BRONCHIAL COLONISATION AND INFLAMMATION IN PATIENTS WITH CLINICALLY STABLE BRONCHIECTASIS – MICROBIOLOGICAL PATTERN AND RISK FACTORS

Dissertation Submitted for

M.D DEGREE EXAMINATION

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BONAFIDE CERTIFICATE

This is to certify that the dissertation '**Bronchial Colonisation and Inflammation in patients with clinically stable Bronchiectasis-Microbiological pattern and Risk factors**' is the Bonafide work done by Dr.R.Nedunchezhian during his MD(Tuberculosis and Respiratory Diseases) course from May 2012 to April 2015 at the Institute of Thoracic Medicine and Rajiv Gandhi Govt General Hospital ,Madras Medical College, Chennai-3

Prof.Dr.D.Ranganathan MD,DTCD,DNB [Chest Medicine]
Director, Institute of Thoracic Medicine,
Professor and Head, Department of Thoracic Medicine,
Rajiv Gandhi Govt General Hospital,
Madras Medical College,
Chennai-3

Prof.Dr.R.Vimala MD
Dean
Rajiv Gandhi Govt General Hospital
Madras Medical College, Chennai-3

DECLARATION

I hereby declare that the dissertation entitled "**Bronchial colonisation and inflammation in patients with clinically stable bronchiectasis-microbiological pattern and risk factors"** submitted for the degree of Doctor of Medicine in MD degree examination branch XVII Tuberculosis and Respiratory Diseases is my original work and the dissertation has not formed the basis for the award of any degree, diploma, associate ship, fellowship or other similar titles. It had not been submitted to any other university or institution for the award of any degree or diploma

Place :

Date : Signature of Candidate

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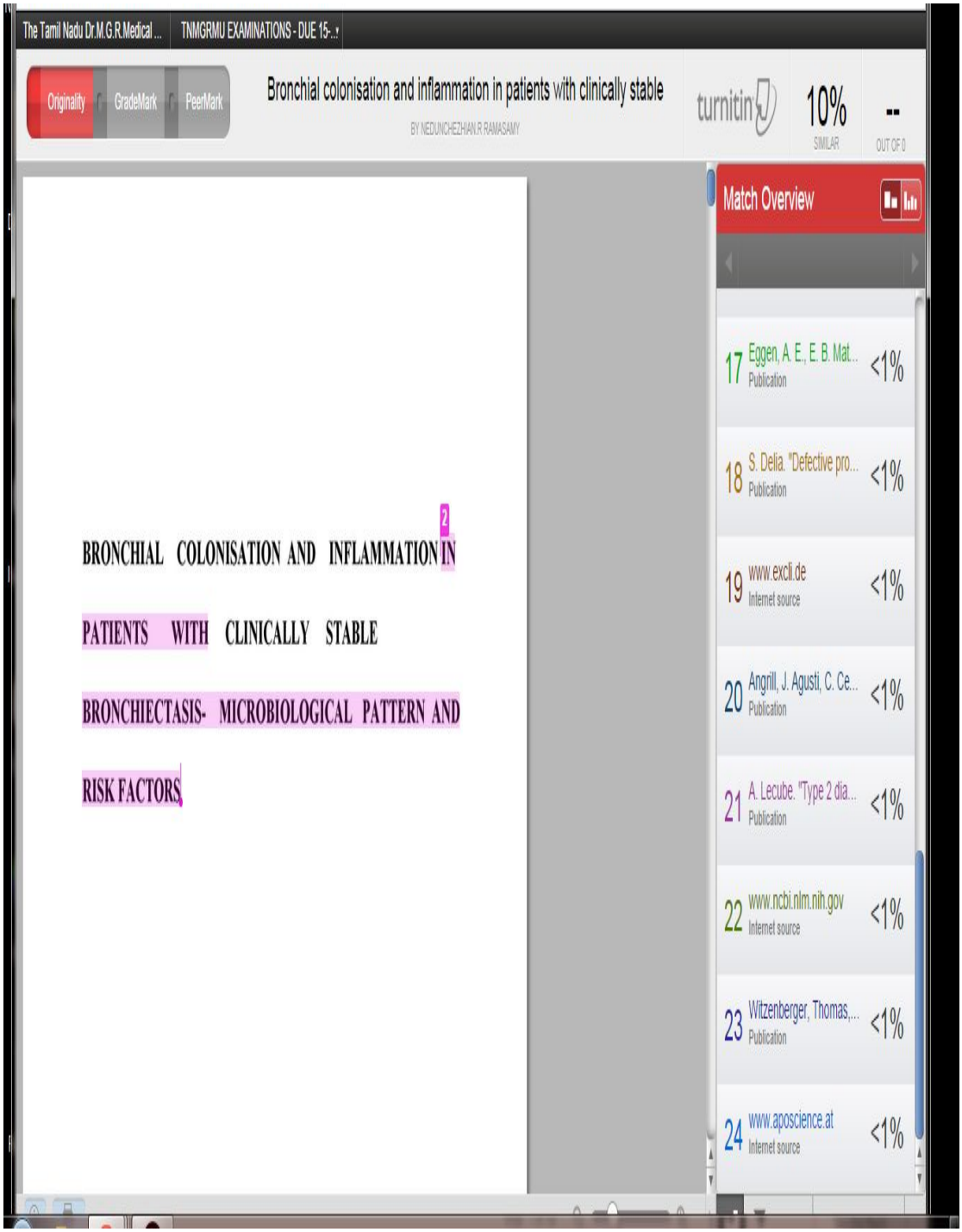
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BRONCHIAL COLONISATION AND INFLAMMATION IN PATIENTS WITH CLINICALLY STABLE BRONCHIECTASIS- MICROBIOLOGICAL PATTERN AND RISK FACTORS

CONTENTS

TITLE:

Bronchial Colonisation and Inflammation in Patients with Clinically Stable Bronchiectasis-Microbiological Pattern and Risk Factors

AUTHORS:

Dr.R.Nedunchezhian, Prof.Dr.D.Ranganathan,

Prof.Dr.A.Mahilmaran, Dr.V.Sundhar

Institute of Thoracic Medicine, Madras Medical College

INTRODUCTION:

Bronchiectasis is a chronic pulmonary disease characterized by an abnormal irreversible dilatation of one or more bronchi often with wall thickening. The current view of the pathogenesis of Bronchiectasis considers initial colonization of the lower respiratory tract by different microorganisms as the first step leading to inflammatory response characterized by neutrophil migration within the airways and secondary secretion of variety of tissue damaging oxidants and enzymes such as neutrophil elastase, myeloperoxidase and cytokines [interleukins] . Persistence of microorganisms in the airways because of impairment in mucus clearance may lead to a vicious circle of events characterized by chronic bacterial colonization, persistent inflammatory reaction and progressive tissue damage and morbidity life.

ABSTRACT:

In order to evaluate the Bacterial colonisation and level of bronchial inflammation in relation with bacterial colonisation; we did bronchoalveolor lavage of patients admitted with HRCT Chest[taken within 3 months] evidence of bronchiectasis and fulfill with inclusion criteria of study. The obtained sample was divided into 3parts-One part was sent to microbiology for bacterial culture ,second one was sent to pathology for Total and Differential cell count

estimation and third part was centrifuged and stored in 2 to 8'C temperature for IL-8 estimation

METHOD OF STUDY:

Continuous prospective study

RESULTS:

Among 90 study population analysis showed females were 58% and males were 42%. Cylindrical bronchiectasis 53%, followed by cystic bronchiectasis36% in predominance: and positive culture growth rate for sputum samples were 68% and BAL samples were 77%.The micro organisms isolated predominantly were H.influenza, Pseudomonas, Streptococci, Staphylocci and etc. Regarding airway inflammation, the total counts of BAL fluid were increased in all the patients with slight higher level in positive culture growth patients with slight Neutrophilic predominance. [90-210X10*3 cells in range and 120x10*3 cells in median]. The IL-8 measured showed increased level in all three groups comparing control groups indicating earlier establishment of inflammation in bronchiectasis. [81-835pg/dl in range and 556pg/dl in median]

CONCLUSION:

A. The risk factors identified for increased colonisation were 1.long duration of symptoms 2.Cystic and Varicose bronchiectasis 3. Presence of alcoholism and smoking

B. Sputum culture is non invasive, alternative to broncho alveolar lavage fluid culture.

KEY WORDS:

Bronchiectasis, Bacterial colonisation, Inflammation

INTRODUCTION

Bronchion-Greek word means wind pipe[1]

ektasis-means stretching out[1]

Bronchiectasis is a disease in which patients spends morbid life having dyspnoea and productive, often foul smelling sputum which produces social isolation and depressive states.[2.] In 1819 Rene Theophile Laennec first discovered Bronchiectasis in post-mortem examination of infant body who died of whooping cough[1], and he described, 'dilated bronchi lose their natural shape and present themselves under the form of cavity , capable of containing hemp seed, a cherry-stone, an almond, or even a walnut. The famous patient Williams Osler who died of road traffic accident on 1919, had respiratory symptoms for many years ,Dr.George Gibson described the bronchiectatic changes of William Osler lungs on post mortem examination with bronchography[1,].

In 1950 Lynne Reid described the pathological types of bronchiectasis with bronchography in 45 patients[1,3,7]

ICD-9 is the international disease classification code.

Article in European respiratory journal april-2009 titled as mortality rate in bronchiectasis patients[2], states the mortality rate of bronchiectasis in 12 year follow up period is 29.7% in the age group at 52

years. 70% cause of death in bronchiectasis is due to respiratory tract infection leading to respiratory failure[2,4]. The acute exacerbation of bronchiectasis is mainly due to growth of micro organisms and newer therapies like hypertonic solution like mannitol, Normal saline (up to maximum 7%) nebulisation and inhaled ciprofloxacin, gentamycin are under trial in reducing the exacerbation. By eliminating the micro organisms in bronchiectasis, the inflammation of airways and deterioration of lung function can be reduced; there by we can lead the bronchiectasis patients for morbidity nil life[5,6]

REVIEW OF LITERATURE

The primary mode of search for articles in this review was through the bronchiectasis related articles in journals like,' Thorax.bmj on dec 13 2013, ATS journals 1st Nov 2011, European Respiratory journal 2009, Journal of Inflammation Research 2013: 6 1-11 and from internet data bases and Google scholar. The general search terms were ,'Bacterial colonisation and inflammation, Exacerbation of Bronchiectasis , and Newer therapies in Bronchiectasis.',Immunology of Bronchiectasis, Broncho alveolar lavage technique, Microbiology of Bronchiectasis, Cell count in BAL fluid of Bronchiectasis, HRCT in Bronchiectasis in identifying aetiology, Pulmonary function in Bronchiectasis'.

The articles that were excluded from search are

1. The articles in language other than English for lack of comprehension

2. The articles based on drug trials in bronchiectasis

EPIDEMIOLOGY

The prevalence of Bronchiectasis in United States was 1.5/1000 cases in 1956, now the exact incidence of this disease is not clearly known. Now it is reduced due to

1. Improvement in living conditions of people
2. Frequent and early use of antibiotics
3. Childhood immunisation.[1,3,7]

But in countries with poor socio economic conditions and less health care generally available, bronchiectasis remains the major cause of morbidity and mortality. Pamela J.University of Chicago described as High prevalence of bronchiectasis has been reported in native Americans of North America, New Zealand and Western Samoans.[1,2,3] In U.S the prevalence of Non cystic fibrosis bronchiectasis in >65 years is 1106/100,000 and the prevalence increased to 8.7% from 2000-2007 in U.S. The prevalence of bronchiectasis is increased with increased age, in females and in Asian population. However it is unclear whether these differences are due to genetic predisposition to bronchiectasis or environmental factors are responsible[1,3,7].As per GOLD guidelines; on HRCT Chest investigation of chronic obstructive pulmonary disease patients, bronchiectasis was identified in 53% of patients .[2,4]

BRONCHIECTASIS

DEFINITION

Bronchiectasis is defined as abnormal permanent destruction and dilatation of one or more bronchi, often with wall thickening [1,3,7]

PREVALENCE IN GENDER

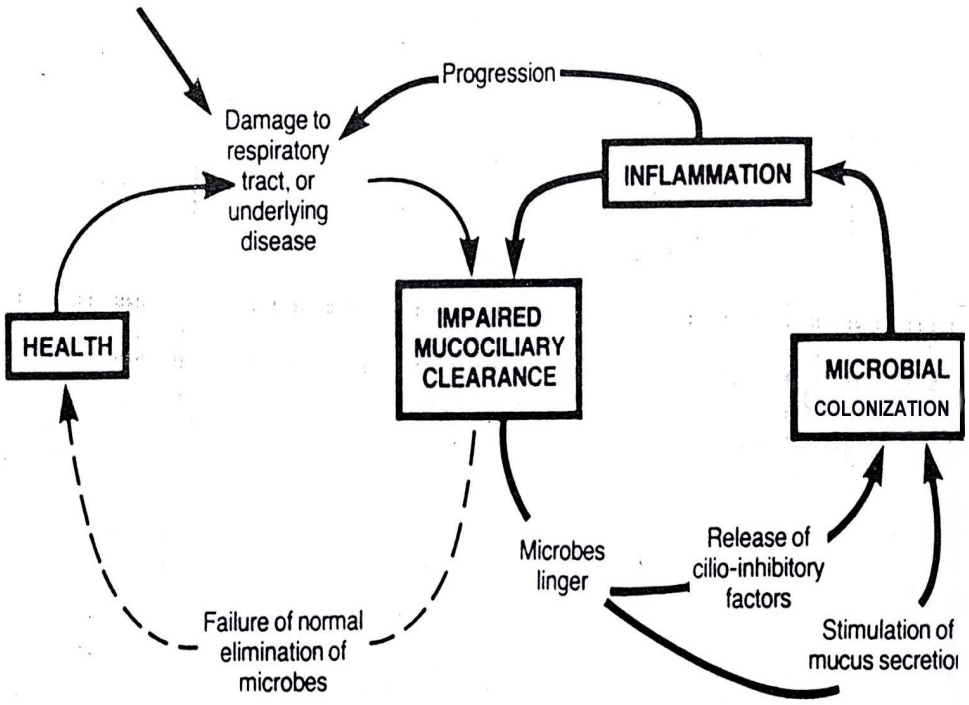
Females have high prevalence than males, the exact reason not clear: May be due to altered inflammatory immune response in females and environmental genetic and anatomical differences[1,3,7]

PATHOPHYSIOLOGY

Chronic bronchial sepsis has been used to describe the chronic bacterial infection of the impaired mucociliary action leads to microbial infection of lower respiratory tract that leads to release of inflammatory mediators. Sepsis is the condition in which bacteraemia occurs, where as this is rare in bronchiectasis because an exuberant immune response confines the infections to the lung.[3,8,9,10] In bronchiectasis, there is chronic inflammation in which lymphocytes predominate in the bronchial wall and Neutrophils in the lumen. As well as B Lymphocytes, plasma cells and CD4 T lymphocytes in the follicles, there is a well developed cell mediated immunity, with increased numbers of activated T lymphocytes, mainly of the suppressor/cytotoxic CD8 phenotype, antigen processing cells and macrophages. A proportion of the CD8 cells express a marker for cytolytic potential[3,11-17]

Mucus is poorly cleared from the bronchiectatic areas for several reasons. There is pooling in the abnormal dilated airways; ciliated cells are lost when the epithelium is damaged and mucus is less elastic more viscous and forms a vicious cycle.[3,7,18,19] .

Coles hypothesis



Coles hypothesis explained the vicious cycle in bronchiectasis. In bronchiectasis, impairment of mucociliary clearance due to chronic inflammation and some congenital causes leads to microbial colonisation of airways with poor elimination of microbes and secretions, leads to architectural damage leading to stagnation of secretion and this stagnation leads to microbial infections[1,3].

Large number of Neutrophils are attracted from the blood stream into the bronchial lumen in response to bacterial infection, by chemotactic products of bacteria themselves and also mediators from the host cells.[e.g, interleukin-8,C5a, Tumour necrosis factor alpha, leukotriene B4,]. Persistence of micro organisms leads to persistent Neutrophilic chemotaxis that leads to liberation of tissue destroying enzymes and cytokines like Proteinase,Elastase,Myeloperoxidase ,Tumour necrosis factor alpha[TNF alpha], and Interleukins 6,8,10,13. Proteinase, Elastase inhibits opsonin and destroys not only elastin but also destroys muscle layer of bronchi that is replaced by fibrous tissue leading to airway modelling'.[7-10,11-17] Serum levels of intercellular adhesion molecule(ICAM-1) and vascular adhesion molecule (VCAM-1) are elevated suggesting the endothelial activation occurs, probably within lung[13-17]. The cell counts in broncho alveolar fluid in normal people is 85% alveolar macrophages, 14% Lymphocytes and 1% will be Neutrophils and Eosinophils.[1,3,7]

In bronchiectasis the sub segmental airways are permanently dilated, tortuous and partially or totally obstructed by copious amounts of secretion. Structural proteins are lost from the bronchial wall and there is variable amount of fibrosis. The elastin layer of the wall is deficient or absent and the muscle and cartilage layer also shows signs of destruction. These changes weakens the wall leading to distortion of normal architecture.[7,11-12,20]

AETIOLOGY:

Congenital/Acquired[1,3,7]

Congenital causes:

Ciliary dysfunction-kartagener syndrome,

Cystic fibrosis

Mounier khun syndrome,

William Campbell syndrome

Young's syndrome,

Yellow nail syndrome

Alpha 1 anti trypsin deficiency

Primary immunodeficiency -Hypogammaglobulinemia

Secondary immunodeficiency-Malignancy

-post transplantation

Acquired causes:

a. Post infectious-

1. Bacterial-Bordetella pertusis,

Staphylococcus aureus

Klebsiella pneumoniae,

Hemophilus influenza

Streptococcus pneumoniae,

Mycobacterium tuberculosis

Mycobaterium avium intracellularae.

2. Viral-Measles, Adeno and Herpes virus

3. Fungal-Allergic bronchopulmonary aspergillosis

4. Parasitic-Trypanosomiasis

b.Post aspiration and inhalation injury

c.Connective tissue disease-Rheumatoid arthritis,Sjogren syndrome,

d.Autoimmune disease-Primary biliary cirrhosis.

e.Inflammatory bowel disease-Ulcerative colitis and Crohns disease

MICROBIOLOGY OF BRONCHIECTASIS:

C.Agusti et al study in Thorax.2002:57:1-15 and U .S Investigation into aetiology of bronchiectasis separately evaluated the microbiological flora from the bronchiectasis patients in 1.Sputum samples 2.Broncho alveolar lavage fluid and 3.Protected specimen brush. C.Agusti et al study identified the micro organisms as below

In sputum samples-26 potentially pathogenic micro organisms from 22 patients(52%) and 48 non potentially pathogenic micro organisms from 25 patients in total 112%; in 59 Broncho alveolar lavage fluid samples , 38 potentially pathogenic micro organisms from 33 patients (56%) and 25 non potentially pathogenic micro organisms from 33 patients (32%) ; and in 75 Protected specimen brush samples 61 potentially pathogenic micro organisms (75%) and 36 non pathogenic potentially pathogenic micro organisms from 22 patients (29%) and the concordant value for sputum and BAL culture was 75% with p value $=0.001$[11,12,20].

In U.S Investigation into aetiology of bronchiectasis study isolated 90% of micro organisms colonisation in 106 patients. Both studies states that in bronchiectasis infections are usually caused by bacterial species that form part of the nasopharyngeal flora like,H.infleunza, Morexella catarrhalis, Streptococus pneumonia and the opportunistic pathogen Pseudomonas. The initial infection by Pseudomonas aeruginosa is usually with a non mucoid strain, which becomes mucoid when the infection is chronic. P.aeruginosa is thought to be directly involved in the deterioration of pulmonary function i,e associated with extensive lung disease and severe airflow obstruction. Isolation of P.aeruginosa could be just marker of disease severity rather than actual disease. When chronic bronchial infection occurs with commensal or opportunistic bacteria it reflects the severity of impairment of lung defences rather than the virulence of organisms.[3,7,21,22]

Both studies classified the micro organisms into potentially pathogenic micro organisms [PPM] , and Non potentially pathogenic micro organisms[Non PPM] depending upon the disease producing capacity.

