobacter baumannii (1 case, 2%), Proteus mirabilis (1 case, 2%). The usual organisms which used to be considered responsible for AECOPD like S. Pneumonia, H. influenza were sensitive to commonly used antibiotics like cephalosporins aminoglycosides, fluoroquinolones. Gram Negative Bacteria were sensitive to colistin & polymyxin compared to above antibiotics. Organism like E. coli were mostly resistant to second and third generation cephalosporins respectably. Our study showed sputum culture positive in 72% who presented with AECOPD & around 50% of exacerbation of COPD had lower respiratory tract isolated organisms. Similar bacterial isolation were found in many Indian based Studies [31-33]. Strep.pneumoniae is the most common organism isolated in our study which correlates to Sanjay Sethi [34] & Patel AK [35] who had similar results in 2015. Several studies [33, 36] found Pseudomonas aeruginosa most common organism to be responsible for AECOPD. Individuals with severe pulmonary function impairment, manifested by FEV1 <50% predicted, are at sixfold higher risk of suffering acute exacerbation caused by H.influenza or Pseudomonas aeruginosa [37].

CONCLUSION

AE-COPD had serious negative impact on patient pulmonary function, quality of life as well as socioeconomic status. COPD can be triggered or exaggerated commonly by bacterial and viral infections with variable profile depending on the geographical areas. COPD has many aetiology, smoking and environmental pollution plays the major role in it. AECOPD can be triggered by bacterial infection since many patients had infection in lower airway or it can be non infectious can be triggered by allergens which needs to be confirmed by laboratory investigation. So it is important to do a routine sputum culture sensitivity for all the COPD patient who attend the routine outpatient department in regular intervals and mandatory for In-Patients to prevent bacterial infections caused by airway infection. It also helps in choosing appropriate antibiotics and prevent antibiotic resistance. All together it reduces morbidity and mortality due to COPD.

REFERENCES

1. 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. American journal of respiratory and critical care medicine. 1998 May 1;157(5):1418-22.
2. Sethi S, Murphy TF. Bacterial infection in chronic obstructive pulmonary disease in 2000: a state-of-the-art review. Clinical microbiology reviews. 2001 Apr 1;14(2):336-63.
3. GOLD (Global Initiative for Chronic Obstructive Lung Disease) -global strategy for the diagnosis, management, and prevention of chronic obstructive pulmonary disease-revised 2006. Http//www.goldcopd.org. 1-100.
4. World Health Report. Geneva: World Health Organization. Available from URL: http://www.who.int/whr/2000/en/statistics.htm; 2000.
5. Lopez AD, Shibuya K, Rao C, Mathers CD, Hansell AL, Held LS, Schmid V, Buist S. Chronic obstructive pulmonary disease: current burden and future projections. European Respiratory Journal. 2006 Feb 1;27(2):397-412.
6. Kochanek KD, Miniño AM, Murphy SL, Xu J, Kung HC. Deaths: final data for 2009. Natl Vital Stat Rep, 2011;60(3):3-11.
7. Soriano JB, Visick GT, Muellerova H, Payvandi N, Hansell AL. Patterns of comorbidities in newly diagnosed COPD and asthma in primary care. Chest, 2005; 128(4): 2099-2107.
8. White AJ, Gompertz S, Stockley RA. Chronic obstructive pulmonary disease. 6: Th e etiology of

exacerbations of chronic obstructive pulmonary disease. Th orax. 2003; 58(1): 73-80.

