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# A Study of Microbiological Profile and Sensitivity Pattern in Patients with Exacerbation of Bronchiectasis in a Tertiary Care Hospital

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**A B S T R A C T**

Bronchiectasis is of clinician's concern because of its frequent exacerbations. The prevalence is currently increasing, probably due to an increased use of thoracic computed tomography (CT). So, it is very crucial to know the causative organism and the drug sensitivity for a rational and precise treatment. After evaluating the diagnosis of exacerbation of bronchiectasis by clinical and radiological parameters, 50 patients were enrolled in the study, among which FOB done in 41 cases. Microbiological study of sputum and BAL was done to detect the causative organisms and their sensitivity patterns. *Pseudomonus aeruginosa* and *Klebsiella* sp. in sputum; and *Pseudomonus aeruginosa* and Haemophilus influenza in BAL are the most abundant organisms. Carbapenems, Aminoglycosides and Coamoxyclav are the most potent antibiotics. Sputum and BAL were found to have nearly the same efficacy in detection of the pathogenic organisms of LRT. So sputum culture can replace BAL fluid culture in the work up of exacerbation of bronchiectasis.

## Introduction

Bronchiectasis is defined as the abnormal, irreversible dilatation and thickening of bronchi. Most of the time bronchiectasis is the result of pathological processes that cause bronchial wall and its surrounding tissues destruction. Bronchiectasis is of clinician's concern because of its frequent exacerbations. A case of exacerbation is defined as a person with bronchiectasis with a deterioration in three or more of the following symptoms for at least 48 hrs:

Cough

Sputum volume and/or consistency

Sputum purulence

Breathlessness and/or exercise tolerance

Fatigue and/or malaise

Haemoptysis

In addition, a clinician determines that a change in bronchiectasis treatment is required

The prevalence is currently increasing, probably due to an increased use of thoracic computed tomography. A number of radiological features are helpful in diagnosing bronchiectasis i.e

bronchus visualised within 1 cm of the pleural surface, especially true for lung adjacent to costal pleura and most helpful sign for early cylindrical change

lack of tapering

bronchoarterial ratio > 1.5

bronchial wall thickening(normally wall of bronchus should be less than half the width of the accompanying pulmonary artery branch)

mucoid impaction

air-trapping and mosaic perfusion Signs described on CT include:

tram-track sign

signet ring sign

string of pearls sign

cluster of grapes sign

In developed countries, broncietasis is divided into 2 types : Cystic Fibrosis bronchiectasis and Non-Cystic Fibrosis bronchiectasis. Cystic fibrosis is a common life-limiting autosomal recessive genetic disorder. Cystic fibrosis affects several body systems, and morbidity and mortality is mostly caused by bronchiectasis, small airways obstruction, and progressive respiratory impairment. There is renewed interest in non-cystic fibrosis bronchiectasis, which is a cause of significant morbidity in adults.

## Materials and Methods

This is an observational and cross sectional study done in the Department of Respiratory Medicine, NRS Medical College and Hospital from June 2019 to June 2020 with 50 adult patients with exacerbation of bronchiectasis admitted in IPD of department of respiratory medicine, N.R.S.MCH Kolkata

### Inclusion criteria

Patients >18 years

Radiologically established cases of bronchiectasis

Showing clinical features of exacerbation

Patients giving consent

### Exclusion criteria

Patients <18years

Patients with stable bronchiectasis

Very poor general health and severe malnutrition or with haemodynamic instability, massive pleural effusion

Sputum with AFB positive or sputum CBNAAT or BAL fluid CBNAAT positive

Recent history of myocardial infarction, heart failure or arrhythmias or any other cardiological causes of dyspnea

Pregnancy

Patients with epilepsy

Patients who did not give consent

Any other causes of dyspnea other than bronchiectasis

HIV, HbSAg, AntiHCV positive patients

Fifty (50) patients with exacerbation of bronchiectasis of both gender admitted in IPD of Dept. of Respiratory Medicine, Nil rattan Sircar Medical College Hospital were selected according to the inclusion and exclusion criteria. Patient's range of age was 18 years – 65 years.