King and Colleagues also isolated and classified the micro organisms.

Potentially pathogenic micro organisms [PPM]: Following micro organisms were classified as PPM[8-12]

1. Hemophilus influenza 47% and para influenza 5%

2. Pseudomonas [various strains] 12%

3. Klebsiella species 6%

4. Staphylococcus aureus 4%

5. Morexella catarrhalis 8%

6. Escherichia coli 3%

7. Streptococcus pneumoniae 7%

8. Proteus species

9. Aspergillus species

Non potentially pathogenic micro organisms [NonPPM]: The following micro organisms were classified as non PPM.[3,7,11]

1. Streptococcus viridans

2. Corynebacterium species

3. Coagulase negative Staphylococcus aureus[CONS]

4. Enterococcus

As per the previous studies the cut off point for significant bacterial culture growth positivity for protected specimen brush [PSB] was more than 10^{x2} cfu; for Sputum 10^{x5} cfu and for Broncho alveolar lavage fluid was more than 10^{x3} cfu [3,7,8,11,12]

AIRWAY INFLAMMATION IN BRONCHIECTASIS

In this study, inflammation of airway was studied mainly based on the following two studies.

1. Joaquim Angrill et al study in .ATS-volume 164.issue 9, Nov-1-2001

2. David A Bergin .Journal of inflammation research 2013:6 1-11

David A Bergin et al study did Interleukin4, 10 and cell counts in 15 microlitre of BAL fluid at 405nm, from 45 bronchiectasis patients and the values were as below.

Total cell counts- median cell count was $23x10^3$ cells and 10-$1086x10^3$ cells in range from Non colonising patients to potentially pathogenic micro organisms growth patients; and the Neutrophilic pattern was observed in 67(in median) 56-88 (in range) patients. Macrophage pattern was observed in 27(median) 8-38(in range)[11,12,20].

Interleukin 4: 28.3pg/ml (in median) against the control 2.12pg/ml with p value =0.9. Interleukin 10: 151.3pg/ml (in median) against control 0.1pg/ml with p value =0.05.[11,12,20]

Jouquim Angrill et al study also evaluated the bronchial inflammation and the values were as below.

Total count: in control group $1.1x10^5$ cells in median

in Non colonised group-$24.75x10^5$ cells in median

in PPM growth group - $22x10^5$ cells in median[11,12,20]

Interleukin-8: in control -0-31pg/ml in range. 195pg/ml in PPM group (median) and 0-5520pg/ml in range with p value =0.001[11,12,20.]

Above studies states that Non colonised patients showed a more intense bronchial inflammatory reaction than did control subjects. This inflammatory reaction was exaggerated in patients colonised by micro organisms with potential pathogenicity, with a clear relationship the bronchial bacterial load and showed that patients with bronchiectasis in a stable clinical condition present an active neutrophilic inflammation in the airways that is exaggerated by the presence of MPP, and the higher the bacterial load the more intense the inflammation.[23-26]

RISK FACTORS FOR BACTERIAL COLONISATION

J.Angrill et al study states that, the people with normal pulmonary function i.e. with FEV1> 80% were present in n=24 of the study population. FEV1< 65% was observed in n=11 of the study population and the later group had higher BAL level of interleukin 1beta (115pg/ml), TNF-alpha (41pg/ml), IL-10 (7.3pg/ml) suggesting the relationship between the level of inflammation and poor pulmonary function. A significant direct relationship between the bacterial load and the Neutrophils and Interleukin-8 was observed i.e. more bacterial load leads to more inflammation[11,12,20,29,30]

C Agusti study et al states that following factors were associated with increased risk of microbial colonisation.

1. presence of chronic expectoration[long duration of symptoms]
2. evidence of cystic and varicose bronchiectasis in HRCT scan
3. diagnosis of bronchiectasis < 14 years of age
4. presence of sinusitis and lung function FEV1< 80%.

Smoking increases the bacterial infections of Respiratory Tract;

by inhibition of Monocytes,Macrophages and Dendritic cells activity [innate immunity], in smokers immunoglobulin E will be increased with lowered level of immunoglobulin G[altered adaptive immunity]and Impairment of ciliary functions.[31]

INTERLEUKINS -8

Interleukin-8 (IL-8) is a member of a family of structurally-related low molecular weight proinflammatory factors known as chemokines. IL-8 is produced by stimulated monocytes but not by tissue macrophages and Tlymphocytes.[1,3,7,11.] IL-8 is a non-glycosylated protein of 8 kDa (72 amino acids). It is produced by processing of a precursor protein of 99 amino acids. Eventhough different interleukins were liberated into the site of inflammation, IL-8 which is mainly responsible for Neutrophilic chemotaxis in bronchiectasis.[1,3,7,11,32-36] Previous studies stated that Broncho alveolar lavage fluid level of IL-8 was significantly raised in Bronchiectasis in comparison with serum levels of IL-8; and it showed the inflammation was compartmentalised[11,12,20,32-36]

DEFINITIONS FOR

1.Stable Bronchiectasis

C Agusti et al study described, patients with bronchiectasis who have not received antibiotics within 4 weeks and not hospitalised within 2 months were labelled as stable Bronchiectasis[11,12,20].

2.Good quality sputum

Murray Washington criteria grade v i.e. >25 Neutrophils and <10 epithelial cells per field using low power magnification lens[x100] was described as good quality sputum[11,12,20].

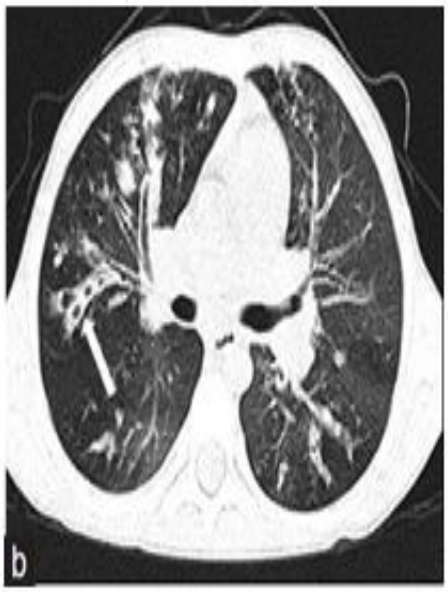
3.HRCT Chest score

[Based on Reiffe and colleagues criteria for HRCT Chest scoring in bronchiectasis] The presence and extent of bronchiectasis in each lobe was graded using a scale from 0 to 3; where 0=no bronchiectasis, 1=involvement of one segment, 2=involvement of more than one segment and 3= presence of cystic bronchiectasis. By considering lingula as a separate lobe maximum points were 18. The score in percentage was derived by total number of points divided by maximum points multiplied by 100][11,12,20]

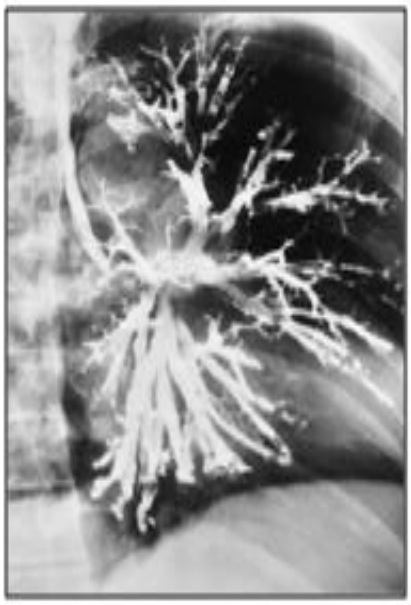
BRONCHIECTASIS TYPES:[REID CLASSIFICATION]

1.Tubular/Cylindrical

Mildform.Regularly outlined, dilated in diameter or straight walls with abrupt end[37,38].Cylindrical bronchiectasis is much more common nowadays in which the damage to bronchial wall is less severe. This type of bronchiectasis is usually bilateral and may be diffuse has been described as modern bronchiectasis.[1,3,7,39,40]



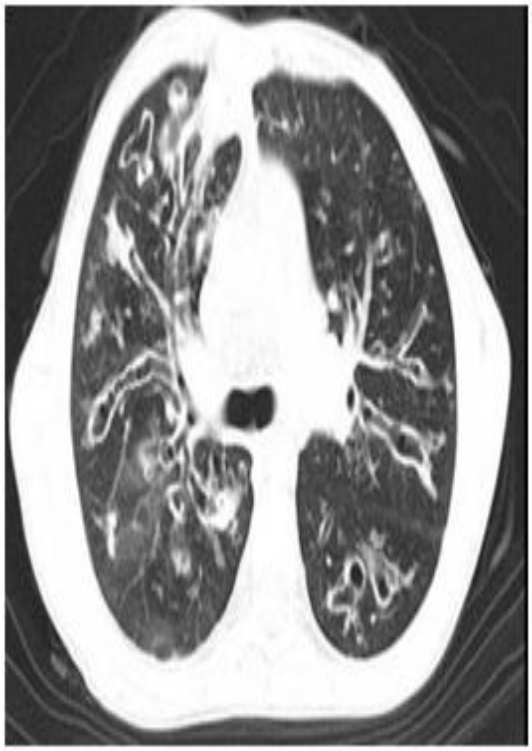
Picture-1:Showing the cylindrical Bronchiectasis in HRCT Chest.



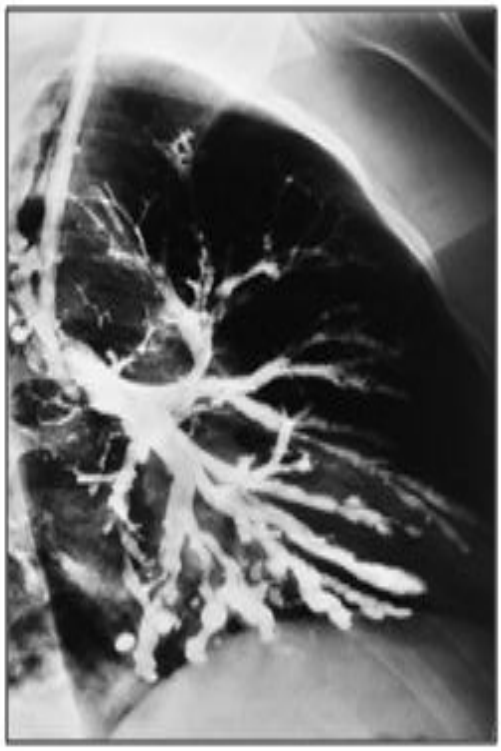
Picture-2:Showing the cylindrical Bronchiectasis in Bronchography.

2.Varicose/Fusiform

Irregular dilatation with outpouching and tortousity of airways [string of pearls appearance][17.] In Varicose bronchiectasis there are local constrictions superimposed on cylindrical changes[1,3,7,39,40].



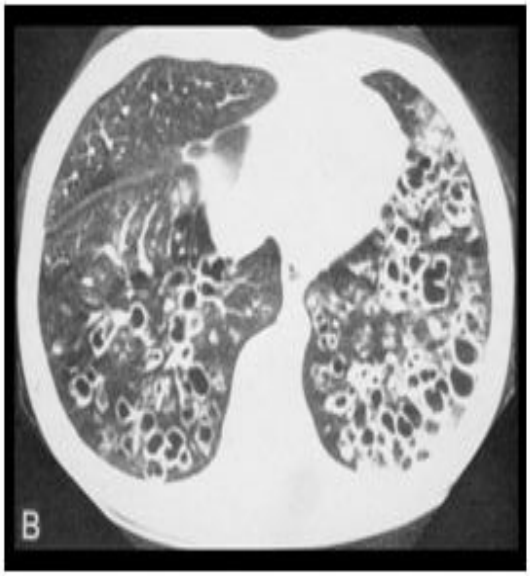
Picture-3: Showing the varicose Bronchiectasis in HRCT Chest.



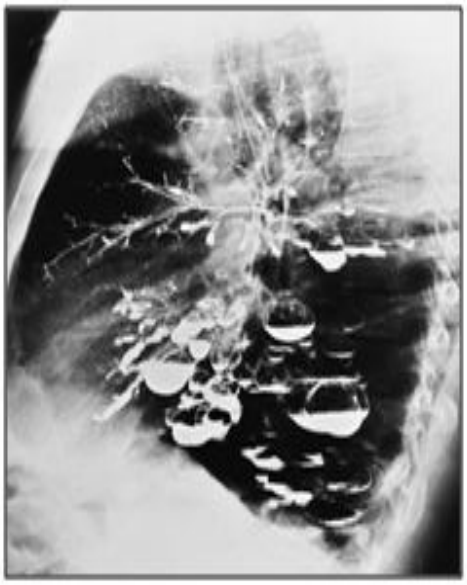
Picture-4:Showing the varicose Bronchiectasis in bronchography.

3.Cystic/Saccula

Severe form. Cystic dilatation of airways[bunch of grapes appearance][37-38] Cystic or saccular bronchiectasis in which severe loss of bronchial wall structure leads to large, balloon like dilatation, are now infrequently seen in developed countries. This bronchiectasis often follows severe lung infection and was characterised by production of large volume of sputum and finger clubbing.[1,7,39,40]



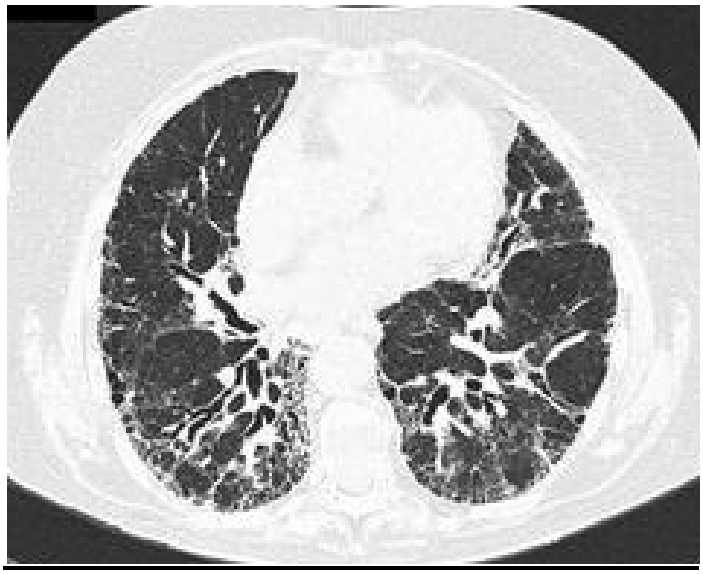
Picture-5:Showing the cystic Bronchiectasis in HRCT Chest



Picture-6:Showing the cystic Bronchiectasis in bronchography.

Other type

Traction bronchiectasis occurs in fibrotic lung conditions such as fibrosing alveolitis in which the airways are pulled apart by the fibrous process.[1,7,39,40]



Picture-7: Showing the Traction bronchiectasis in HRCT Chest.

DIAGNOSIS

It is based on History, clinical examination and is confirmed by radiology[1,3,7]

1. History of Chronic expectoration [purulent or Mucopurulent sputum]

2. Positive Family history and consanguinity[1,3,7]

On examination: bilateral pan digital finger clubbing,Halitosis[1,3,7]

On auscultation: coarse leathery persistent crackles heard on both phases of respiration. Chest X ray: Ring shadows on end on view of airway. Tramline shadow on side on view of airway[1,3,7,11,12].

HRCT Chest: the investigation of choice

1.The non tapering of bronchi >2cm from costal pleura

2.Visualisation of bronchi within 1 cm to the visceral pleura

3.Bronchial wall dilatation and thickening seen as signet ring sign

Other signs- Mosaic attenuation pattern, Tree in bud appearance, Mucus plugging of airways, lobar volume loss.[39,40]

BRONCHO ALVEOLAR LAVAGE TECHNIQUE

1.SAMPLING OF CELLS

Broncho alveolar lavage with using Fiber optic bronchoscope was introduced on 1974 by Raynolds and Newballs. BAL is a safe procedure in human research and clinical implications. In BAL sampling 1.the cells in alveolus in contact with broncho alveolar lavage fluid are sampled, 2. the cells in alveoli not in contact with BAL fluid are not sampled , 3.airway cells that lines the bronchial walls are sampled by washing[41-46].

2.UNDERLYING CONDITION

The broncho alveolar lavage is useful in following conditions as diagnostic and therapeutic

1.Interstitial lung diseases-

2.In identifying Infectious aetiology including Pneumocystis pneumonia

3.Asthma and Chronic obstructive pulmonary disease

4.Pulmonary alveolar proteinosis

In Cigarette smokers, BAL fluid macrophages will be increased often to 10 fold than non smoker[41-46]

3.SUCTION PRESSURE

During BAL if we use high pressure suction, the airway wall will collapse. So the recommended suction pressure is < 60mmHg to 100 mmHg.[41-46]

4.VOLUME OF FLUID INSTILLED

The cellular and protein component of BAL fluid will vary depending upon the amount of fluid instilled and the amount of fluid aspirated. For good cell count and protein analysis, at least 100 ml of fluid [0.9 % Normal saline is ideal] to 150ml of fluid to be used. About 50 to 60 % of instilled fluid should be aspirated[41-46]

5.HANDLING OF ASPIRATED FLUID

The first aliquot about 20 ml contain more epithelial cells and less cells than subsequent aliquot, So for estimating cell count and protein, the first aliquot 20ml is better to be discarded ; But in microbiological analysis , the first aliquot to be sent to Microbiology for better result as the first aliquot contain more epithelial cells and debris [41-46]

6.POSITION OF PATIENT

As the broncho alveolar lavage is mainly dependant on gravity aided drainage, patient position can affect the lavage. Supine position is the ideal position for lavage with gravity aided and less suction pressure.[41-46]

7.AREA OF LAVAGE

Previously the BAL was done mainly from lower lobes. But in diffuse lung disease BAL should be done from the disease affected parts of lung and even from all lobes of lung.[41-46]

8.STORAGE OF FLUID

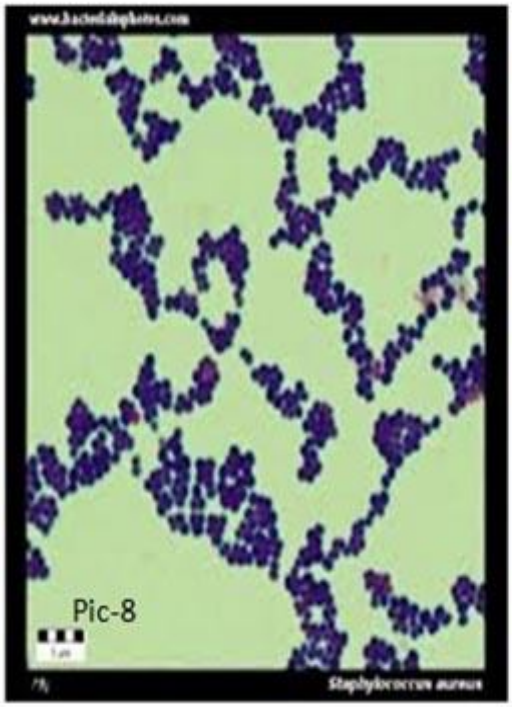
The obtained fluid by broncho alveolar lavage is to be stored in 2 to 4 degree Celsius for cell count and protein determination.[41-46]