9. Connors Jr AF, Dawson NV, Thomas C, Harrell Jr FE, Desbiens N, Fulkerson WJ, Kussin P, Bellamy P, Goldman L, Knaus WA. Outcomes following acute exacerbation of severe chronic obstructive lung disease. The SUPPORT investigators (Study to Understand Prognoses and Preferences for Outcomes and Risks of Treatments). American journal of respiratory and critical care medicine. 1996 Oct;154(4):959-67.
10. Connors Jr AF, Dawson NV, Thomas C, Harrell Jr FE, Desbiens N, Fulkerson WJ. SUPPORT investigators (Study to Understand Prognoses and Preferences for Outcomes and Risks of Treatments). Outcomes following acute exacerbation of severe chronic obstructive lung disease. Am J Respir Crit Care Med. 1996;154(4 Pt 1):959-67.
11. Celli BR, MacNee WA, Agusti AA, Anzueto A, Berg B, Buist AS, Calverley PM, Chavannes N, Dillard T, Fahy B, Fein A. Standards for the diagnosis and treatment of patients with COPD: a summary of the ATS/ERS position paper. European Respiratory Journal. 2004 Jun 1;23(6):932-46.
12. Cabello H, Torres A, Celis R, El-Ebiary M, De La Bellacasa JP, Xaubet A, Gonzalez J, Agusti C, Soler N. Bacterial colonization of distal airways in healthy subjects and chronic lung disease: a bronchoscopic study. European Respiratory Journal. 1997 May 1;10(5):1137-44.
13. Lindberg A, Eriksson B, Larsson LG, Rönmark E, Sandström T, Lundbäck B. Seven-year cumulative incidence of COPD in an age-stratified general population sample. Chest. 2006 Apr 1;129(4):879-85.
14. Haas H, Morris JF, Samson S, Kilbourn JP, Kim PJ. Bacterial flora of the respiratory tract in chronic bronchitis: comparison of transtracheal, fiberbronchoscopic, and oropharyngeal sampling methods. American Review of Respiratory Disease. 1977 Jul;116(1):41-7.
15. Laurenzi GA, Potter RT, Kass EH. Bacteriologic flora of the lower respiratory tract. New England Journal of Medicine. 1961 Dec 28;265(26):1273-8.
16. Donaldson GC, Seemungal TA, Bhowmik A, Wedzicha JA. Relationship between exacerbation frequency and lung function decline in chronic obstructive pulmonary disease. Thorax. 2002 Oct 1;57(10):847-52.
17. Vestbo J, Edwards LD, Scanlon PD, Yates JC, Agusti A, Bakke P, Calverley PM, Celli B, Coxson HO, Crim C, Lomas DA. Changes in forced expiratory volume in 1 second over time in COPD. New England Journal of Medicine. 2011 Sep 29;365(13):1184-92.
18. Bhowmik A, Seemungal TA, Sapsford RJ, Wedzicha JA. Relation of sputum inflammatory markers to symptoms and lung function changes in COPD exacerbations. Thorax. 2000 Feb; 55(2):114–120.
19. Shaheen SO, Barker DJ, Shiell AW, Crocker FJ, Wield GA, Holgate ST. The relationship between pneumonia in early childhood and impaired lung function in late adult life. American journal of respiratory and critical care medicine. 1994 Mar;149(3):616-9.
20. Bailgeman W. Quantitative sputum Gram strains in chronic bronchial disease. Lung. 1979; 156:265.
21. Isada CM. Pro antibiotics for chronic bronchitis with exacerbations. Semin Respir Infect. 1993; 8:243.
22. Gump DW, Philips CA, Forsyth BR. Role of infection in chronic bronchitis. Am Rev Respir Dis. 1976; 113:465.
23. Nouira S, Marghi S, Belgith M, Basbes L. Once daily oral ofloxacin in chronic obstructive pulmonary disease exacerbation requiring ventilation; a randomized placebo-controlled trial. Lancet. 2001; 358:2020–2025.
24. MacIntyre N, Huang YC. Acute exacerbations and respiratory failure in chronic obstructive pulmonary disease. Proceedings of the American Thoracic Society. 2008 May 1;5(4):530-5.
25. Montemayor T, Alfajeme I, Escudero C, Morera J, Agudo LS, de la SEPAR GD. Normativa sobre diagnóstico y tratamiento de la enfermedad pulmonar obstructiva crónica. Archivos de Bronconeumología. 1996 Jun 1;32(6):285-301.
26. Anthonisen NR, Manfreda J, Warren CP, Hershfield ES, Harding GK, Nelson NA. Antibiotic therapy in exacerbations of chronic obstructive pulmonary disease. Annals of internal medicine. 1987 Feb 1;106(2):196-204.
27. Balows A, Hansler WJ, Herrmann KL. Manual of clinical microbiology. 5th ed.Washington, DC: American Society of Microbiology, 1991;1226-1314.
28. National Committee for Clinical Laboratory Standards. In: Performance standards for antimicrobial susceptibility testing. Vol 17. NCCLS, Philadelphia, PA; 1997 (M57–M100; Vol 1).
29. American Lung AssociationTrends in COPD (chronic bronchitis and emphysema): morbidity and mortality Research and Program Services Division, Epidemiology and Statistics Unit, American Lung Association, Washington, DC, 2011.
30. H. SharanAerobic bacteriological study of acute exacerbations of chronic obstructive pulmonary disease. Journal Clin Diagn Res, 2015;9(8);DC10-DC12.
31. Monso E, Ruiz J, Rosell A, Manterola J, Fiz J, Morera J, Ausina V. Bacterial infection in chronic obstructive pulmonary disease. A study of stable and exacerbated outpatients using the protected specimen brush. American journal of respiratory

and critical care medicine. 1995 Oct;152(4):1316-20.

32. Chawla K, Mukhopadhyay C, Majumdar M, Bairy I. Bacteriological profile and their antibiogram from cases of acute exacerbations of chronic obstructive pulmonary disease: A hospital based study. Journal of clinical and diagnostic research. 2008;2(1):612-6.
33. Sethi S. Bacteria in exacerbations of chronic obstructive pulmonary disease: phenomenon or epiphenomenon?. Proceedings of the American Thoracic Society. 2004 Apr;1(2):109-14.
34. Patel AK, Luhadia AS, Luhadia SK. Sputum bacteriology and antibiotic sensitivity pattern of patients having acute exacerbation of COPD in India: a preliminary study. J Pulm Respir Med. 2014;5(1):238
35. Madhavi S, Rao MR, Rao RJ. Bacterial etiology of acute exacerbations of chronic obstructive pulmonary disease. Journal of Microbiology and Biotechnology Research. 2012;2(3):440-4.
36. Eller J, Ede A, Schaberg T, Niederman MS, Mauch H, Lode H. Infective exacerbations of chronic bronchitis: relation between bacteriologic etiology and lung function. Chest. 1998 Jun 1;113(6):1542-8.
37. Wilson R. Infections of the airways. Curr Opin Infect DIS, 1991; 4:166-177.