Detailed demographic and clinical parameters including age, sex, smoking history, past H/O ATT with duration, clinical symptoms,(cough, fever, sputum production, haemoptysis, chest pain, SOB, weight loss) and clinical signs (pallor, cyanosis, clubbing, oedema, tachypnea, tachycardia, raised temperature)were evaluated in all patients. Presences of leucocytosis and any comorbidity, especially diabetes mellitus and HTN were documented. Blood for complete hemogram, blood glucose, urea, creatinine, liver function test, and chest X-rays (posteroanterior and lateral view. All patients had undergone a HRCT scan of thorax. A fresh sample of sputum is collected, before starting of antibiotic, in a sterile, wide-mouthed, dry, leak-proof and break-resistant plastic-container and sent to the laboratory for testing for Gram stain, culture sensitivity, fungal stain, AFB stain and CBNAAT. Fibrotic bronchoscopy (FOB) was planned next in all patients. Macroscopic appearance of bronchial tree during FOB (intraluminal growth, presence of secretions/pus, appearance of bronchial mucosa, etc.) was noted. Bronchoalveolar lavage (BAL) fluid was sent for AFB smear, CBNAAT, Gram stain and culture sensitivity, fungal stain in all patients.

In the laboratory, samples were inoculated on McConkey agar, Nutrient agar, Chocolate agar and Blood agar. The inoculum on the plate was streaked out for discrete colonies with a sterile wire loop. The culture plates were incubated at 37°C for 24 hours and observed for growth through the formation of colonies. All the bacteria were isolated and identified using morphological, Gram staining & microscopy and biochemical tests following standard procedures.

Antimicrobial sensitivity testing was performed for all the isolates by Kirby-Bauer disc diffusion method on Mueller-Hinton agar plates. Three to four colonies were inoculated in peptone water and incubated for two hours at 37ºC, to bring the organism to logarithmic phase. The turbidity of the suspension was adjusted to 0.5 McFarland standards. Within fifteen minutes of preparation of the suspension, a sterile cotton swab was immersed in the suspension and the excess suspension is removed by rotating the swab against the wall of the test tube. A lawn culture of the inoculum was made by streaking the swab over the surface of the plate in three directions. After about 10 to 15 minutes, the antibiotic discs were placed, five on each plate and incubated at 37ºC for 20 to 24 hours. Zone of inhibition of bacterial growth around the antibiotic discs were measured using the Himedia scale. Interpretations were made using the Clinical and Laboratory Standards Institute, USA guidelines.

## Results and Discussion

The present study was done in the Department of Respiratory Medicine, NRS Medical College. Fifty (50) patients were included in the study after they had fulfilled the inclusion and exclusion criteria.

Frequency of organisms from sputum culture of the patients with bronchiectasis exacerbation shows (vide Fig :1)

*Pseudomonus aeruginosa* to be the most abundant organism followed by *Klebsiella pneumoniae* followed by *Acinetobacter baumanii*, *Escherichia coli* and *Staphylococcus aureus*, then *Pseudomonus putida* and Coagulase Negative Staphylococcus.

Clinically significant fungal elements are not seen in any of the sputum samples

Frequency of organisms from BAL culture of the patients with bronchiectasis exacerbation shows

(vide Fig :2). The most abundant to be *Pseudomonus aeruginosa*, *Haemophilus influenza* and *Acinetobacter baumanii*, followed by *Escherichia coli*, *Klebsiella pneumoniae*, *Staphylococcus aureus* and Citrobacter species.

No clinically significant fungal elements are seen.

Total number of sputum samples = 50 Total number of organisms detected =44

% of positivity = 88 %

Total number of BAL samples = 4I

Total number of organisms detected =40

% of positivity = 97.56 %

P value for this Z score = 0.088

So, the difference between these two positivities is not statistically significant

The drug sensitivity pattern shows : (vide Fig :3,4,5)

*Pseudomonas aeruginosa* in both sputum and BAL is mostly sensitive to Aminoglycosides, Cefoperazone Sulbactam, Carbapenems And mostly resistant to Ceftazidime, Ceftriaxone, Cefepime, Piperacillin Tazobactam.