9.SAFETY OF BAL

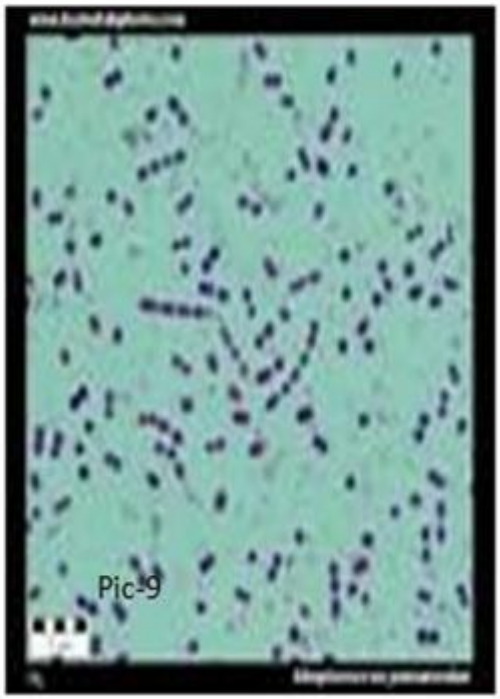
Broncho alveolar lavage is safe and effective in human research and clinical conditions like Asthma, COPD,Ventilator associated pneumonia and Adult respiratory distress syndrome. The important complication associated with this procedure is 1. Hypoxia 2. Fever. The bronchoscopist should get a detailed history of patient i.e. previous procedure and complications, trauma, associated medical conditions and medicines using, coagulation studies and ,Renal and Liver function test for safe procedure..However the meticulous asepsis technique is to be practised always.[41-46]

THE MICROSCOPIC PICTURES OF MICRO ORGANISMS

Staphylococcus aureus ; Streptococcus pneumoniae

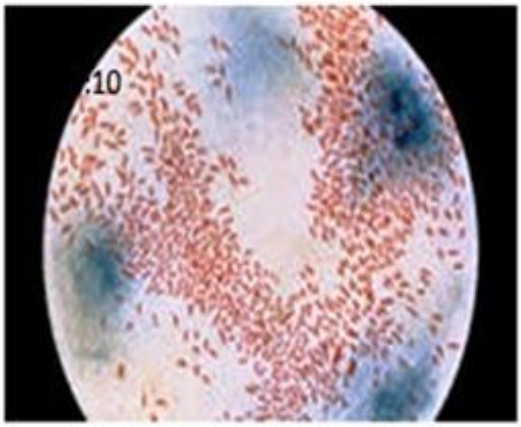


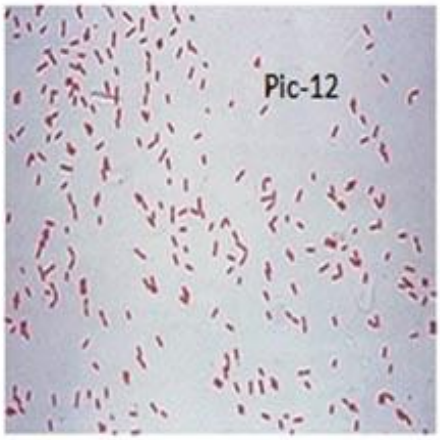
Picture 8: Showing the staphylococcus Aureus- gram positive , round cocci, grapes Like cluster appearance on gram staining

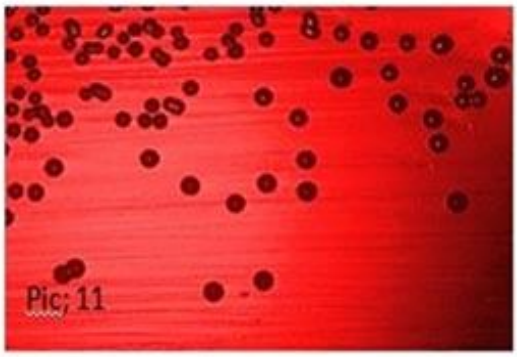


Picture 9: showing the streptococcus Pneumoniae , round shaped, gram po Cocci occurs in chain.

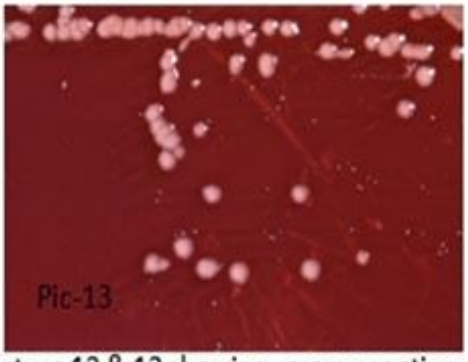
H.influenza and Pseudomonas aeruginosa





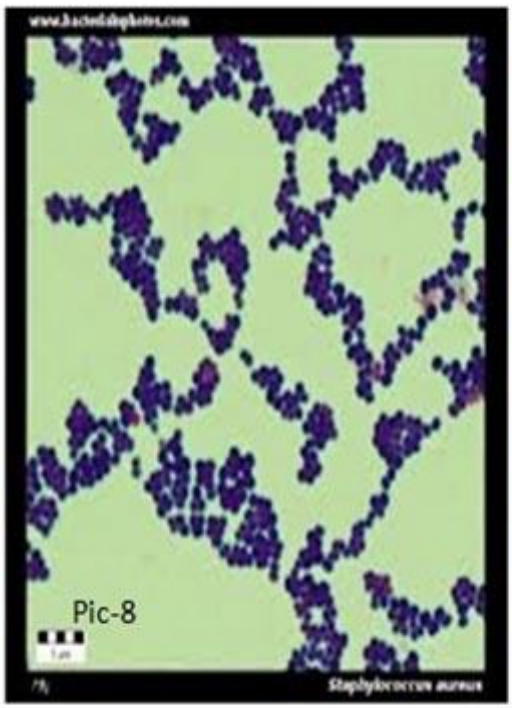


Picture 10 & 11 showing H.influenza in 40x, 100x lens- rod shaped, coccobacilli,



Picture 12 & 13 showing gram negative Pseudomonas aeruginosa in 40x, 100x lens- rod shaped.One or more polar flagella

Klebsiella pneumoniae and Morexella catarrhalis



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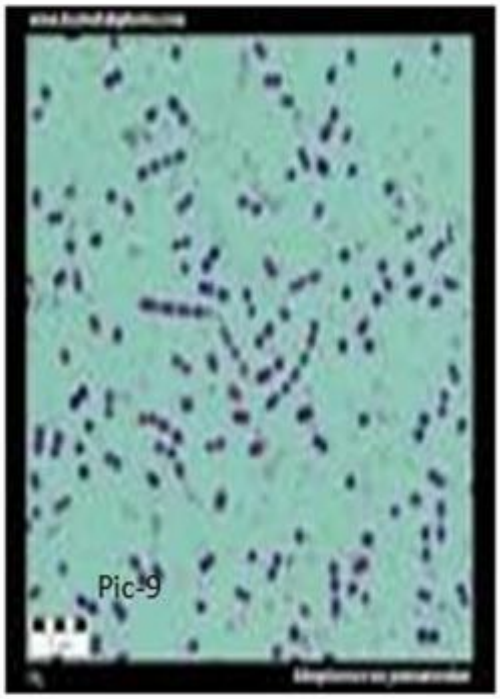

Picture 9: showing the streptococcus Pneumoniae , round shaped, gram po Cocci occurs in chain.

British Thoracic Society Guidelines

1.Sputum Microbiology:

1. All children and adults with bronchiectasis should have assessment of lower respiratory tract microbiology

2. Persistent isolation of staphylococcus aureus and or pseudomonas leads to consideration of underlying ABPA or cystic fibrosis

3. To get the maximum yield of H.influenza and Streptococcus pneumoniae the sputum sample should reach the lab within 3 hours

2.Pharmacotherapy

1.Inhaled B_2 agonist and anti cholinergic therapy will improve lung symptoms and function

2.No role of routine use of methylxanthines in bronchiectasis

3.No evidence of use of interleukin receptor antagonist or other anti inflammatory drugs

4.It does not support routine use of inhaled corticosteroid in adults(out side of use for those having additional bronchial asthma)

3.Indications for Bronchoscopy

[BTS] British Thoracic Society guidelines recommends FOB in following conditions not as routine

In localised bronchiectasis-to exclude proximal obstruction

In patients whose serial sputum culture is negative and who is not responding to treatment and HRCT features suggestive of Atypical mycobacterial infection and whom sputum culture is negative

STUDY JUSTIFICATION:

The main Goal in treating Bronchiectasis[as per BTS guideline]

1.To identify and treat the underlying cause to prevent disease progression

2.To improve or maintain lung function

3.To reduce exacerbation

4. To improve quality of life by reducing daily symptoms

Hence this study aims to evaluate the microbiological pattern in patients with bronchiectasis and study the appropriate antibiotic to be administered to eliminate the bacterial colonisation and reduce the bronchial inflammation. Comparing previous studies in this study, i have excluded the pharyngeal swab culture because of, while on doing pharyngeal swab the violent coughing of patients, the chance investigator developing infection from patient is high; and Protected Specimen Brush is also not done due to cost; In this study sputum culture and Bronchoalveolar fluid culture were done and compared the concordant value for sputum and broncho alveolar lavage fluid culture.

AIMS & OBJECTIVES

1.To compare the Sputum culture and Bronchoalveolar fluid culture yield

2.To analyse the potential risk factor associated with presence of pathogenic microorganism in airway

3.To evaluate the Bronchial inflammatory response and its relationship to bacterial colonisation in Bronchiectasis

STUDY DESIGN

This is prospective continuous study to evaluate 1.the Bacterial flora from Lower Respiratory tract of Bronchiectasis patients who attended the Thoracic medicine outpatient department with diagnosis confirmed by radiologist.

2.To assess the Risk factor for Bacterial colonisation

3.To evaluate the Airway inflammation and its relation with colonisation

Broncho alveolar lavage was done as invasive procedure in 90 patients with bronchiectasis and from 6 patients admitted with chronic upper respiratory symptoms as laboratory control in Interleukin-8 estimation.

Patients were selected randomly

STUDY PERIOD

This study was done for a period of 7 months from February 2014 to August 2014

STUDY POPULATION: 90 patients

STUDY CENTRE

This study was done in Rajiv Gandhi Govt General Hospital, Madras Medical College, Chennai-03

INCLUSION CRITERIA:

1.Patients admitted with HRCT Chest diagnosis of Bronchiectasis{non cystic fibrosis}

2.Age >14 years

3.SpO_2> 90% in Room air

EXCLUSION CRITERIA

1.Patients with complications like Hemoptysis, Lung abscess and Amyloidosis.

2.patients with Cystic fibrosis.

3.SPO2<90% in room air.

4.Patients with prior Hospitalisation within 2 months and had antibiotic within 4 weeks.

5.Patients with cardiac illness and Recent Myocardial infection

6. Patients with Renal failure

MATERIALS AND METHODS

Proforma was designed and ethical committee clearance was obtained.

The nature and purpose of the study was explained in detail to all the study Patients and written informed consent was obtained from all of them included in this study.

Data collection was done as per the proforma

STUDY PROCEDURE:

Patients admitted with HRCT Chest taken within 3 months and confirmed bronchiectasis were studied with

1.Sputum for-

a)Modified Zhiel Neelson staining for Acid Fast Bacilli smear in RNTCP Lab

b)Bacterial culture and sensitivity in Microbiological Lab

c)Gram staining in Microbiology Lab

2.Spirometry to asses Pulmonary Function

3.Broncho alveolar lavage with Fiber optic bronchoscope

METHODOLOGY

Patients admitted with HRCT Chest evidence of Bronchiectasis taken within 3 months duration were evaluated for study after inclusion and exclusion criteria analysis. Informed consent was obtained from all the patients and from parents of patients with age <18 years. The study purpose and methods, procedures were all explained to the patients in their mother tongue. On the day of admission after obtaining complete clinical history and complete clinical examination was done; the type of bronchiectasis as per Reid classification was noted and HRCT Chest scoring was done as per Reiff and Colleagues criteria; and after doing base line investigation, sputum was given for 2 samples for Acid fast bacilli smear in RNTCP Lab, and One sample for bacterial culture sensitivity and Gram staining in Microbiology lab . Good quality sputum was accepted as per the Murray Washington grade V classification. Other criteria used for clinically assessing good quality sputum-was Barlette criteria which consists of a) Mucus b) Mucopurulent c) Purulent d) Blood stained -four types of sputum . In which Purulent type sputum yielded high bacterial culture growth rate and considered as good quality sputum. After sputum culture, patients were allowed to undergo pulmonary function test with spirometry.

Pulmonary function test:

Spirometry was performed using a computerized Easy one spirometer.Patient was made to sit or erect and asked to wear a nose clip and spirometry was performed fulfilling the acceptability and reproducibility criteria according to American thoracic society recommendation.[31]

The parameters measured in spirometry include Forced Vital Capacity (FVC), Forced expiratory volume in 1 second (FEV1), ratio of FEV1 to FVC (FEV1%).

Lung function impairments were classified as

1.Normal spirometry:

FEV1 and FVC>80% and FEV1/FVC>70% predicted.

2.Obstructive pattern:

FEV1<80% and FEV1/FVC<70% predicted.

3.Restrictive pattern :

FEV1 and FVC <80% and FEV1/FVC ratio >70% predicted.

4.Mixed pattern:

FEV1 and FVC<80% and FEV1/FVC ratio<70%.

The Bronchoalveolar lavage was done on the next day of admission. Topical anaesthesia was achieved by nebulisation of 8ml of 5%Lignocaine for 15 minutes. Bronchoalveolar lavage was done with 150 ml of 0.9%Normal saline divided into three alliquots each consists of 50 ml. Broncho alveolar lavage was done with Pentax 18P Fiber optic bronchoscope [pentax india private ltd] from Bronchiectasis affected part of lung which was identified as per HRCT Chest. About 60-70% of administered fluid is aspirated. The obtained fluid was mixed well in a single sterile container and divided into three parts and patients were managed with standard treatment protocol.

First part was sent to Microbiology lab for bacterial culture

The samples were serially diluted [dilutions of 1:10, 1:100, 1:1000] and plotted in Blood agar, Nutrient broth agar, Chocolate agar,Wilkins-Chalgren ,and Saburauds agar.The cultures were evaluated for growth after 48 hours .Negative cultures for Bacteria were discarded after 5 days and for Fungi after 4 weeks. Susceptibility testing was performed using broth micro dilution or Beta lactamase test and classified as sensitive and as Resistant as per the National committee for clinical laboratory standards, Performance standards for antimicrobial susceptibility testing.[5]

Second part was sent to Pathology lab for Cell Count[Total and Differential cell count] estimation;- The obtained fluid was assessed macroscopically for colour and volume, and the fluid was filtered with a layer of sterile gauze and centrifuged for 15 to 20 minutes at 500g and then resuspended in sterile saline. Total count was assessed in Newbauer chamber. Direct smear and cytopsin smear were prepared. The cytopsin preparation was centrifuged at 200g for 6minutes. The cytopsin smear was air dried. Both direct and cytopsin smear were stained with modified wright stain. About 100 to 200 nucleated cells from 5 or more fields of cytopsin preparation were examined in 100xlens for differential cell count[47-49].

Third part was centrifuged and stored in Temperature 2 to 4 degree Celsius for IL-8 estimation. After obtaining 90 samples from Bronchiectasis patients and six samples from patients presented with upper respiratory tract symptoms [as laboratory control] ; all the centrifuged and stored samples were submitted for Interleukins estimation in National Institute of Research in Tuberculosis[NIRT] Chennai by Elisa method with RayBio Human IL-8 ELISA[Enzyme linked immunosorbent assay] Kit.

INTERLEUKIN-8 ESTIMATION:

ELISA PROCEDURE

IL-8 levels in BAL fluid samples were measured using ELISA (Ray Bio Human IL8 ELISA Kit) as per the manufacturer's instructions. Briefly the steps include:

1.All reagents and samples were brought into room temperature (18-25 degree Celsius)before use.

2.**Preparation of standard**

Briefly spin the vial of Human IL-8 standard.800 micro litre 1x assay diluent buffer was added into human IL-8 standard vials to prepare 50ng/ml standard. The powder was dissolved thoroughly by gentle mix,10 microliter IL-8 standard from the vial of human IL-8 standard into a tube with 823microliter of assay diluent buffer to prepare a 600pg/ml stock standard solution. 400microlitre 1x assay diluent buffer to prepare a 600pg/ml stock standard solution. 400 microlitre 1x assay diluent buffer was added into each tube. Using the stock standard solution a dilution series was produced as below. Each tube was mixed thoroughly before the next transfer. 1x assay diluent buffer served as the zero standard (0 pg/ml)

Preparation of BAL Sample

All samples were centrifuged at 2000 rpm for 10 minutes, 100 microlitre of supernatant was diluted with equal volume (diluent factor:2) of 1x assay diluent buffer before use.

3.Addition of sample/standard

100 microlitre of each standard and sample was added into appropriate wells. Plate was covered well and incubated overnight at 4degree Celsius with gentle shaking.

4.Addition of secondary antibody

The solution was discarded and washed 4 times with 300 microlitre of 1x wash buffer. For every wash plate was inverted and blot it against clean paper towels. 100 microlitre 1x prepared biotinylated antibody was added to each well and incubated for 1 hour at room temperature with gentle shaking.

5.Conjucate addition

After incubation the plates were washed as in stage 4, to each well 100 microlitre of conjucate (streptovidin-horse radish peroxidase was added in 1 in 600 dilutions and incubated at room temperature for 45 minutes with gentle shaking.

6.Substrate addition

The plates were again washed and 100 microlitre of substrate TMB, one step substrate reagent was added to each well and incubated at room temperature for 30 minutes till the blue colour developed with gentle shaking.

7.Arresting the reaction

The reaction then was stopped using 50microlitre of stop solution. The optical density was measured at 450 nm on ELISA reader (molecular devices USA), the mean of the sample values were subtracted from the reading. The unknown concentration of cytokines in the samples were evaluated by plotting the unknown optical density values against the standard curve. The IL-8 concentration in BAL samples were determined by referring to a standard curve and expressed as pg/ml.

STATISTICAL ANALYSIS

All the collected data's were incorporated into microsoft XL sheets, statistical analysis was done with the help of a professional statistician. Broncho alveolar lavage fluid culture as the gold standard, the BAL fluid culture was analysed with demographical factors and inflammatory cells including Interleukin-8 with Fischer scale.

Fischer exact p value = < 0.05 is considered as high significant

> 0.05 is considered as weak significant.

OBSERVATION AND RESULTS

AGE DISTRIBUTION

Table 1: Showing age distribution

Age in years	Frequency	Percent(%)
16-25 years	10	11.1
26-35 years	17	18.9
36-45 years	29	32.2
46-55 years	15	16.7
56-65 years	16	17.8
66-75 years	3	3.3
Total	90	100

Among the 90 patients in the study group starting from 16 years to 75 years; the patients with age 36-45 years were presented in majority (32%) followed by age group in 26-35 years , followed by56-65 years ,followed by 46-55 years, followed by 16-25 years and lastly 66-75 years

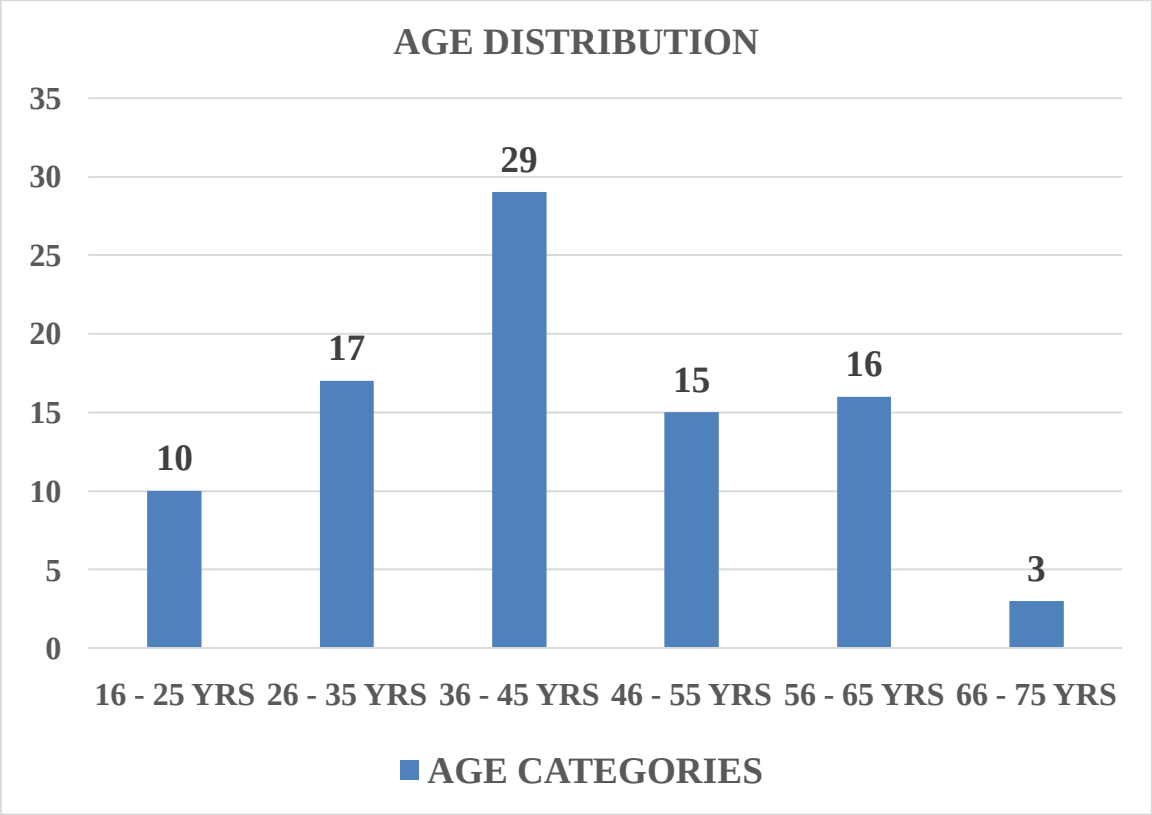

Chart 1: Bar diagram showing age distribution

Among the 90 patients in the study group starting from 16 years to 75 years; the patients with age 36-45 years were presented in majority (32%) followed by age group in 26-35 years , followed by56-65 years ,followed by 46-55 years, followed by 16-25 years and lastly 66-75 years

SEX DISTRIBUTION

Table 2: Showing sex distribution:

Sex	Frequency	Percent(%)
Male	38	42.2
Female	52	57.8
Total	90	100.0

Among the 90 patients in this study population 57.8% were females and 42.2% were males

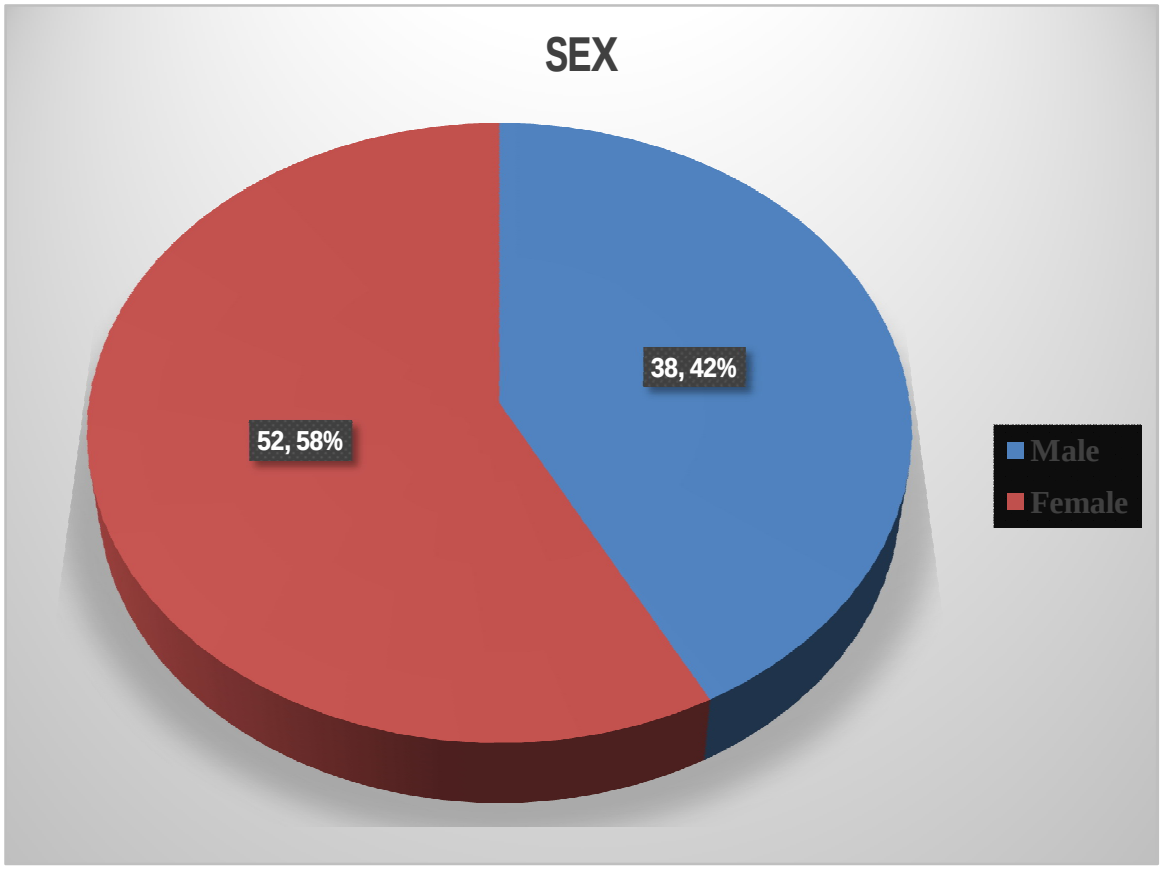

Chart 2: Pie diagram showing sex distribution

Among 90patients, 58% 0f patients were females and 42% of patients were males

DURATION OF SYMPTOMS

Table 3 : Showing description of duration of symptoms with bronchoalveolar Lavage fluid culture

Symptom duration	No growth	Percent %	Non PPM	Percent %	PPM	Percent %
< 1 year N=6/6.6%	5	83.5%	1	16.5%	0	0.0
1-5 years N=59/65.4 %	15	25.4%	3	5.6%	41	69%
> 5 years N=25/27%	1	3.7%	2	8.3%	22	88%

Based on the duration of symptoms at the time of admission, a) 65% of patients had symptoms from 1 year to 5 years; and their BAL fluid culture reported as no growth in 25% of patients, 5.6% positive for NonPPM growth and 69% were positive for PPM growth

b) 27% of patients had symptoms more than 5 years maximum up to 30 years; and their BAL fluid culture reported as 88% positive for PPM growth, 8% positive for NonPPM growth and 4% were no growth.

c) 6.6% of patients had symptoms less than 1 year and 83.5% of their BAL fluid cultures were reported as no growth.

SMOKING DISTRIBUTION

Table 4 : Showing distribution of patients with smoking history

Smoking	Frequency	Percent (%)
Smoker	11	12.2
Ex smoker	6	6.7
Non smoker	73	81.1
Total	90	100.0

Out of 90 patients in this study population 12% were smokers and 6.7 were past smoker and 73% were non smoker

ALCOHOLISM DISTRIBUTION

Table 5 : Showing distribution of patients with history of alcoholism

Alcoholism	Frequency	Percent (%)
Alcohol consumer	12	13.3
Ex alcoholism	4	4.4
Non alcoholic	74	82.2
Total	90	100.0

Out of 90 patients in this study population 13.3% were alcohol consumers and 4.4% were past alcoholism history and 82 % were non alcoholic.

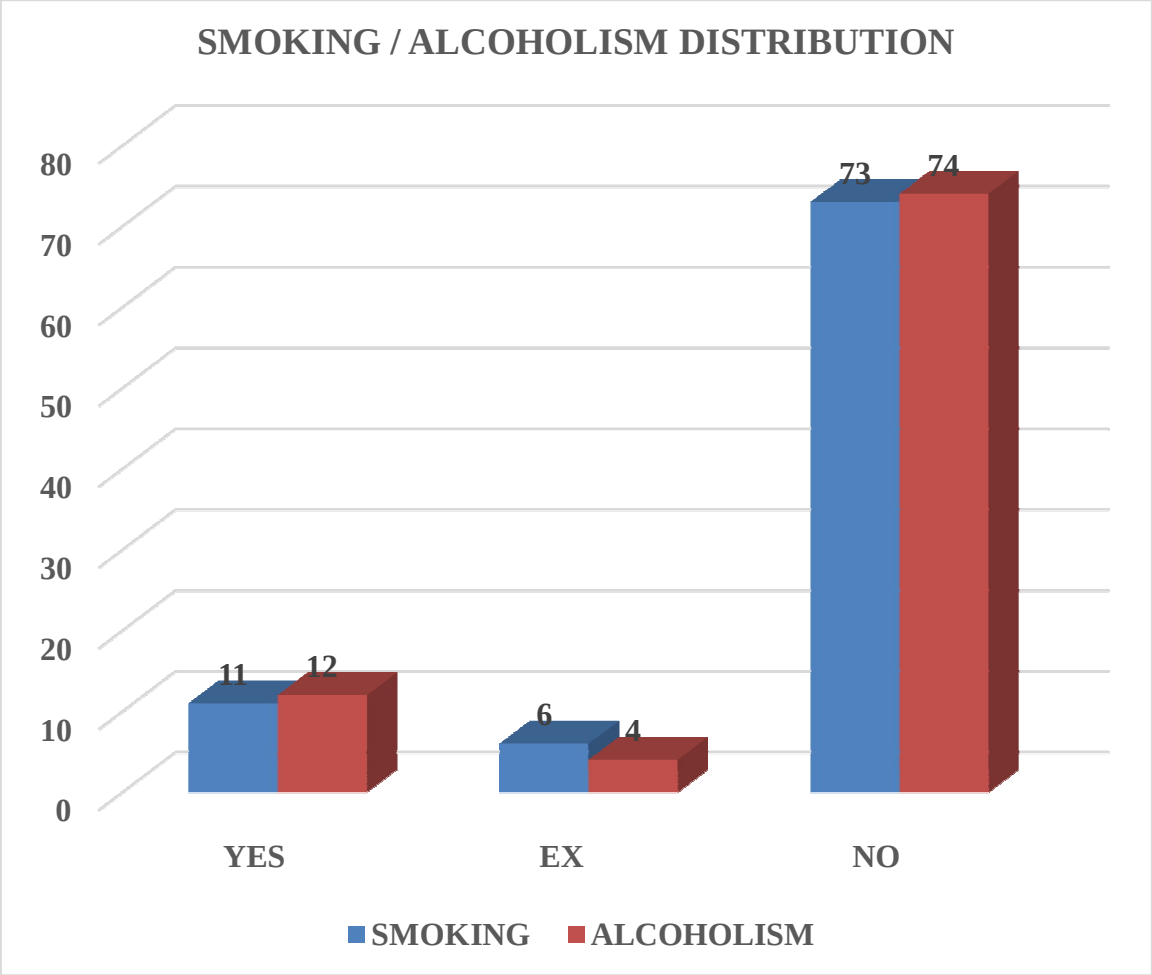

Chart 3: Showing Smoking & Alcoholism distribution

Among the 90 patients, 11 patients were smoker,6 patients were exsmoker and 12 of them were alcoholic and 4 of them were exalcoholic

SPUTUM GRAM STAINING

Table 6 : Showing the sputum gram staining results

Normal Throat commensal	Gram positive cocci	%	Gram negative bacilli	%	Mixed- both gram +ve and gram -ve	%
29	16	19%	42	47%	3	3.3%

Showing among the 90 patients, the sputum gram staining reported as, the 29 patients sputum samples contained the normal throat commensals and 19% were contained gram positive cocci and 47% were contained gram negative bacilli and 3.3% samples were contained both gram positive cocci and gram negative bacilli.

AETIOLOGICAL DISTRIBUTION

Table 7 : Showing Aetiological distribution

Aetiology	**Frequency**	**Percent (%)**
Idiopathic	38	42.2
Post infectious	19	21.1
Post Pulmonary TB	9	10.0
Congenital(undiagnosed)	7	7.8
MCTD	4	4.4
Rheumatoid arthritis	3	3.3
Kartagener syndrome	2	2.2
Sjogren syndrome	2	2.2
Young's syndrome	1	1.1
Cong.Hypogamaglobulinemia	1	1.1
CVID	1	1.1
Post aspiration	2	2.2
? Occupational	1	1.1
Total	90	100.0

[CVID-common variable immuno deficiency, MCTD-mixed connective tissue disease.]

Among 90 patients in this study population, regarding aetiology of Bronchiectasis Idiopathic bronchiectais were 42%, Post infectious bronchiectasis were 21% , Congenital type in which undiagnosed aetiology were 7.8% and secondary to connective tissue disease and kartagener syndrome, Common variable immuno deficiency 3% respectively .

BRONCHIECTASIS- RADIOLOGICAL TYPES

Table 8 : Showing the radiological types of Bronchiectasis

Bronchiectasis type	Frequency	Percent (%)
Cylindrical	48	53.3
Cystic	32	35.6
Traction bronchiectasis	3	3.3
Varicose	4	4.4
Cystic & Cylindrical	2	2.2
Traction& Cylindrical	1	1.1
Total	90	100.0

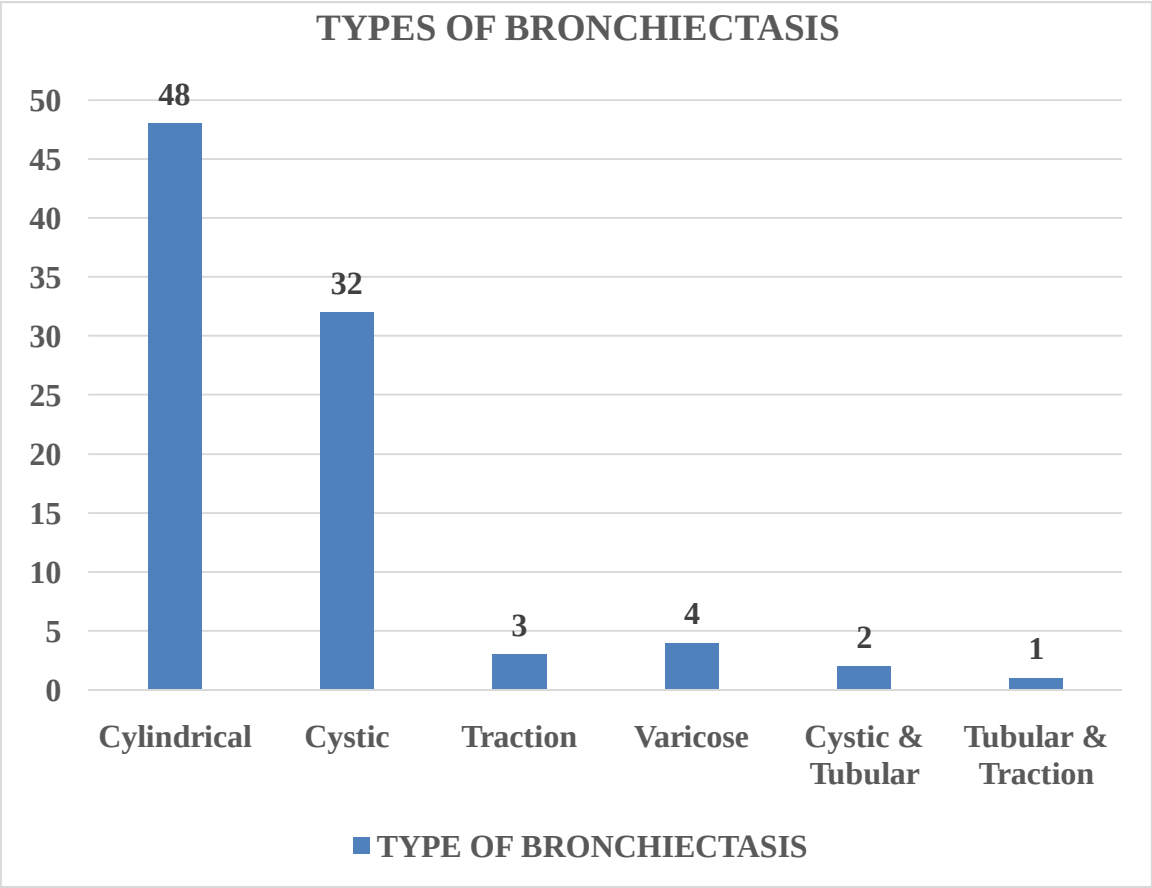

Chart 4: Showing radiological types of Bronchiectasis

Among the 90 patients in this study Cylindrical types were 53% , Cystic types were 35%, Varicose types were 4.4% , Traction bronchiectasis were 3% and 3% were mixed types i.e. Cystic plus cylindrical and Traction plus cylindrical.

PULMONARY FUNCTION FEV1% DISTRIBUTION

Table 9 : Showing pulmonary function FEV1 % distribution:

FEV1 in Spirometry	**Frequency**	**Percent %**
>80 %	13	14.4%
70-79%	10	11.1%
50-69%	52	58%
<50 %	15	16.5%
Total	90	100.0

Table:10 : Showing Spirometry pattern distribution

Spirometry pattern	**Frequency**	**Percent %**
Normal	13	14.1%
Obstructive	58	64%
Restrictive	14	15%
Mixed	6	6.6%

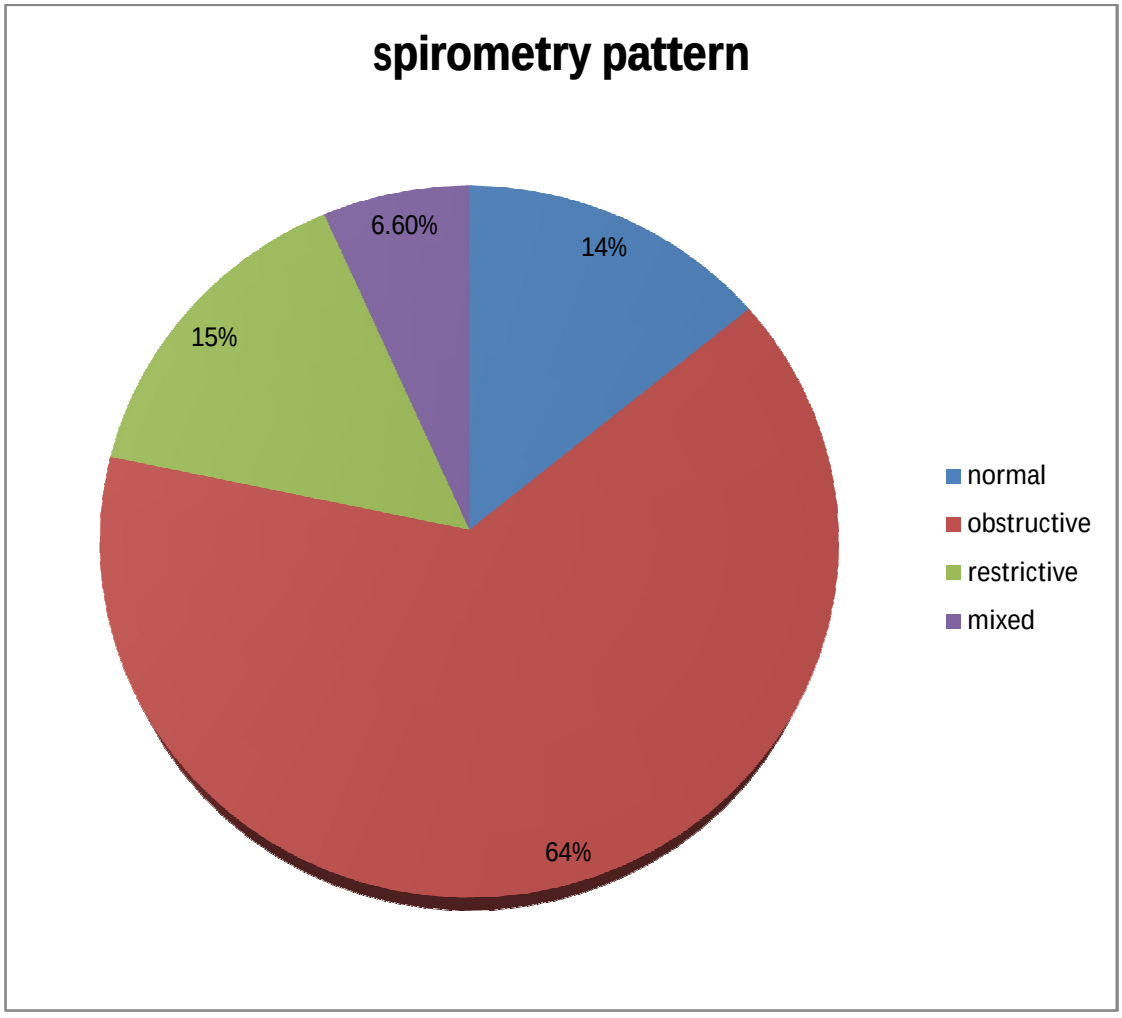

Chart:5 – Spirometry pattern distribution showing Normal spirometry in 14% of patients, Obstructive pattern observed in 64% of patients, Restrictive pattern observed in 15% of patients and mixed pattern was observed in 7% of patients