*Klebsiella pneumoniae* in sputum and BAL is mostly sensitive to Colistin, Carbapenems, Tigecycline, Piperacillin Tazobactam, Aminoglycosides And mostly resistant to Cefotaxime, Quinolones, followed by Coamoxyclav, Cefepime. Escherichia coli in both sputum and BAL is mostly sensitive to Colistin, Tigecycline, Aminoglycosides, followed by Carbapenems, Piperacillin Tazobactam, Clindamycin

And mostly resistant to, Cefepime,Cefoperazone Sulbactam, Quinolones, Ceftriaxone, Coamoxyclav.

*Acinetobacter baumanii* in both sputum and BAL is mostly sensitive to Carbapenems followed by Cefoperazone Sulbactam, Piperacillin Tazobactam, Ceftazidime, Ceftriaxone, Aminoglycosides and mostly resistant to Quinolones and Cefotaxime.

Staphylococcus aureus in both sputum and BAL is mostly sensitive to Coamoxyclav, Linezolid, Vancomycin folled by Teicoplanin, Tigecycline and mostly resistant to Ceftriaxone, Quinolone, Clindamycin, Macrolides.

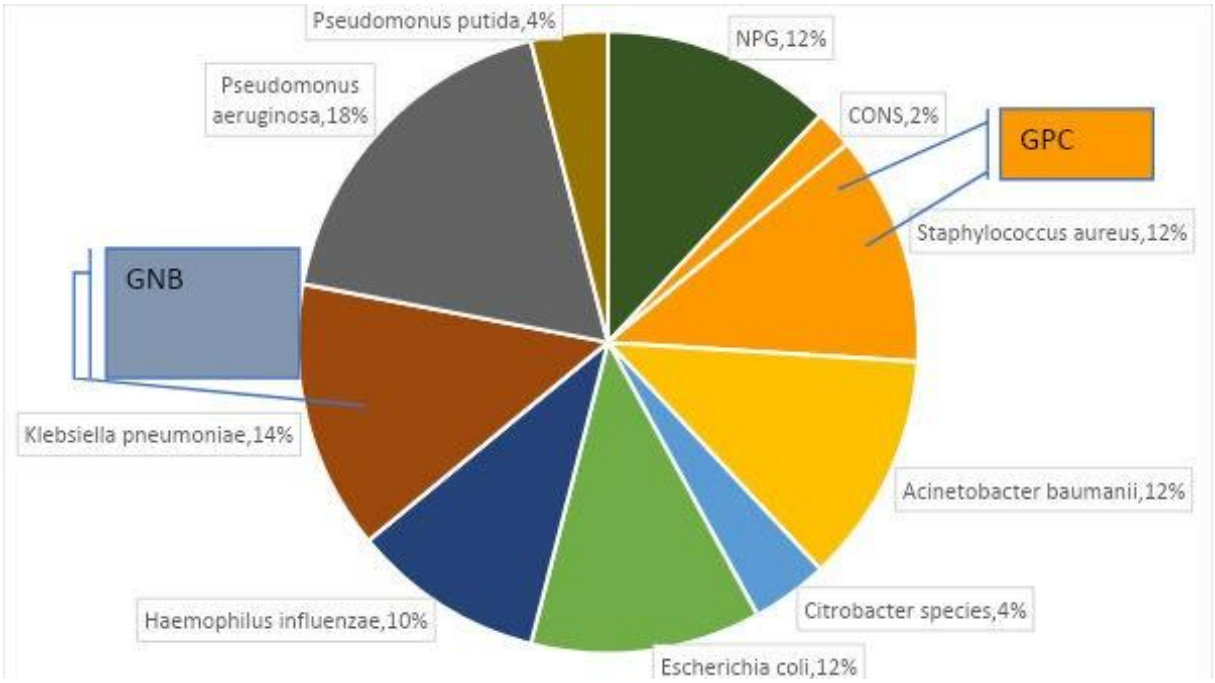
*Haemophilus influenzae* in sputum is mostly sensitive to Carbapenems, Cefotaxime, Coamoxyclav and mostly resistant to Quinolones and Macrolides.

*Haemophilus influenzae* in BAL is mostly sensitive to Carbapenems, Piperacillin Tazobactam, Tigecycline, Cefoperazone Sulbactam Cefotaxime, Coamoxyclav and mostly resistant to Quinolones, Macrolides, Ceftriaxone.