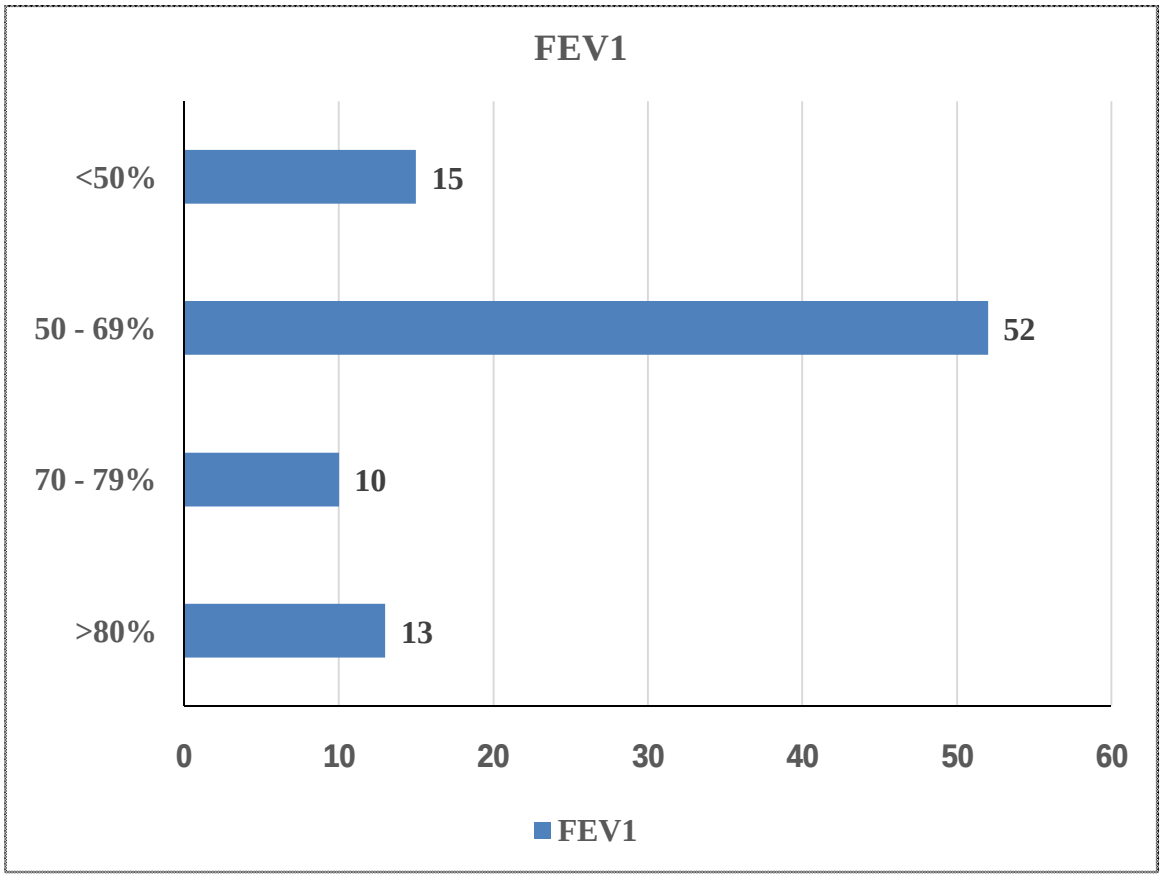

Chart 6: Showing pulmonary function FEV1 distribution

Out of 90 patients in this study 14% of patients had FEV1 > 80%, 11 % of patients had FEV1 80-79%, 52% of patients had FEV1 50-69% and 15% of patients had FEV1 <50%; Normal spirometry in 14% of patients, Obstructive pattern observed in 64% of patients, Restrictive pattern observed in 15% of patients and mixed pattern was observed in 7% of patients

HRCT SCORE DISTRIBUTION

Table 11 : Showing distribution of HRCT score.

HRCT Score	Frequency	Percent (%)
< 33%	60	66.7
33-50%	22	24.4
> 50%	8	8.9
Total	90	100.0

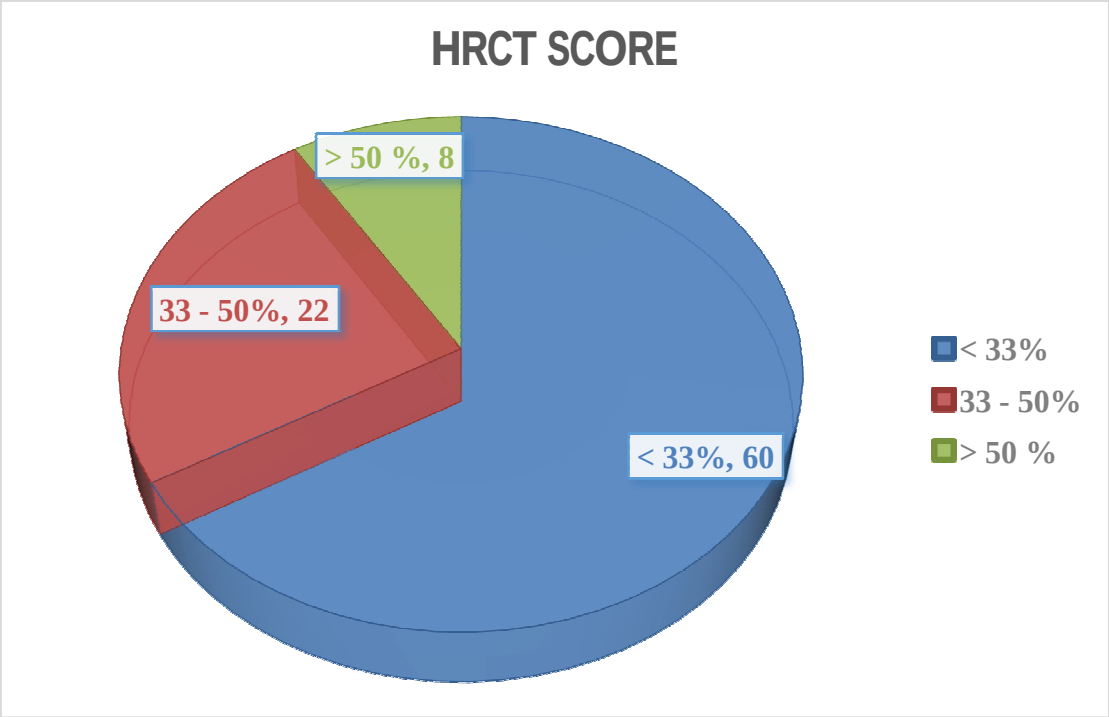

Chart 7: Showing HRCT Score Distribution

Among the 90 patients in this study, 66 % patients were presented with HRCT score <33%, 24% patients were presented with 34-50% and 8.9% patients were presented with above 50%.

SPUTUM MICROBIOLOGY DISTRIBUTION

Table 12 : Showing the micro organisms distribution in Sputum culture

Micro organism grown[>10x^{5}cfu]	Frequency	Percent (%)
No Growth	29	32.2
Hemophilus influenza	13	14.4
Pseudomonas aeruginosa	11	12.2
Streptococcus pneumoniae	9	10.0
Klebsiella pneumoniae	6	6.7
Staphylococcus aureus	5	5.6
Morexella catarrhalis	4	4.4
Acinetobacter species	3	3.3
Klebsiella oxytacea	2	2.2
Proteus mirabilis	1	1.1
Escherichia coli	1	1.1
Enterococcus	3	3.3
Streptococcus viridans	1	1.1
Coagulase negative staphylococcus aureus	2	2.2
Total	90	100.0

Out of 90 patients in this study the micro organisms isolated in sputum culture were, Potentially pathogenic micro organisms [PPM] 61%, Non potentially pathogenic micro organisms[Non PPM] were 6.6% and No growth in 32% of patients.

InPPM(potentially pathogenic micro organisms) the following micro organisms were isolated

1.Hemophilus influenza were 14.4% 2. Pseudomonas aeruginosa 12%, 3.Streptococcus pneumoniae 10%, 4.Klebsiella pneumoniae 6.7%, 5.Staphylococcus aureus 5.6% , 6.Morexella catarrhalis 4.4%, 6.Acinetobacter species 3%, 7.Klebsiella oxytacea 2%, 8.Proteus mirabilis 1% and Escherichia coli 1%.

The Non PPM isolated were 1.Enterococcus 3.3% , 2.Coagulase negative staphylococcus 2.2% 3. Streptococcus viridans 1%.

BRONCHO ALVEOLAR LAVAGE FLUID CULTURE

DISTRIBUTION

Table 13 : Showing the micro organisms distribution in Broncho alveolar lavage fluid.[$10x^3$ cfu]

Micro organism grown	Frequency	Percent(%)
No Growth	21	23.3
Hemophilus influenza	15	16.7
Pseudomonas aeruginosa	14	15.6
Streptococcus pneumoniae	9	10.0
Klebsiella pneumoniae	4	4.4
Staphylococcus aureus	2	2.2
Morexella catarrhalis	4	4.4
Acinetobacter species	2	2.2
Klebsiella oxytacea	3	3.3
Proteus mirabilis	1	1.1
Escherichia coli	1	1.1
Coagulase negative staphylococcus aureus	2	2.2
Enterococcus	2	2.2
Streptococcus viridans	2	2.2
Hemophilus parainfluenza+MRSA	1	1.1
MRSA+Acinetobacter species	2	2.2
H.influenza+Pseudomonas aeruginosa	1	1.1
Staph.aureus+Pseudomonas aeruginosa	2	2.2
CONS+Pseudomonas aeuruginosa	2	2.2
Total	90	100.0

[MRSA-methicillin resistant staphylococcus aureus]

Out of 90 patients in this study the micro organisms isolated in broncho alveolar lavage fluid culture were, potentially pathogenic micro organisms [PPM] 70%, Non potentially pathogenic micro organisms [Non PPM] were 6.6% and No growth in 23%.

InPPM the following micro organisms were isolated

1.Hemophilus influenza were 16.7% 2. Pseudomonas aeruginosa 15.6%, 3.Streptococcus pneumoniae 10%, 4.Klebsiella pneumoniae 4.4%, 5.Staphylococcus aureus 2.2% , 6.Morexella catarrhalis 4.4%, 6.Acinetobacter species 2%, 7. Klebsiella oxytacea 3%, 8.Proteus mirabilis 1% and Escherichia coli 1%.

The Non PPM isolated were 1.Enterococcus 2.2%, 2.Coagulase negative staphylococcus 2.2% 3. Streptococcus viridans 2.2%.In 9% patients, two different pathogenic bacteria were isolated i.e. Pseudomonas with acinetobacter and Hemophilus influenza and Coagulase negative staphylococcus aureus.

TOTAL CELL COUNT in BRONCHO ALVEOLAR LAVAGE FLUID

Table 14 : Showing the Total cell counts in broncho alveolar lavage fluid

Micro organisms grown

Micro organisms grown	**Total cell count in range $x10^3$**	**Total cell count in median $x10^3$**
PPM Positive = 63	90 to 210 cells$x10^3$/ml	120 cells $x10^3$/ml
Non PPM positive = 06	50 to 220 cells$x10^3$/ml	90 cells $x10^3$/ml
No growth = 21	40 to 180 cells $x10^3$/ml	50 cells $x10^3$/ml

DESCRIPTION OF TOTAL COUNTS in BAL FLUID

Table 15 : Showing the analysis of Total cell counts in broncho alveolar lavage fluid.

Growth pattern	No. of patients	Mean cell count[x10^3]	Standard deviation
PPM	63	154.92	41.246
Non PPM	6	111.67	54.924
No growth	21	55.38	15.167
Total	90	128.81	56.400

Difference between groups by ANOVA, p=0.0001

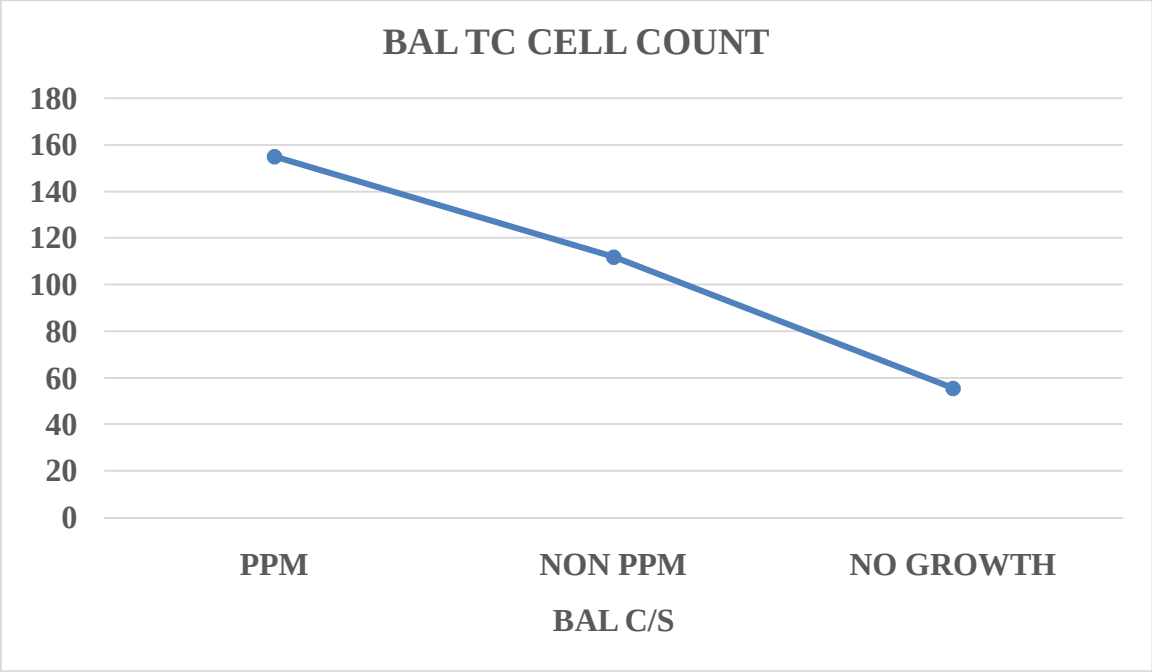

Chart 8: Showing the Total cell count in BAL Fluid

Out of 90 patients in this study, the Total cell counts calculated in broncho alveolar lavage fluid were listed as below.

In PPM patients: 90-220x10^3 cells in range and 120 x10^3 cells in median;

In Non PPM patients were 50-220x10^3 cells in range and the median cell counts were 90x10^3 cells and in no growth patients were 40-180x10^3 cells and 50x10^3 cells in median

DIFFERENTIAL CELL COUNTS in BAL FLUID

Table 16 : Showing differential cell counts in broncho alveolar lavage fluid

Predominant cells	No of patients	Percent (%)
Neutrophils >50%	44	49%
Lymphocytes>50%	40	44%
Mixed[Both cells in equal]	06	07%

Among the 90 patients in this study population the differential cell counts calculated in broncho alveolar lavage fluid were 1.Neutrophils >50% of total count in 44 patients , Lymphocytes >50% of total counts in 40 patients and in 6 patients both Neutrophils and Lymphocytes were in equal proportion.

DESCRIPTION OF DIFFERENTIAL CELL COUNTS

Table 17 : Showing the differential cell counts in broncho alveolar lavage fluid.

Cell pattern	Growth type	No.of patients	Mean	Std.deviation
Neutrophils > 50%	PPM	63	46.59	16.530
	Non PPM	6	49.17	10.206
	No growth	21	44.05	15.217
	Total	90	46.17	15.812
Lymphocytes > 50%	PPM	63	44.52	13.581
	Non PPM	6	45.00	8.367
	No growth	21	47.62	13.474
	Total	90	45.28	13.226

Difference between groups (Neutrophils) by ANOVA, p=0.731

Difference between groups (Lymphocytes) by ANOVA, p=0.654

Among the 90 patients in this study population the differential cell counts calculated in broncho alveolar lavage fluid were 1.Neutrophils >50% of total count in 44 patients , Lymphocytes >50% of total counts in 40 patients and in 6 patients both Neutrophils and Lymphocytes were in equal proportion.

INTERLEUKIN-8 DESCRIPTION in BAL FLUID

Table 18: Showing the interleukin 8 levels in broncho alveolar lavage fluid.

Growth type	No.of patients	IL-8 in Range. pg/ml	Mean	Standard deviation
PPM	63	220 -835	556.95	133.604
Non PPM	6	220-770	651.67	222.478
No growth	21	81-320	155.81	83.816
Total	90	81-835	463.67	214.394
Control	6	2-7	5	

Difference between groups by ANOVA, p=0.0001

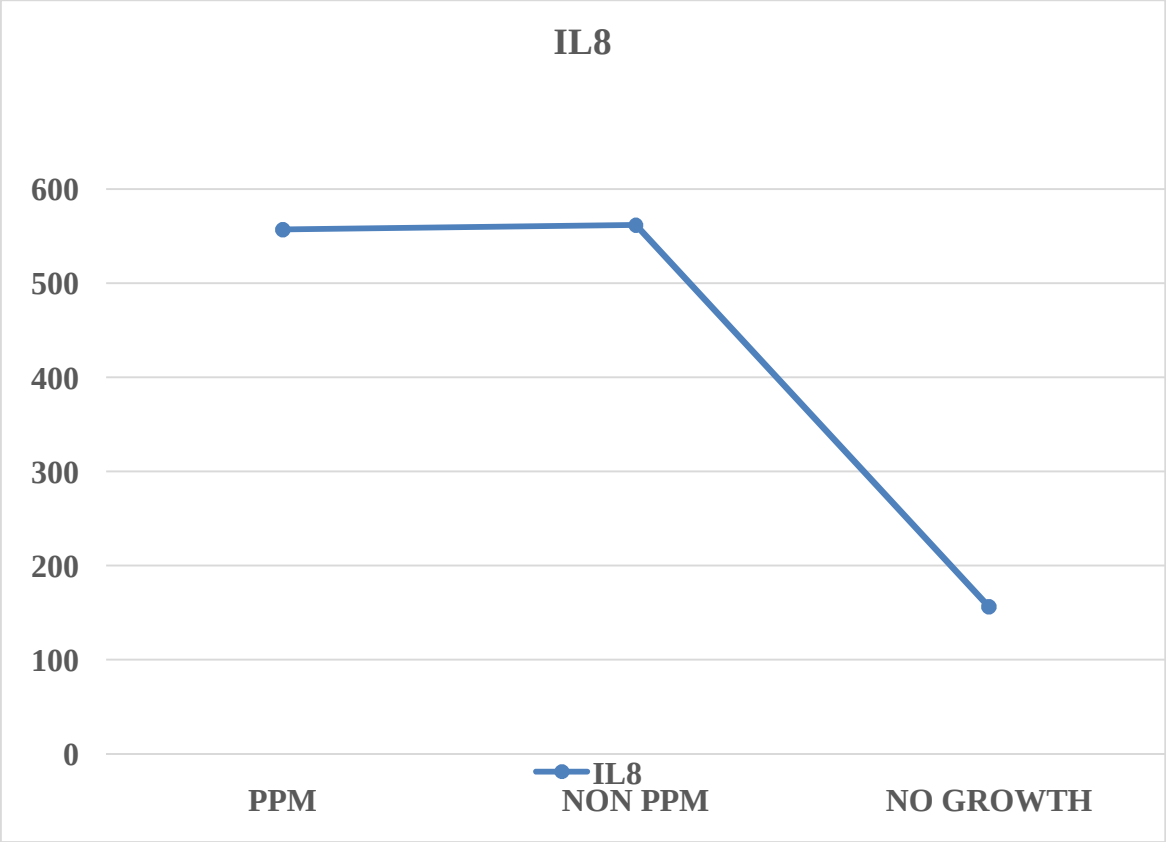

Chart 9: Showing Interleukin – 8 descriptions in BAL fluid

Out of 90 patients in this study the interleukin 8 estimated were 1.in PPM patients 220-835pg/ml in range and 556pg/ml in mean: in Non PPM patients were 220-770pg/ml and 561pg/ml in mean and in No growth patients were 81-320pg/ml and 155pg/ml in mean.

COMPARING BAL GROWTH WITH AGE DISTRIBUTION

Table 19 : Showing the Broncho alveolar lavage with age distribution comparison

Age in years	PPM		Non PPM		No Growth		Total
	N	% in Age group	N	% in Age group	N	% in Age group	
16-25	5	50.0%	2	20.0%	3	30.0%	10/ 100%
26-35	11	64.7%	1	5.9%	5	29.4%	17/100%
36-45	22	75.9%	0	0.0%	7	24.1%	29/100%
46-55	12	80.0%	1	6.7%	2	13.3%	15/100%
56-65	12	75.0%	1	6.3%	3	18.8%	16/100%
66-75	1	33.3%	1	33.3%	1	33.3%	3 /100%
Total=90	63	70.0%	6	6.7%	21	23.3%	90/100%

Fischer exact test , p value =0.026

In this comparison of broncho alveolar lavage fluid culture with age distribution , the micro organisms growth were more in age group 36-45 years followed by in age groups 46-55 years.

COMPARING BAL CULTURE WITH SEX DISTRIBUTION

Table 20 : Showing the comparison of broncho alveolar lavage fluid culture with sex distribution

Sex	PPM		Non PPM		No Growth		Total
	N	% in age group	N	% in age group	N	% in age group	
M	26	68.4%	3	7.9%	9	23.7%	38 / 100%
F	37	71.2%	3	5.8%	12	23.1%	52 /100%
T	63	70.0%	6	6.7%	21	23.3%	90 /100%

Fischer exact test , p value =0.098

By comparing the broncho alveolar fluid culture in relation with sex distribution; the micro organisms growth were observed in females 77% and males in 76%

COMPARING BAL CULTURE WITH SMOKING HISTORY

Table 21 : Showing the comparison of broncho alveolar lavage fluid growth with smoking history

Smoking History	PPM		Non PPM		No Growth		Total
	N	%	N	%	N	%	
YES	8	72.7%	1	9.1%	2	18.2%	11/100%
Exsmoker	4	66.7%	1	16.7%	1	16.7%	6 /100%
NO	51	69.9%	4	5.5%	18	24.7%	73/100%
Total	63	70.0%	6	6.7%	21	23.3%	90/100%

Fischer exact test, p value =0.042

In comparison of broncho alveolar lavage fluid culture with smoking history, in 72% of smokers PPM growths were observed and in 25% of non smokers no growth was observed.

COMPARING BAL CULTURE WITH ALCOHOLISM

Table 22 : Showing the comparison of broncho alveolar lavage fluid with alcoholism history.

Alcoholism History	PPM		Non PPM		No Growth		Total
	N	%	N	%	N	%	
YES	8	66.7%	1	8.3%	3	25.0%	12/100%
Exalcoholic	2	50.0%	1	25.0%	1	25.0%	4 /100%
NO	53	71.6%	4	5.4%	17	23.0%	74/100%
Total	63	70.0%	6	6.7%	21	23.3%	90/100%

Fischer exact test value, p=0.029

In comparison of broncho alveolar fluid culture with alcoholism history; in 87 % of patients with alcoholism PPM and Non PPM growths were observed and in 13% of non alcoholic patients, No growths were observed.

COMPARING BAL CULTURE WITH FEV1%

Table 23 : Showing the broncho alveolar culture comparison with pulmonary function FEV1%

FEV1%	PPM		Non PPM		No Growth		Total
	N	%	N	%	N	%	
> 80%	4	30.8%	1	7.7%	8	61.5%	13/100%
70-80%	5	50.0%	2	20%	3	30%	10/100%
50-70%	40	76.9%	3	5.8%	9	17.3%	52/100%
Total	63	70.0%	6	6.7%	21	23.3%	90/100%

Fischer exact test value, p=0.001

COMPARING FEV1 WITH MICRO ORGANISM GROWTH

Table 24 : Showing statistical analysis of FEV1 with micro organisms

Growth Type	N	Mean	Std. deviation
PPM	63	58.06%	12.025%
Non PPM	6	68.17%	10.591%
No Growth	21	70.57%	11.565%
Total	90	61.66%	12.956%

Difference between groups by ANOVA, p =0.0001

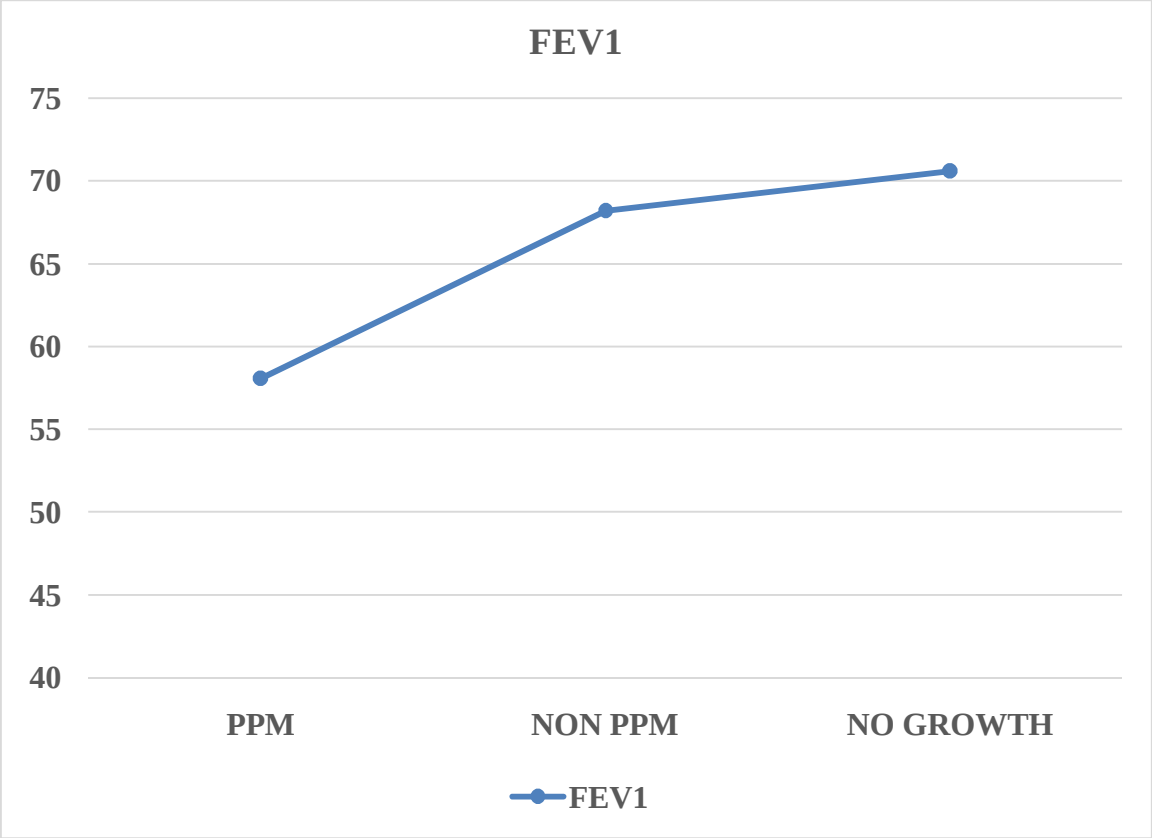

Chart 10: Showing Comparison between FEV1 with BAL Culture

In comparison of broncho alveolar lavage fluid culture, among 90 patients 14% of patients were with FEV1 more than 80% in which all were negative growths in BAL culture, 11% of patients were with FEV1 80 to 70% (in which 4% were negative growth, 7% of patients were Non PPM positive growth in BAL culture) and remaining 75% of patients were with FEV1 less than 70% (in which 70% of patients were positive for PPM growth and 5% of patients were negative growth in BAL culture).

COMPARING BAL CULTURE WITH HRCT SCORE

Table 25 : Showing the comparison of broncho alveolar lavage fluid culture with HRCT score

HRCT Score	PPM		NonPPM		No Growth		Total
	N	%	N	%	N	%	
< 50%	42	63.6%	5	7.6%	19	28.8%	66/100%
> 50%	21	87.5%	1	4.2%	2	8.3%	24/100
Total	67	70.0%	6	6.7%	21	23.3%	90/100%

Fischer exact test value, p=0.009

By this comparison of broncho alveolar lavage fluid culture with HRCT score; >50% score was associated with 87.5% PPM growths and in HRCT score <50% was associated with 29% of No growths.

COMPARING BAL CULTURE WITH BRONCHIECTASIS TYPES

Table 26 : Showing the comparison of broncho alveolar lavage fluid culture with radiological types of bronchiectasis.

Bronchiectasis Types	PPM		NonPPM		No Growth		Total
	N	%	N	%	N	%	
Cylindrical	28	58.3%	4	8.3%	16	33.3%	48/100%
Cystic	27	84.4%	1	3.1%	4	12.5%	32/100%
Traction	2	66.7%	0	0.0%	1	33.3%	3 /100%
Varicose	3	75.0%	1	25.0%	0	0.0%	2 /100%
Cystic+cylindrical	2	100.0%	0	0.0%	0	0.0%	2 /100%
Traction+cylindrical	1	100.0%	0	0.0%	0	0.0%	1 /100%
Total	63	70.0%	6	6.7%	21	23.3%	90/100%

Fischer exact test value, p= 0.006

In comparison of broncho alveolar lavage fluid culture with bronchiectasis types; Cystic bronchiectasis was associated with 84% positive growth followed by varicose bronchiectasis 75%; and Cylindrical and Traction bronchiectasis was associated with 33% of negative growth respectively.

COMPARING SPUTUM & BAL CULTURE RESULTS

Table 27: Showing the comparison of sputum and broncho alveolar lavage fluid culture.

SPTUM_C_S		BAL _C_S_CAT			Total
		PPM	**NonPPM**	**No growth**	
PPM=55	No.of pts	54	1	0	55
	Percent	98.2%	1.8%	0.0%	100.0%
Non PPM=6	No.of pts	2	4	0	6
	Percent	33.3%	66.7%	0.0%	100.0%
No Growth=29	No.of pts	7	1	21	29
	Percent	24.1%	3.4%	72.4%	100.0%
Total	No.of pts	63	6	21	90
	Percent	70.0%	6.7%	23.3%	100.0%

Observed agreement: 0.877778

Expected agreement: 0.507407

k coeff : 75.18797

In detailed analysis of sputum and broncho alveolar lavage fluid culture ; the negative growth was observed in 29 sputum samples and 21 broncho alveolar lavage fluid samples, and 21 samples (both sputum and broncho alveolar lavage fluid) were negative growth pattern; In regarding positive growth, 61sputum samples were positive growth and 69 broncho alveolar lavage samples were positive growth and the 55 samples of both sputum and broncho alveolar fluid sample were same micro organisms pattern having concordant value of 75%.

SUMMARY

Among the 90 patients in this study population 29% of patients were in age group between 36-45 years, 16% were in between 56-65 years, 15% were in between 46-55 years and 10% were between 16-25 years: Females were 58% and males were 42%:

Regarding personal habits 12 % of patients were smokers, 7% were past smokers and 81% of patients were never smokers: 13% of patients were alcohol consumers and 5% were past alcohol consumers and 82% were not alcohol consumers.

Regarding aetiology of bronchiectasis ,42% of bronchiectasis were idiopathic , 21% were due to post infectious , 10% were due to post pulmonary tuberculosis , undiagnosed cause of congenital types were 8% and mixed connective tissue disease 11%, and diagnosed congenital causes [kartagener syndrome, young's syndrome, and immuno deficiency states]5%.

Regarding radiological types of bronchiectasis, Cylindrical types were 53%, Cystic 35%, and Varicose types 4%: and depending upon the disease severity in HRCT chest, HRCT score was calculated. Among 90 patients, 67% of patients were <33%, 24% were 34-50% and 9% of patients were >50%.

Regarding the pulmonary function measured by FEV1% in Spirometry; normal spirometry measured in 14% of patients, obstructive pattern measured in 64% of patients [with FEV1- 70-79%= 11% of patients, with FEV1 50-69%= 49.6% of patients and with FEV1 <50% =4.4% of patients]. Restrictive pattern was observed in 15% of patients [with FEV1<70 in 3 patients and with FEV1<50% in 11 patients] and Mixed pattern [both obstruction+restriction] was observed 7% of patients.

Regarding the micro organisms isolated in sputum and broncho alveolar lavage culture, Positive sputum culture growths were 68%, negative culture growths were 32%: In Broncho alveolar lavage fluid culture, positive culture growths were 77% and negative culture growths were 23%. The predominant organisms isolated were 1.H.influenza 2.Pseudomonas species 3.Streptococci pneumoniae 4.Staphylococcus aureus 5.Morexella catarrhalis 6. Klebsiella species etc.

Regarding inflammation of airways in bronchiectasis, the total counts were raised in potentially pathogenic micro organisms grown patients followed by nonpotentially pathogenic micro organisms grown and lesser amounts in patients with no growth. The differential cell count Neutophils more than 50% of total count was observed in 44 patients ,Lymphocytes more than 50% were observed in 40 patients and both Neutrophils and Lymphocytes in equal portion in 6 patients indicating slight increase of Neutrophilic pattern followed by Lymphocytic pattern.

The interleukin-8 measured in BAL fluid showed, the increased level [range 81-835pg/ml , from no growth to PPM growth] was observed in all the three groups i,e in PPM , NonPPM and No Growth patients with comparing 6 controls ranged from 2-7pg/ml, indicating the earlier establishment of airway inflammation and the airway inflammation was worsened by micro organisms colonisation.

Regarding the risk factor for micro organisms colonisation, the increased risk was observed in patients presented with

1. Long duration of symptoms > 5 years
2. with habits of smoking and alcoholism
3. Cystic bronchiectasis and varicose bronchiectasis

DISCUSSION

DEMOGRAPHICAL FACTORS

Based on the guidance studies, David A Bergin et al study, J Angrill et al study, C Agusti et al study, U.S study of investigation into aetiology of bronchiectasis; in this study closely matched with the above studies in demographical description[11,12,20] , except the median age in this study was 42 years against 65 years and 58 years in above studies. Females were 58%, males were 42%; smokers and alcohol consumers were 19%.

The predominant radiological type of bronchiectasis was cylindrical(53%) followed by cystic bronchiectasis(35%); and regarding the aetiology of bronchiectasis, idiopathic bronchiectasis(42%), post infectious(21%), post pulmonary tuberculosis(10%), and 2-3% of kartagener syndrome, young's syndrome, common variable immunodeficiency, congenital hypogammaglobulinemia and Rheumatoid arthritis and mixed connective tissue diseases respectively.

MICROBIOLOGY OF BRONCHIECTASIS

The micro organisms from the airways of clinically stable bronchiectasis patients were isolated by sputum culture and broncho alveolar lavage fluid culture. The BAL fluid culture (77%positive growth rate) was slightly higher than sputum positive culture (68%).

The micro organisms isolated from sputum and BAL culture were H.influenza (17%), Pseudomonas species (16%), Streptococcus pneumoniae (10%), Morexella, Klebsiella and Staph aureus were 3-4% respectively as similar to previous studies[1,3,7].

By comparing the sputum and BAL fluid culture, among the 61 (68%) positive culture in sputum samples; 56 samples were positive for PPM growth and 5 samples were positive for Non PPM growth in BAL fluid culture. Among the 29 negative cultures in sputum samples - 7 samples were positive for PPM growth and 1sample was positive for Non PPM growth and 21samples were found to be No growth in BAL fluid culture-with k co efficiency of 75%.

In J Angrill et al study and C Agusti et al study; the positive sputum culture was observed in 66% of patients, in BAL-positive culture was observed in 79% of patients; and both techniques agreed same positive growth in 30 patients /60 cases, and agreed negative growth in 18 patients /60 cases with concordant value of 75% for both techniques; this observation in this study fully matched with this studies.[8-12]

ANALYSIS OF BAL CULTURE WITH DEMOGRAPHIC DATA

J Angrill et al study and C Agusti et al study evaluated the increased colonisation in age groups > 60 years , patients with smoking and alcoholism, in cystic bronchiectasis; and observed the inverse relationship between the airway colonisation and pulmonary function i,e more colonisation leads to poor pulmonary function, and similar findings were evaluated in this study.[11,12]

By keeping the Broncho alveolar lavage fluid culture as gold standard the following analysis were done.

AGE- The increased culture positive was observed in age group 46-55 years (80%), 36-45 years (75%) and 26-35 years (65%). The people in age group between 36-55 years were presented with long duration of symptom i.e. more than 5 years and up to 30 years duration -co relating the long duration of symptoms, the micro organisms colonisation was high[11,12].

SEX: The females in this study were 52(58%) in comparison with males, the negative cultures observed in females were 12 /21 no growths, in contrast males were 9/21 no growths.

HABITS: Regarding the personal habits like smoking and alcoholism 17 patients(19%) were used to smoking and alcohol ; the No growths observed in 18(25%) patients who were never smoker and never alcohol consumers and in 75% of smokers and alcoholics PPM and Non PPM growths were observed - co relating that smoking and alcoholism increases the micro organisms colonisation[11,12]

PULMONARY FUNCTION

Among the 90 patients, normal spirometry[FEV1> 80%] was measured in 13 (14.4%) patients and in 53(59%) patients obstructive pattern (FEV1< 70%) and Restrictive pattern was observed in 14 patients (15%). The negative growths were observed in 18/21 of patients who had FEV1 >70% [14% normal spirometry +5.5% mild obstructive pattern]. Among the 69(77%) positive growths observed patients, patients with FEV1 <70% were 67(74%) with moderate obstructive pattern 53(58%) and severe restrictive pattern 14(15%) -co relating that micro organisms colonisation in the airways of bronchiectasis lead to more tissue damage and poor pulmonary function[11,12,29,30]

RADIOLOGICAL TYPES

Among the 90 study patients the predominant radiological type of bronchiectasis was Cylindrical 48 patients (53%) Cystic 32 patients (36%) and Varicose type 4cases (4.4%). Among 32 cystic types 28 cystic bronchiectasis were positive for PPM and Non PPM growth. Among Varicose type the 4 patients were positive for micro organisms growth. In the Cylindrical type of bronchiectasis 16 patients against 48 cases were found to be No growths in BAL culture - co relating that micro organisms colonisation were more in Cystic and Varicose type[39,40] .

RELATION BETWEEN THE COLONISATION AND INFLMMATION:

J Angrill et al study and C Agusti et al study, David A Bergin et al study evaluated the BAL fluid inflammatory cells and the interleukins level in proportion with airway bacterial colonisation. The total counts measured were $1086x10^3$ cells(maximum) ,and $100x10^3$-$1086x10^3$ cells [in range] with Neutrophilic predominance in 66% of patients and Interleukin-8 level measured were 120-5520 pg/ml [in range] and 195pg/ml in median were measured in PPM positive group; where as in 9 control group , total counts measured were $110x10^3$ cells with Neutrophilic pattern in 1% and the interleukin-8 measured were 0-32pg/ml (in range) - indicating the increased level of inflammation in direct proportion with microbial colonisation[11,12,23-26,32-36]

Regarding the total count measured in BAL fluid; there was increased amount of inflammatory cells in the airways of bronchiectasis in all the three groups i.e. in PPM, Non PPM and No growth patients. The mean cell counts observed were $120x10^3$ cells for PPM patients, $90x10^3$ cells in Non PPM patients and $50x10^3$ cells in No growth patients. The maximum cell counts measured were $220x10^3$ cells in PPM growth patients. The least cell counts measured were $40x10^3$ cells in No growth patients.