*Pseudomonas putida* in sputum is mostly sensitive to Aminoglycosides, Carbapenems, Cefepime and mostly resistant to Cefoperazone Sulbactam, Ceftazidime, Ceftriaxone, Quinolone, Piperacillin Tazobactam, and Tigecycline.

Citrobacter in both sputum and BAL is mostly sensitive to Carbapenems, Aminoglycosides, Piperacillin Tazobactam, Tigecycline, Colistin followed by Cefoperazone Sulbactam, Ceftriaxone and mostly resistant to Quinolones,Cefepime, Clindamycin, Coamoxyclav, Cefotaxime.

**Fig.1** Distribution of organism in sputum culture




NPG : No pathogenic growth GNB : Gram Negative Bacilli GPC : Gram Positive Cocci

**Fig.2** Distribution of organism in BAL culture

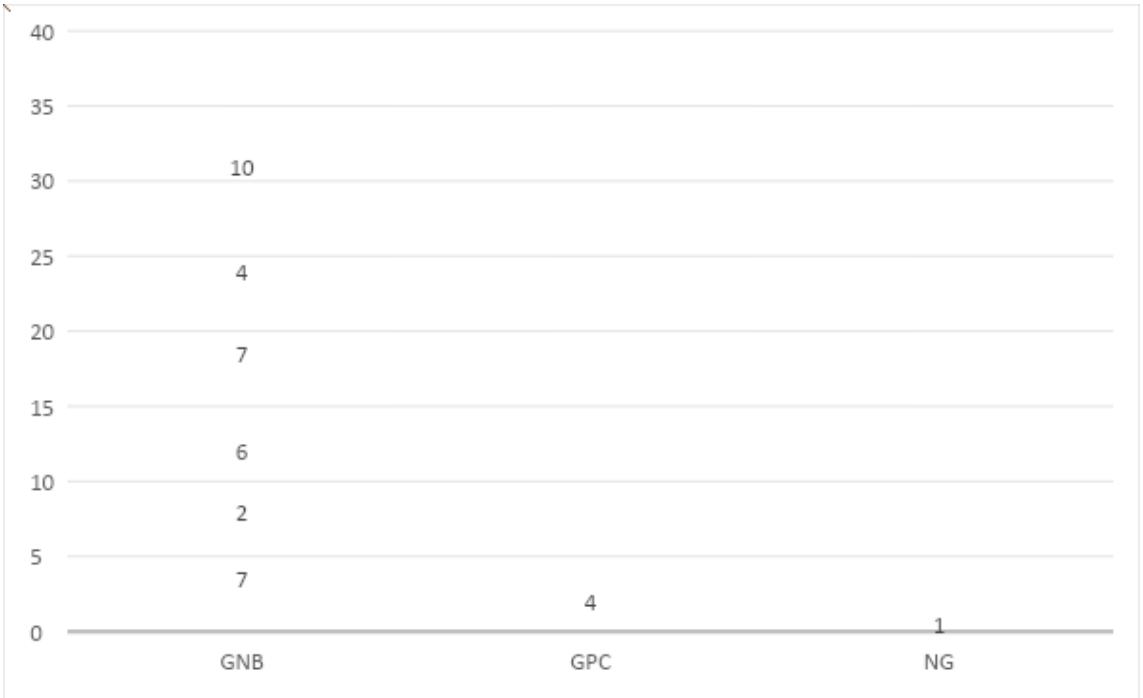