Regarding the differential cell count: Out of 90 patients in 44 patients (49%) the Neutrophils more than 50% of total cell count were measured, and in 40 patients (44%) the Lymphocytes more than 50% of total cell counts were measured and in 6 patients (7%) equal proportions of neutrophils and lymphocytes were measured.

In bronchiectasis airways more cellularity was measured and there was slight Neutrophilic pattern followed by lymphocytic pattern. The more number of neutrophils leads to liberation of elastase, proteinase, tumour necrosis factor alpha and cytokines predominantly interleukins-8, and this interleukin-8 causes neutrophilic chemotaxis in airways and the net result of more airway inflammation and more tissue damage.[32-36.]

INTERLEUKIN-8:

In this study interleukin-8 was measured from 6 normal people as control group and the values in range 2 - 7 pg/dl and 5pg/dl in mean. The 90 patients in this study were divided into three groups i,e PPM, Non PPM group and No growth group. The mean values measured were 556.95pg/dl in PPM, 561.67pg/dl in Non PPM, and 155.81pg/dl in No growth patients respectively. The highest level of IL-8 835pg/dl was measured in PPM group and the lowest value of IL-8, 81pg/dl was measured in patients with No growth sample. The total range of IL-8 among the 90 patients was 81-835pg/dl.

With this, the interleukin 8 was grossly raised in the airways of bronchiectasis patients irrespective of the micro organisms colonisation, but the peak of raise of IL-8 was higher in patients with micro organisms colonisation especially in potentially pathogenic micro organisms colonisation -indicating the earlier establishment of airway inflammation and the airway inflammation was worsened by the micro organisms colonisation especially the potentially pathogenic micro organisms.[11,12,23-26]

CONCLUSION

With complete analysis of entire study results, the following conclusions were arrived.

1. Increased incidence of bronchiectasis in females (58%).

2. Cylindrical bronchietasis was the commonest type followed by Cystic bronchiectasis

3. Regarding aetiology of Bronchiectasis, 42% of bronchiectasis were Idiopathic followed by post infectious 21%

4. The pulmonary function FEV1< 70% was associated with micro organisms colonisation of bronchiectatic airways.

5. With concordant value of 75% for sputum culture and broncho alveolar lavage fluid culture, the Sputum culture is the non invasive, alternative technique for broncho alveolar lavage fluid culture.

6. As the airway inflammation was worsened by micro organisms colonisation, by elimination of these micro organisms, the airway inflammation and tissue damage can be reduced and

7. Following factors were identified as the Risk factors for micro organism's colonisation of airways

a) Patients with long duration of symptoms

b) Associated personal habits like smoking and alcoholism

c) Cystic bronchiectasis and varicose bronchiectasis

STUDY LIMITATION

In comparing the previous studies, in this study the protective specimen brush technique was not done

In Neutrophilic chemotaxis the Elastase activity is more, and various cytokines are liberated in the airway of bronchiectasis, this study measured the interleukins 8 only and not measured the other cytokines [TNF alpha, Myeloperoxidase,and Leukotrienes] and sputum elastase activity, due to cost .

With comparing the previous studies the accuracy of data collected is questionable.

Selection bias could have happened as patients represented in this study, all belong to a single tertiary care centre.

Since the sample size is small, this may not represent the exact nature of disease in general population

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BRONCHIAL COLONISATION AND INFLAMMATION IN PATIENTS WITH CLINICALLY STABLE BRONCHIECTASIS

Name : IP No :

Age : Sex :

Address : Mobile No :

COMPLAINTS :

☐ Hemoptysis ☐ Expectoration ☐ Fever

☐ Breathlessness ☐ Others

EXPECTORATION : ☐ White Mucoid ☐ Purulent

☐ Foul Smelling ☐ Others

Duration of Symptoms :

PAST HISTORY :

☐ Joint Pain (S/o.Rneumatoid)

☐ Dry Eyes, Dry Mouth (S/o.Sjogren

☐ Fever

☐ Exanthematous Fever

☐ Aspiration

☐ Prior ATT

☐ Recurrent cough and cold since child hood

Personal History: ☐ Smoker ☐ Alcoholism

☐ Tobaco Chewer ☐ Beetel nut chewer

Family History :- ☐ Cansanguinous marriage

☐ Infertility

☐ Other family member has similar problem

Occupational History :-

ON EXAMINATION

Nutritional Status ☐ Normal ☐ Anemia

☐ Emaciated ☐ Obese

Head to Foot :- ☐ Clubbing ☐ Comfortable

☐ Tachypneic ☐ Dyspneic

☐ Pedal Edema ☐ Ascites

Vitals :- RR :

HR :

BP :

SPO_2

Temp

Respiratory System Examination:-

Investigations:-

CBC : Total Count :

Differential Count : P : L : E :

M : B :

Hb% :

ESR : ½ hr : 1 hr.

Platelet :

RBC :

Spirometry

FVC

:

FEV1

B1 – Sugar :

B1 – Urea :

FVC/FEV1

Sr. Creatinine :

PEFR

Total Bilirubin :

FEF 25to 75

Total : - Protein :

Allumin :

Sputum – AFB :

Sputum Culture :

ECG :

CXR :

Echocardiogram :

HRCT Chest :- {Score -o-no bronchiectasis.1-bronchiectasis involving one segment/lobe .2-bronchiectasis involving >one segment/lobe. 3-cystic bronchiectasis} Max Score-18

1. No. of Segments involved
2. Pattern of branchiectasis
3. **Total score**

Rheumatologist Opinion :-

Bronchiectasis Due to :- (Based on clinico radiological evidence)

FOB Findings :-

Broncho alveolar Lavage Fluid:

1. Cell Count :
2. Cytology :
3. Culture :
4. IL-8 :

INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE, CHENNAI-3

EC Reg No.ECR/270/Inst./TN/2013
Telephone No : 044 25305301
Fax : 044 25363970

CERTIFICATE OF APPROVAL

To
Dr. R. Nedunchezhian,
PG in Thoracic Medicine,
Institute of Thoracic Medicine,
Madras Medical College, Chennai-3.

Dear Dr. R. Nedunchezhian,

The Institutional Ethics Committee of Madras Medical College, reviewed and discussed your application for approval of the proposal entitled **"Bronchial Colonisation and Inflammation in patients with clinically stable Bronchiectasis – Microbiological Pattern and Risk Factors "** No.08032014

The following members of Ethics Committee were present in the meeting held on 11.03.2014 conducted at Madras Medical College, Chennai-3.

1.	Dr. C. Rajendran, M.D.	-- Chairperson
2.	Prof. Kalaiselvi, MD Vice-Principal, MMC, Ch-3	-- Member Secretary
3.	Prof. Nandhini, M.D. Inst. of Pharmacology, MMC, Ch-3.	-- Member
4.	Prof. Bhavani Shankar, M.S. Prof & HOD of General Surgery, MMC, Ch-3.	-- Member
5.	Prof. V. Padmavathi, M.D. I/c Directory of Pathology, MMC, Ch-3.	-- Member
6.	Thiru. S. Govindasamy, BABL	-- Lawyer
7.	Tmt. Arnold Saulina, MA MSW	-- Social Scientist

We approve the proposal to be conducted in its presented form.

Sd/Chairman & Other Members

The Institutional Ethics Committee expects to be informed about the progress of the study, and SAE occurring in the course of the study, any changes in the protocol and patients information / informed consent and asks to be provided a copy of the final report.

20/3/14

VICE PRINCIPAL
MADRAS MEDICAL COLLEGE

Member Secretary, Ethics Committee

INFORMATION SHEET

PLACE OF STUDY - RAJIV GANDHI GOVERNMENT GENERAL HOSPITAL, CHENNAI – 600 003

Title

"BRONCHIAL COLONISATION AND INFLAMMATION IN PATIENTS WITH CLINICALLY STABLE BRONCHIECTASIS-MICROBIOLOGICAL PATTERN AND RISK FACTORS"

Participants Name : **Age :**
Sex : **IP NO :**

Investigator Name: Dr.R.Nedunchezhian

- We are conducting a study on Branchoalveolar Lavage Fluid among patients with Bronchiectasis attending Government General Hospital, Chennai.
- The purpose of this study To study the Bronchial inflammation and Colonisation in patients with clinically stable Bronchictasis.

Patients admitted with clinical findings and HRCT CHEST evidence of Bronchiectasis are evaluated with detailed History and thorough Clinical examination and

On day 1.

1) Base line investigation (CBC, RFT, LFT, BT, CT, ICTC, ECG)
2) Microbiology -Sputum for bacterial culture,AFB,Gramstaining
3) Blood ESR, for assessing systemic level of inflammation will be done.

   On day 2.
4) Chest X Ray -
5) Pulmonary function test -Soirometry will be done
6) Number of segment involved in HRCT CHEST to be estimated

   On day 3.
7) Finally Broncho alveolar lavage to be done with consent of the patients.Upper airway anesthesia will be achieved by nebulisation of 5%lignocaine 8ml for 15 minutes.The bronchoscope [pentax-18v.pentax medical india pvt ltd] will be passed transnasally and BAL will be done from the lobes more involved by bronchiectasis using 150ml 0.9%Normal saline in three 50ml aliquots. The collected fluid will be sent to.

   a. Microbiology lab – for bacterial culture.
   b. Pathology lab – for cell count[total&differential] and cytology of the fluid
   c. Biochemistry lab for-IL-8 estimation

- The privacy of the patients in the research will be maintained throughout the study. In the event of any publication or presentation resulting from the research, no personally identifiable information will be shared.

- Taking part in this study is voluntary. You are free to decide whether to participate in this study or to withdraw at any time; your decision will not result in any loss of benefit to which you are otherwise entitled.
- The results of the special study may be intimated to you at the end of the study period or during the study if anything is found abnormal which may aid in the management or treatment.

Signature of Participant/ Parent / Guardian

Signature of Investigator

Place :

Date :

ஆராய்ச்சி ஒப்புதல் கடிதம்

ஆராய்ச்சி தலைப்பு :

மூச்சுக்குழாய் தளர்ச்சி அடைந்த நோயாளிகளின் மூச்சுக்குழாய் தளர்ச்சியினால் நோய்க் கிரிமிகளின் ஆதிக்கம் மற்றும் மூச்சுக் குழாய் பாதிக்கப்படுவதை கண்டறிதல்.

பெயர் தேதி

வயது உள் நோயாளி எண்

பால் ஆராய்ச்சி சேர்க்கை எண்

இந்த ஆராய்ச்சியின் விவரங்களும் அதன் நோக்கங்களும் முழுமையாக எனக்கு தெளிவாக விளக்கப்பட்டது.

எனக்கு விளக்கப்பட்ட விஷ்யங்களை நான் புரிந்து கொண்டு நான் எனது சம்மதத்தைத் தெரிவிக்கிறேன்.

1, எனக்கு நுரையீரல் சலி பரிசோதனை செய்து கொள்ள சம்மதம்.

இந்த ஆராய்ச்சியில் பிறரின் நிர்ப்பந்தமின்றி என் சொந்த விருப்பத்தின் பேரில் தான் பங்கு பெறுகிறேன் மற்றும் நான் இந்த ஆராய்ச்சியிலிருந்து எந்நேரமும் பின்வாங்கலாம் என்பதையும் அதனால் எந்த பாதிப்பும் ஏற்படாது என்பதையும் நான் புரிந்து கொண்டேன்.

2, நான் மூச்சுக் குழாய் தளர்ச்சி பற்றிய இந்த ஆராய்ச்சியான விவரங்களைக் கொண்ட தகவல்களைப பெற்றுக் கொண்டேன்.

நான் என்னுடைய சுய நினைவுடன் மற்றும் முழு சுதந்திரத்துடன் இந்த மருத்துவ ஆராய்ச்சியில் என்னை சேர்த்துக்கொள்ள சம்மதிக்கிறேன்.

3, மூச்சுக் குழாய் உள்வாங்கி செலுத்தி சலி எடுப்பதற்கு முன் வலி தெரியாமல் இருப்பதற்கு லிக்னோகெய்ன இன்ஜெக்சன் டெஸ்ட் ஊசி போடுவதற்கும், லிக்னோகெய்ன் ஜெல் மருந்தை மூச்சில் போட்டுக் கொள்வதற்கும் சம்மதம்.

மேற்கண்ட ஊசியை போடும் போது ஏதேனும் பின் விளைவுகள் (அரிப்பு, தோல் வீக்கம், மயக்கம், தலைச்சுற்றல், வாந்தி முதலியன) ஏற்படலாம் என மருத்துவர் மூலம் தெரிந்து கொண்டேன்.

கையொப்பம்

ஆராய்ச்சி தகவல் தாள்

நுரையீரல் மருத்துவ பகுதி

சென்னை அரசு பொது மருத்துவமனை வரும் நோயாளிகளின் மூச்சுக்குழாய் தளர்ச்சி அடைதல் பற்றிய ஆராய்ச்சி நடைபெற உள்ளது.

மூச்சுக்குழாய் தளர்ச்சி எதனால் ஏற்பட்டது. மூச்சுக்குழாய் தளர்ச்சியினால் நுண்ணியிரிகளின் ஆதிக்கம் என்ன ?. அதனால் உடலுக்கு ஏற்படும் விளைவுகள் என்ன என்பதை பற்றி அறிவதற்கு மூச்சுக்குழாய் உள் வாங்கி மூலம் நோயாளிகளின் நுரையீரல் இருந்து சலி எடுக்கப்பட்டு, அது பரிசோதனைக்கு அனுப்பப்படுகிறது.

நீங்களும் இந்த ஆராய்ச்சியில் பங்கேற்க நாங்கள் விரும்புகிறோம். இந்த ஆராய்ச்சியில் உங்களுடைய நுரையீரல் சலியை எடுத்து சில சிறப்புப் பரிசோதனைக்கு உட்படுத்தி அதன் தகவல்களை ஆராய்வோம். அதனால் தங்களது நோயின் ஆய்வறிக்கையோ அல்லது சிகிச்சையோ பாதிப்புக்கு ஏற்படாது என்பதையும் தெரிவித்துக்கொள்கிறோம்.

முடிவுகளை அல்லது கருத்துக்களை வெளியிடும் போதோ அல்லது ஆராய்ச்சியின் போதோ தங்களது பெயரையோ அல்லது அடையாளங்களையோ வெளியிட மாட்டோம் என்பதையம் தெரிவித்துக்கொள்கிறோம்.

இந்த ஆராய்ச்சியில் பங்கேற்பது தங்களுடைய விருப்பத்தின் பேரில் தான் இருக்கிறது. மேலும் நீங்கள் எந்நேரமும் இந்த ஆராய்ச்சிலிருந்து பின்வாங்கலாம் என்பதையும் தெரிவித்துக்கொள்கிறோம்.

இந்த சிறப்புப் பரிசோதனைகளின் முடிவுகளை ஆராய்ச்சியின்போது அல்லது ஆராய்ச்சியின் முடிவின் போது தங்களுக்கு அறிவிப்போம் என்பதையும் தெரிவித்துக்கொள்கிறோம்.

ஆராய்ச்சியாளர் கையொப்பம் பங்கேற்பாளர் கையொப்பம்

தேதி :

S.NO	NAME	AGE	SEX	SMOKING	ALCOHOLISM	Aetiology	Duration of symptoms	PFT-FVC	FEV1	FEV1%	HRCT Score	Gram stain	*HRCT-TYPE OF BRONCHIECTASIS*	SPUTUM C/S>10*5CFU	BAL C/S>10*3	BAL TOTAL COUNT	BAL DIFF COUNT	ANTIBIOTIC RESISTENCE	IL-8 pg/dl
1	Chandru	23	m	nil	nil	congenital8 yrs	8 years	53%	41%	o.63	67%	gm - ve ba	*Cystic bronchiectasis*	K.Pneumoniae	Staph aureus,P aeuruginosa	290 x10*3cells/ml	N-50%,L-40%	Not resistant to drug	760.000
2	Vijayakumar	35	m	nil	nil	Post infect	2 yrs	65%	69%	0.78	50%	NTC	*Cystic bronchiectasis*	No growth	No Growth	103X10*3	N-60%.L-35%		153.000
3	Palraj	65	m	present	present	Post PT	15 YRS	60%	54%	1	50%	gm - ve ba	*Cystic+ Tubular bronchiectsis*	Psed.aeuruginosa	H.Influenza	190X10*3	N-65%.L-25%	S-AGS,Quinolone,Cepha	360.000
4	Theivanayagi	42	f	nil	nil	MCTD	2 yrs	65%	62%	0.76	33%	gm +ve co	*Cylindrical*	Strepto pneumoniae	Stepto pneumoniae	170X10*3	N-60%L-35%	S-Quinolon,Piperacillin	450.000
5	Savithri	35	f	nil	nil	post infect	15 yrs	60%	67%	0.76	33%	gm-ve ba	*Cystic bronchiectasis*	Psed.aeuruginosa	K.OXYTACEA	350X10*3	N-65%.L-25%	Cephalosporin resist	797.000
6	Sekar	40	m	EX SMOK	nil	Post infect	10 yrs	59%	65%	0.7	33%	gm-ve ba	*cylindrical bronchiectasis*	Psed.aeuruginosa	Psed.aeuruginosa	325X10*3	N-50%,L-40%	S-AGS,Cefaperazone	609.000
7	Stella mary	43	f	nil	nil	Idiopathic	2 yrs	69%	72%	0.76	33%	gm-ve ba	*cylindrical bronchiectasis*	Psed.aeuruginosa	Psed.aeuruginosa	310X10*3	N-45%,L-50%	S-AGS,Cefaperazone	807.000
8	Nithya kalyani	35	f	nil	nil	Post infect	5 yrs	63%	54%	0.7	33%	gm-ve ba	*cylindrical bronchiectasis*	H.influenza	H.influenza	150X10*3	N-50%,L-40%	Penicillin Resistant	757.000
9	Shyamala	39	f	nil	nil	Idiopathic	5 yrs	59%	63%	0.77	50%	gm-ve ba	*cylindrical bronchiectasis*	H.influenza	H.influenza	300X10*3	N-70%,L-25%	S-Quinolon,Piperacillin	757.000
10	Gangasalam	52	m	present	present	Karatagener	10yrs	41%	39%	0.66	67%	gm-ve ba	*Cystic bronchiectasis*	Psed.aeuruginosa	Psed.aeuruginosa	300X10*3	N-65%.L-25%	Quinolon resistant	654.000
11	Vaduvambal	50	f	nil	nil	Idiopathic	10 yrs	47%	42%	0.56	17%	gm-ve ba	*cylindrical bronchiectasis*	H.influenza	H.influenza	200X10*3	N-55%,L-40%	S-Erythro,amoxy	450.000
12	Ponnaiah	42	m	nil	present	Post infect	2 yrs	54%	51%	0.69	33%	NTC	*Cylindrica bronchiectasis*	No growth	No Growth	110X10*3	N25%.L60%	S-AGS,Cefaperazone	207.000
13	Geetha	42	f	nil	nil	Idiopathic	2 yrs	63%	67%	66%	50%	NTC	*cylindrical bronchiectasis*	No growth	No Growth	100X10*3	N-20%.L-60%		81.000
14	Paranchothi	43	f	nil	nil	Idiopathic	5 yrs	61%	54%	0.72	33%	gm+ve co	*cylindrical bronchiectasis*	Staph.aureus	Staph aureus,P aeuruginosa	210X10*3	N-70%,L-25%	S-Amoxy,Quinolon	557.000
15	Arjunan	55	m	present	present	Post infect	15 yrs	58%	52%	0.7	50%	NTC	*Cystic bronchiectasis*	No growth	Psed.aeuruginosa	110X10*3	N-50%,L-40%	S-AGS only	660.000
16	Lakshmi	45	f	nil	nil	Rheumatoid	5 yrs	71%	69%	0.73	33%	gm+ve co	*Tubular bronchiectasis*	Strepto pneumoniae	Strepo.pneumoniae	120X10*3	N-40%,L-55%	S-Erythro,amoxy	557.000
17	Kanniammal	48	f	nil	nil	Post TB	15yrs	50%	56%	68%	33%	NTC	*Cystic bronchiectasis*	No growth	H.influenza	170X10*3	N-55%,L35%	S-Erythro,amoxy	207.000
18	Vanaja	42	f	nil	nil	Idiopathic	5 yrs	71%	74%	73%	33%	gm-ve ba	*Tubular bronchiectasis*	H.influenza	H.influenza	170X10*3	N-50%,L-45%	Penicillin Resistant	757.000
19	murumayee	45	f	nil	nil	Idiopathic	2 yrs	69%	71%	77%	33%	NTC	*Tubular bronchiectasis*	No growth	No Growth	150X10*3	N-20%,L-65%		85.000
20	Sujatha	40	f	nil	nil	Idiopathic	5yrs	64%	68%	0.71	33%	gm+ve co	*Cystic bronchiectasis*	Staph.aureus	Staph aureus	190X10*3	N-70%,L-25%	Penicillin Resistant	600.000
21.	Gopal	58	m	present	present	Post TB	25 yrs	51%	55%	1	50%	gm+ve co	*Tubular+Traction bronchiectasis*	Strepto Pneumoniae	Strepo.pneumoniae	270X10*3	N-75%,L-20%	S-Erythro,cotrimoxazole	457.000