**Fig.3** Drug sensitivity - GPC

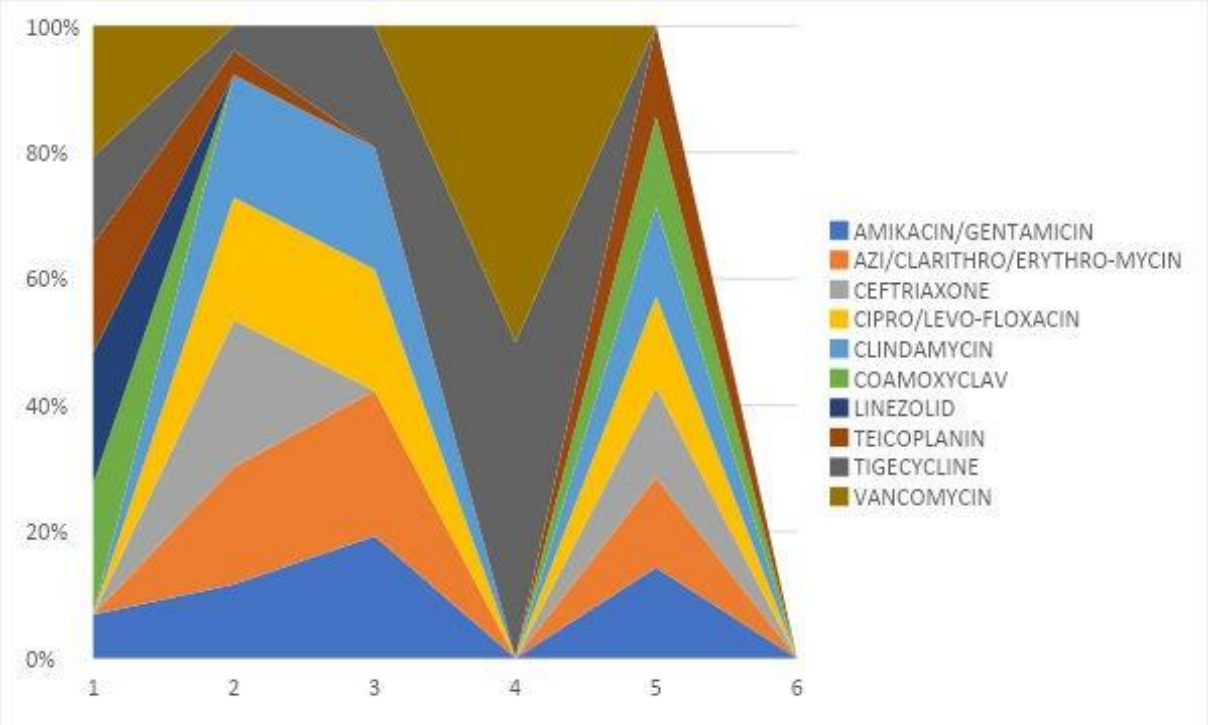



**Fig.4** Drug sensitivity – GNB

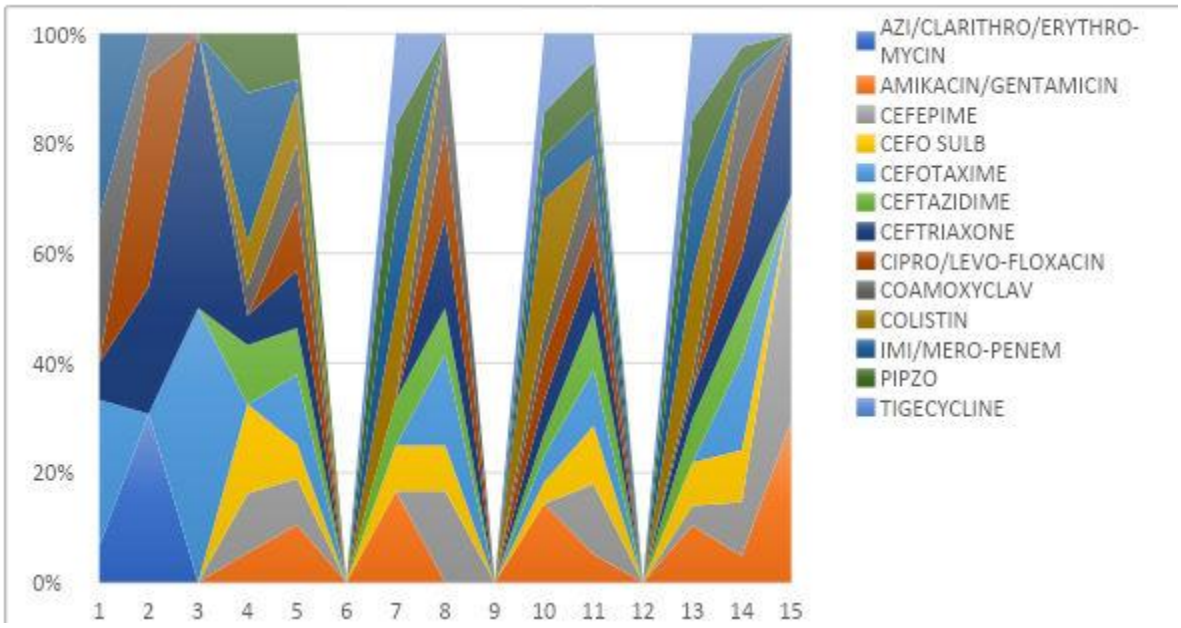
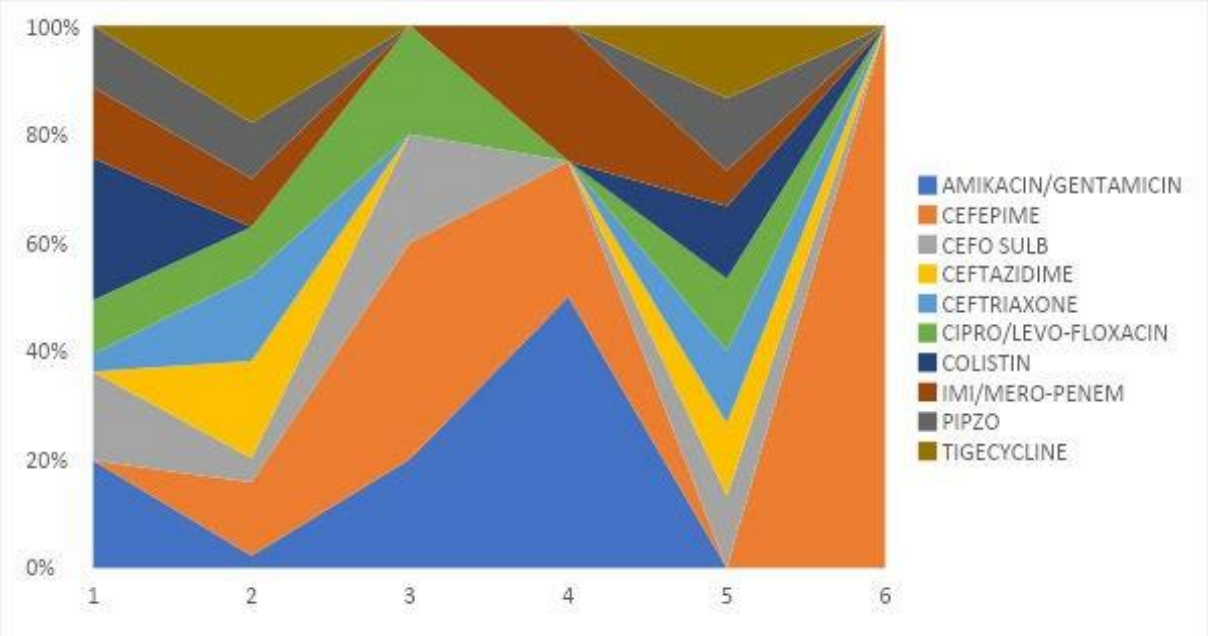


**Fig.5** Drug sensitivity - *Pseudomonas sps*




Coagulase Negative Staphylococcus in sputum is mostly sensitive to Vancomycin and Tigecycline and mostly resistant to Ceftriaxone, Quinolone, Clindamycin, Macrolides, Coamoxyclav, Linezolid, Teicoplanin.

So, it may be concluded that, *Pseudomonas aeruginosa* and *Haemophilus influenzae* in BAL are the most abundant organisms. *Pseudomonus aeruginosa* and Klebsiella sp. in sputum; and *Pseudomonus aeruginosa, Haemophilus influenzae* and *Acinetobacter baumanii* in BAL are the most abundant organisms. Carbapenems, Aminoglycosides and Coamoxyclav are the most potent antibiotics.

Sputum and BAL have nearly the same efficacy in detection of the pathogenic organisms of LRT. So sputum culture can replace BAL fluid culture in the work up of exacerbation of bronchiectasis.

With continuously changing bacterial flora of bronchiectasis, choice of antibiotic should be based on the local bacterial sensitivity pattern. Periodic studies to identify probable agents and their antibiotic sensitivity pattern would assist in formulating a cost effective antibiotic strategy in reducing the emergence of drug resistance.

This study will help in selection of appropriate antibiotic protocol, which will help to break the vicious cycle of infection, inflammation and damage and injury to the bronchial wall and prevention of emergence and dissemination of MDR strains.

In this study, sputum culture was positive in 88% cases and the predominants are *Pseudomonas aeruginosa, Klebsiella pneumoniae, Acinetobacter baumanii, Escherichia coli, Staphylococcus aureus, Haemophilus influenzae*.

The predominant organisms seen to be *Pseudomonas aeruginosa* and *Haemophilus influenzae* causing bronchiectasis exacerbation, in many of the previous studies by Redondo *et al.*, (2016); Lee *et al.*, (2018); Tunney *et al.*, (2013); Richardson *et al.*, (2019) and Miao *et al.*, (2015) (the positive culture rate using sputum sample was 74%). Contrary to these studies, *Haemophilus* had been shown to cause very few episodes of exacerbation, by Rogers *et al.*, (2014).

In this study, in 97.5 % cases BAL culture is positive.

Frequency of organisms from BAL culture of the patients with bronchiectasis exacerbation shows

*Pseudomonus aeruginosa* > *Haemophilus influenzae* = *Acinetobacter baumanii*> *Escherichia coli* >*Klebsiella pneumoniae* =*Staphylococcus aureus* > Citrobacter species.

As stated by Miao *et al.,* (2015) the positive culture rate for BAL was 48%. Organisms predominant in BAL samples were *H. influenzae*, *P. aeruginosa* and *S. aureus*.

*Pseudomonas aeruginosa* in both sputum and BAL is mostly sensitive to Aminoglycosides, Cefoperazone Sulbactam, Carbapenems in accordance to the study by Lõivukene *et al.,* (2006) and Alişkan *et al.,* (2008)

*Acinetobacter baumanii* in both sputum and BAL is mostly sensitive to Carbapenems and mostly resistant to Quinolones and Cefotaxime, supported by the studies by Lõivukene *et al.,* (2006) and Alişkan *et al.,* (2008) but goes contrary to the study by Prakhar Sharma *et al.,* (2017) with significant in vitro resistance to carbapenems.

*Klebsiella pneumoniae* in both sputum and BAL is mostly sensitive to Colistin, Carbapenems, Tigecycline, Piperacillin Tazobactam, Aminoglycosides and mostly resistant to Cefotaxime, Quinolones, supported by the previous studies done by Alişkan *et al.,* (2008) and Mahdi Yahya Mohsen *et al.,* (2016)

*Escherichia coli* in both sputum and BAL is mostly sensitive to Colistin, Tigecycline, Aminoglycosides, And mostly resistant to, Cefepime, Cefoperazone Sulbactam, Quinolones, Ceftriaxone, Coamoxyclav, though in the study by Wang *et al.,* (2000), coamoxyclav was intermediately sensitive, and in contrary to the study by Mahdi Yahya Mohsen *et al.,* (2016) where Piperacillin/tazobactam and carbapenems were the mostly active drugs. *Haemophilus influenzae* in sputum is mostly sensitive to Carbapenems, Cefotaxime, Coamoxyclav and mostly resistant to Quinolones and Macrolides.

*H. influenzae* in BAL is mostly sensitive to Carbapenems, Piperacillin Tazobactam, Tigecycline, Cefoperazone Sulbactam Cefotaxime, Coamoxyclav and mostly resistant to Quinolones, Macrolides, Ceftriaxone. Blosser-Middleton *et al.,* (2003) and Zhanel *et al.,* (2000) stated in their study that Coamoxyclav and Macrolides were among the most potent drugs.

*Staphylococcus aureus* in both sputum and BAL is mostly sensitive to Coamoxyclav, Linezolid, Vancomycin followed by Teicoplanin, Tigecycline and mostly resistant to Ceftriaxone, Quinolone, Clindamycin, Macrolides. Maximum sensitivity to Vancomycin is supported by the study of Nwankwo and Nasiru (2011) but this study finding goes contrary to the study by Abdulhadi Sale Kumurya (2017) where Clindamycin and Macrolides were shown to be sensitive.

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