22	Poongodi	45	f	nil	nil	Idiopathic	2 yrs	67%	73%	0.77	17%	NTC	*cylindrical bronchiectasis*	No growth	No Growth	150X10*3	N-25%,L-65%		81.000
23	Kamalanathan	32	m	nil	nil	Postinfect	15 yrs	49%	33%	0.65	67%	gm-ve ba	*Cystic bronchiectasis*	Psed.aeuruginosa	Psed.aeuruginosa	410X10*3	N-65%,L-30%	Quinolon resistant	>835
24	Suresh	17	m	nil	nil	Karatagener	2 yrs	67%	81%	0.77	33%	gm-ve ba	*Cystic bronchiectasis*	K.Pneumoniae	K.pneumoniae	260X10*3	N-50%,L-40%	Cephalosporin resist	800.000
25	Chandra	35	f	nil	nil	congenital8 yrs		59%	63%	0.68	50%	gm-ve ba	*Cystic bronchiectasis*	H.influenza	H.influenza	220X10*3	N-40%,L-55%	Penicillin Resistant	429.000
26	Sathya	35	f	nil	nil	Idiopathic	2yrs	79%	81%	0.79	17%	NTC	*Tubular bronchiectasis*	No growth	No Growth	140X10*3	N-20%,L-65%		90.000
27	Anbu	30	m	nil	nil	Postinfect	1 yrs	81%	85%	0.8	17%	NTC	*cylindrical bronchiectasis*	No growth	No Growth	150X10*3	N-25%,L-65%		107.000
28	Jeyalaxmi	43	f	nil	nil	Post TB	5 yrs	74%	69%	0.75	33%	gm-ve ba	*Cystic bronchiectasis*	H.influenza	H.influenza	170X10*3	N-45%,L-50%	Penicillin Resistant	557.000
29	Nirmala	42	f	nil	nil	Congenital	15 yrs	54%	51%	0.66	50%	gm+ve co	*cylindrical bronchiectasis*	Strepto pneumoniae	Strepo.pneumoniae	110X10*3	N-40%,L-55%	S-Erytho,Amoxy	459.000
30	Suresh	22	m	nil	nil	Postaspirat	1 yr	83%	81%	0.79	17%	NTC	*Tubular bronchiectasis*	No growth	No Growth	150X10*3	N-25%,L-65%		162.000
31	Rathinambal	54	f	nil	nil	MCTD	3 yrs	59%	63%	0.74	33%	gm+ve co	*Tubular bronchiectasis*	Strepto pneumoniae	Strepo.pneumoniae	190X10*3	N-45%,L-50%	S-Quinolon,Piperacillin	800.000
32	Sumathi	39	f	nil	nil	Sjogren	5yrs	65%	61%	0.69	33%	gm+ve co	*Tubular bronchiectasis*	Strepto pneumoniae	Strepo.pneumoniae	230X10*3	N-55%,L-35%	S-Quinolon,Piperacillin	550.000
33	Vinodhini	31	f	nil	nil	Idiopathic	2 yrs	84%	85%	0.8	17%	NTC	*Cylindricalbronchiectasis*	No growth	No Growth	150X10*3	N-20%,L-65%		107.000
34	Vidhya	30	f	nil	nil	CVID	28 YRS	39%	41%	0.63	67%	gm-ve ba	*Cystic bronchiectasis*	Acinetobater species	MRSA+Acinetobater	150X10*3	N-55%,L-40%	S-Piperacillin,Quinolon	665.000
35	Sundar	37	m	present	present	Idiopathic	18yrs	41%	35%	0.66	67%	NTC	*Cystic+ Tubular bronchiectsis*	No growth	H.influenza	180X10*3	N-40%,L-55%	S-Erthro,Amoxy	560.000
36	Nagalaxmi	17	f	nil	nil	Idiopathic	2 yrs	78%	81%	0.81	17%	NTC	*Tubularbronchiectasis*	No growth	No Growth	110x10*3	N-20%,L-65%		127.000
37	Ramesh	46	m	EX SMOK	EX aicoho	Congenita	35yrs	64%	54%	0.67	67%	gm-ve ba	*Cystic bronchiectasis*	Enterococcus	Enterococcus	190X10*3	N-35%,L-50%	S-cotrimaxazole,Quinolon	360.000
38	Kalyani	43	f	nil	nil	Idiopathic	5yrs	59%	61%	0.7	50%	gm-ve ba	*Tubularbronchiectasis*	H.influenza	H.influenza	120X10*3	N-55%,L-35%	Penicillin Resistant	600.000
39	Viswanathan	51	m	nil	nil	Idiopathic	15 yrs	51%	45%	0.67	67%	gm-ve ba	*Cystic bronchiectasis*	H.influenza	H.influenza+Pseudom	210X10*3	N-50%,L-45%	Penicillin Resistant	557.000
40	Palani	75	m	EX SMOK	EX aicoho	MCTD	3 yrs	53%	42%	1	33%	NTC	*Cylindricalbronchiectasis*	No growth	No Growth	150X10*3	N-25%,L-65%		330.000
41	Selvi	30	f	nil	nil	Conghypoglobulinemia		43%	37%	0.61	67%	gm-ve ba	*Cysticbronchiectasis*	Psed.aeuruginosa	Psed.aeuruginosa	270X10*3	N-65%,L-30%	S-AGS,Cefaperazone	450.000
42	Revathi	43	f	nil	nil	Sjogren	5yrs	69%	71%	0.7	33%	gm+ve co	*Tubularbronchiectasis*	Strepto pneumoniae	Strepo.pneumoniae	120X10*3	N-45%,L-50%	S-Quinolon,Piperacillin	350.000
43	Thanikachalam	63	m	EX SMOK	EX aicoho	Rheumatoid	2 yrs	69%	53%	1	33%	gm+ve co	*Tubular bronchiectasis*	Strepto pneumoniae	Strepto pneumoniae	195x10*3	N-35%,L-50%	Not resistant to drug	457.000
44	Krishnaveni	55	f	nil	nil	Idiopathic	2 yrs	65%	71%	0.74	33%	NTC	*cylindrical bronchiectasis*	No growth	No Growth	145x10*3	N-20%,L-65%		97.000
45	Anthonyammal	45	f	nil	nil	Rheumatoid	2 yrs	69%	80%	0.79	33	NTC	*Tubular bronchiectasis*	No growth	No Growth	165x10*3	N-20%,L-65%		107.000
46	Govindaraj	36	m	EX SMOK	nil	Postaspirat	5 yrs	57%	63%	0.72	33%	gm-ve ba	*Cysticbronchiectasis*	Psed.aeuruginosa	Psed.aeuruginosa	290X10*3	N-70%,L-25%	S-AGS,Quinolone	600.000

47	Rajiya	65	f	nil	nil	Idiopathic	30 yrs	61%	52%	1.02	50%	gm-ve ba	*Cystic bronchiectasis*	H.influenza	Hparainfluenza	170X10*3	N-65%,L-30%	S-Erythro, AGS	650.000
48	Saroja	78	f	nil	nil	Idiopathic	3 yrs	59%	43%	1.04	33%	gm-ve ba	*Cylindricalbronchiectasis*	M.Catarrhalis	M.catarrhalis	180X10*3	N-50%,L-50%	S-Piperacillin,Quinolon	757.000
49	Kalyanasundaram	35	m	present	present	Idiopathic	10 yrs	69%	63%	0.68	47%	gm-ve ba	*Cysticbronchiectasis*	K.Pneumoniae	K.pneumoniae	320X10*3	N-45%,L-50%	S-Erythro,AGS	520.000
50	Ashok	31	m	nil	nil	Idiopathic	5 yrs	74%	83%	0.77	17%	gm-ve ba	*Cysticbronchiectasis*	H.influenza	H.influenza	325x10*3	N-35%,L-50%	S-Erythro,amoxy	757.000
51	Nagapusanam	22	f	nil	nil	Congenita	5 yre	76%	71%	0.72	33%	gm+ve co	*Cylindricalbronchiectasis*	Enterococcus	Streptoc viridans	150X10*3	N-45%,L-50%	S-cotrimaxazole,Quinolon	650.000
52	Devi	20	f	nil	nil	Congenita	2 yrs	84%	81%	0.8	17%	NTC	*Cysticbronchiectasis*	No growth	No Growth	155x10*3	N-25%,L-65%		320.000
53	Sasikumar	25	m	nil	nil	Postinfect	5yrs	85%	83%	0.8	17%	NTC	*Tubular bronchiectasis*	No growth	CONS	120X10*3	N-40%,L-55%	S-methicillin,Amoxy,	220.000
54	Ganesan	63	m	nil	present	Postinfect	5yrs	61%	45%	1.03	33%	gm-ve ba	*Cysticbronchiectasis*	Proteus species	proteus species	190X10*3	N-55%,L-40%	S-Quinolo,cotrimaxazol	540.000
55	Veeramani	53	m	nil	nil	Postinfect	5yrs	67%	56%	0.99	50%	gm-ve ba	*Cysticbronchiectasis*	K.Pneumoniae	K.pneumoniae	130X10*3	N-60%,L-35%	Quinolon resistant	650.000
56	Cinthamani	36	f	nil	nil	Idiopathic	3 yrs	69%	65%	0.83	33%	NTC	*Tubular bronchiectasis*	No growth	No Growth	145x10*3	N-20%,L-65%		320.000
57	Muniammal	60	f	nil	nil	Idiopathic	3 yrs	67%	59%	0.73	33%	NTC	*Cysticbronchiectasis*	No growth	No Growth	190X10*3	N-25%,L-65%		107.000
58	Madhi	57	m	present	present	Occupational 10yrs		76%	75%	0.77	33%	gm-ve ba	*Cylindricalbronchiectasis*	Enterococcus	Enterococcus	120X10*3	N-35%,L-50%	S-Quinolon,cotrimoxazol	750.000
59	Indirani	50	f	nil	nil	postinfect 1yrs		62%	56%	0.99	33%	NTC	*Cylindricalbronchiectasis*	No growth	No Growth	155x10*3	N-20%,L-65%		230.000
60	Malliga	45	f	nil	nil	postinfectious	12 yrs	59%	60%	0.76	45%	gm+ve co	*Cysticbronchiectasis*	Acinetobater species	Acinetobacter specie110x10*3	120X10*3	N-50%,L-50%	S-AGS,Piperacillin	805.000
61	Kandasamy	60	m	nil	nil	Post TB	5yrs	70%	69%	0.77	33%	NTC	*Tractionbronchiectasis*	No growth	No Growth	105x10*3	N-20%,L-65%		237.000
62	Rajamani	45	m	nil	nil	Idiopathic	5yrs	58%	63%	0.9	50%	gm-ve ba	*Tubularbronchiectasis*	K.Oxytacea	K.OXYTACEA	240x10*3	N-65%,L-30%	Quinolon resistant	770.000
63	Kannayan	53	m	nil	nil	Idiopathic	6yrs	67%	56%	0.87	33%	gm-ve ba	*Cylindricalbronchiectasis*	H.influenza	H.influenza	210X10*3	N-55%,L-35%	Penicillin Resistant	680.000
64	Sarasu	50	f	nil	nil	Idiopathic	5yrs	57%	49%	0.99	50%	Mixed	*Varicose bronchiectasis*	CONS	CONS+Acinetobacter	420x10*3	N-60%,L-35%	Penicillin Resistant	560.000
65	Manjula	37	f	nil	nil	postinfection	2 yrs	81%	78%	0.78	33%	gm-ve ba	*Tubularbronchiectasis*	Psed.aeuruginosa	Psed.aeuruginosa	200X10*3	N-70%,L-25%	S-AGS,Quinolone	760.000
66	Venkatesen	35	m	nil	nil	Idiopathic	1 yrs	84%	81%	0.77	27%	NTC	*Cysticbronchiectasis*	No growth	No Growth	150x10*3	N-20%,L-65%		107.000
67	Rukkmani	48	f	nil	nil	Idiopathic	5 yrs	55%	45%	0.67	33%	Mixed	*Varicose bronchiectasis*	Staph.aureus	MRSA+Acinetobater	210X10*3	N-65%,L-30%	S-Vancomycin,AGS	560.000
68	Saraswathi	50	f	nil	nil	Idiopathic	5 yrs	65%	54%	0.98	33%	gm-ve ba	*Tubularbronchiectasis*	M.Catarrhalis	M.catarrhalis	190X10*3	N-50%,L-50%	Not resistant to drug	660.000
69	Laksmanan	27	m	nil	nil	postinfectious	2 yrs	80%	83%	0.89	17%	NTC	*Fusiform bronchiectasis*	No growth	Pseud.aeuruginosa	370X10*3	N-55%,L-40%	S-AGS,Quinolone	790.000
70	Vijayakumari	30	f	nil	nil	Post tuberculous	3 yrs	74%	67%	0.76	33%	gm+ve co	*Varicose bronchiectasis*	Staph.aureus	CONS	210X10*3	N-55%,L-35%	Quinolon resistant	770.000

71	Anjalai	43	f	nil	nil	Post tuberculous	2 yrs	80%	75%	0.75	27%	gm-ve ba	*Tractionbronchiectasis*	E.coli	E.coli	120X10*3	N-50%,L-40%	S-Quinolon,cotrimoxazol	450.000
72	Venkatesen	30	m	nil	nil	postinfectious	1 yrs	85%	83%	o0.78	17%	NTC	*Tubular bronchiectasis*	No growth	H.influenza	120X10*3	N-45%,L-50%	Not resistant to drug	770.000
73	Mari	60	m	nil	nil	Idiopathic	5 yrs	53%	49%	1	33%	gm-ve ba	*Cysticbronchiectasis*	K.Pneumoniae	K.pneumoniae	130X10*3	N-65%.L-25%	Quinolon resistant	650.000
74	Ettiyammal	60	f	nil	nil	Idiopathic	10 yrs	56%	51%	0.99	27%	NTC	*Cylindricalbronchiectasis*	No growth	H.influenza	150X10*3	N-35%,L-50%	Not resistant to drug	350.000
75	Shanthi	36	f	nil nil		Post tuberculous	3 yrs	55%	67%	67%	42%	gm+ve co	*Tubular bronchiectasis*	Strepto pneumoniae	Strepto.pneumoniae	190X10*3	N-45%,L-50%	S-Erythro,Quinolon	450.000
76	Rajammal	55	f	n	nil	Posttuberculous	5 yrs	61%	55%	0.98	33%	gm-ve ba	*Cystic bronchiectasis*	Acinetobater species	Acinetobacter specie110x10*3	185X10*3	N-50%,L-45%	S-AGS,Piperacillin	620.000
77	Ravi	42	m	present	present	Idiopathic	2 yrs	73%	67%	0.88	33%	NTC	*Tubular bronchiectasis*	No growth	No Growth	165x10*3	N-20%,L-65%		120.000
78	Desammal	57	f	nil	nil	MCTD	2 yrs	67%	56%	0.99	27%	gm-ve ba	*Tractionbronchiectasis*	K.Oxytacea	K.OXYTACEA	290X10*3	N-45%,L-50%	Quinolon resistant	432.000
79	Selvakumar	40	m	nil	nil	Young syndrome	10 yrs	59%	56%	0.76	33%	gm-ve ba	*Cysticbronchiectasis*	Psed.aeuruginosa	Psed.aeuruginosa	210x10*3	N-60%,L-35%	Quinolon resistant	754.000
80	Seeniammal	60	f	nil	nil	Idiopathic	12 yrs	64%	53%	1	50%	gm-ve ba	*cylindrical bronchiectasis*	H.influenza	H.influenza	170X10*3	N-55%,L-40%	Penicillin Resistant	450.000
81	Murugesan	56	m	present	present	Idiopathic	5 yrs	59%	53%	0.98	42%	gm-ve ba	*Tubular bronchiectasis*	M.Catarrhalis	M.catarrhalis	160X10*3	N-50%,L-40%	S-Quinolon,AGS	800.000
82	Ponmani	25	f	nil	nil	Idiopathic	5 yrs	54%	45%	0.67	50%	Mixed	*Cysticbronchiectasis*	CONS	CONS+Acinetobacter	200X10*3	N-55%,L-40%	Cephalosporin resist	770.000
83	Ranganathan	65	m	present	nil	postinfectious	10 yrs	65%	56%	1.04	33%	NTC	*Tubularbronchiectasis*	No growth	Psed.aeuruginosa	200X10*3	N-35%,L-50%	S-AGS,Quinolone	320.000
84	Thulasiammal	69	f	nil	nil	Idiopathic	15 yrs	60%	59%	99%	38%	gm+ve co	*Cylindricalbronchiectasis*	Streptococci viridans	Streptoc viridans	180x10*3	N-40%,L-55%	S-Erythro,amoxy	620.000
85	Thirumalai	41	m	EX SMOK	EX aicoho		7 yrs	68%	58%	75%	33%	gm-ve ba	*Cysticbronchiectasis*	K.Pneumoniae	Psed.aeuruginosa	210X10*3	N-55%,L-40%	Quinolon resistant	>835
86	Rajamani	56	f	nil	nil	Idiopathic	3 yrs	73%	65%	0.97	42%	gm-ve ba	*Tubularbronchiectasis*	M.Catarrhalis	M.catarrhalis	170X10*3	N-55%,L-35%	S-Quinolon,.AGS	550.000
87	Dominiic	58	m	present	present	Idiopathic	5 yrs	72%	67%	0.7	33%	NTC	*Tubularbronchiectasis*	No growth	No Growth	145x10*3	N-30%,L-45%		97.000
88	Mariammal	29	f	nil	nil	postinfectious	2 yrs	51%	54%	0.67	50%	gm-ve ba	*Cystic bronchiectasis*	Psed.aeuruginosa	Psed.aeuruginosa	390X10*3	N-55%,L-40%	S-AGS,Piperacillin	>835
89	Sakthi	25	f	nil	nil	postinfectious	2 yrs	70%	69%	0.77	33%	gm+ve co	*Cylindricalbronchiectasis*	Staph.aureus	Staph aureus	170X10*3	N-65%,L-30%	S-methicillin, Amoxy,	450.000
90	Abdullah	19	m	nil	nil	Congenital	1 yrs	83%	81%	0.78	27%	gm-ve ba	*Cysticbronchiectasis*	H.influenza	Psed.aeuruginosa	290x10*3	N-60%,L-30%	Penicillin Resistant	767.000

gm-veba: gram negative bacilli, gm+ve co: gram positive cocci, miexd-both gram positive cocci and gram negative bacilli