ORIGINAL ARTICLE

Clinical Profile and Aetiology of Bronchiectasis

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Abstract:

Background: Bronchiectasis is a complex suppurative airway disease with multifarious etiologies and prodigious sequelae and complications. Hence we conducted a study pertaining to the same to obtain a better comprehension of its profile. *Aim and Objectives:* To study the demography, etiological factors, clinical presentations and complications in patients with Bronchiectasis. *Material and Methods:* Prospective observational study conducted in the Pulmonary Medicine department of a tertiary health care center in Mumbai after institutional ethics committee approval. *Results:* Fifty patients were included. Mean age was 34.8 years with a male preponderance. Chronic productive sputum was most common symptom while digital clubbing and crackles were the most common examination findings. Radiology played a key role in diagnosis with High Resolution Computed Tomography (HRCT) of the chest proving to be more sensitive than chest X-ray. Post infectious causes dominated the etiology followed by inherited causes, post obstructive and miscellaneous causes. *Conclusion:* Obstructive abnormality was the most common spirometric pattern. Respiratory failure was a complication observed only in cases associated with small airway disease.

Keywords: Bronchiectasis, Small Airway Disease, Pulmonary Hypertension

Introduction:

Bronchiectasis is an airway disease customarily encountered by pulmonologists and having a spectrum of presentations. The word bronchiectasis is derived from the Greek roots, Bronchion = Windpipe and Ektasis = stretching out. Bronchiectasis is present when one or more bronchi are abnormally and permanently dilated. First described by Laennec in 1819 [1] and later detailed by Sir. William Osler in the late 1800s, it has undergone significant changes in regard to prevalence, etiology, presentation and treatment. Bronchiectasis has been described as an orphan disease [2]. It can emanate from a panorama of causes broadly congregated into post infectious, inherited, auto inflammatory and post mechanical obstruction [3]. In a study carried out previously over a 3-year period, which included 150 patients having bronchiectasis using High-Resolution Computerized Tomography (HRCT) identification of one or more causative factors was possible in 47% of cases [4]. The causes identified were mainly intrinsic defects or noninfectious extrinsic insults predisposing to bronchial inflammation or infection. Literature available pertaining to the etiology and profile of bronchiectasis in a non-white Caucasian population is exiguous. The present study was thus undertaken to evaluate the demography, causative factors, clinical presentation, radiology and complications of bronchiectasis in a population where the incidence of pulmonary tuberculosis, human immunodeficiency viral disease, and other childhood lower respiratory tract infections are higher.

Material and Methods:

The study population consisted of 50 consecutive patients with bronchiectasis. Since Bronchiectasis is an orphan disease with exact prevalence not documented uniformly in literature, accurate sample size calculation was not possible. Hence patients were enrolled on a consecutive basis during a specific time frame spanned over a period of 2 years. They were diagnosed on the basis of history, clinical findings and HRCT of the chest. It was a prospective study undertaken in the department of Pulmonary Medicine at a tertiary care centre in Mumbai conducted over a period of two years. The Institutional Ethics Committee approval was obtained and patients were included in the study after obtaining an informed written consent. Their demographic profile was obtained which included name, age, sex, residence, occupation and addictions if any with emphasis on smoking. Clinical history was obtained with the aid of a standard forma which included details of cough, expectoration, haemoptysis, dyspnoea, and fever with their onset and duration. To obtain a clue to the possible etiology, history of sinusitis, atopy in self or related family members, history of childhood pneumonia or viral exanthema prior to onset of symptoms was noted.

History of consanguineous marriage in parents, similar respiratory illness in family members, sibling deaths due to respiratory infections, and history of infertility if any was documented to attain a clue to possible inherited etiology. History of any past significant illnesses such as pulmonary tuberculosis with details of diagnosis and treatment and other co-morbid conditions such as diabetes mellitus, hypertension, and ischemic heart disease was obtained. The frequency of infective exacerbations in the form of increased sputum production, purulent sputum and fever was noted. A note of whether hospitalization was required for the same and details of treatment in the form of oxygen, non-invasive or invasive ventilation was made. History of pedal edema with or without oliguria was obtained which was suggestive of right heart failure due to cor-pulmonale or due to secondary renal amyloidosis. Details of lung surgeries if any in the form of lobectomy or pneumonectomy with its indications and operative details were noted. Clinical examination included presence of clubbing with its grade and presence of crackles and or rhonchi on respiratory system examination.

The patients included in the study were subjected to following investigations-

1) Hematological and biochemical investigations: hemoglobin, complete blood counts with differential, white blood cell count, liver function tests, renal function tests, and pretest counseling Enzyme Linked Immunosorbent Assay (ELISA) for Human Immunodeficiency Virus (HIV).
2) Radiological investigations: chest X-ray, HRCT of the thorax using expiratory scans with measurement of tracheal diameters on inspiration and expiration.
3) Assesssment of lung function with spirometry and recording the variables Forced Expiratory Volume in First Second (FEV1), and Forced Vital Capacity (FVC) ratio.
4) Cardiac assessment with Electrocardiogram (ECG) and 2-dimensional Echocardiography (2D ECHO)

5) Serum Immunoglobulin E (IgE), X-ray paranasal sinuses, serum specific IgE against *Aspergillus fumigatus* in patients with history of atopy, allergy, or bronchial asthma.
6) Patients who were suspected to have a inherited cause for bronchiectasis were further evaluated by selected investigations, including serum Immunoglobulin G (IgG), Immunoglobulin M (IgM) and Immunoglobulin A (IgA) for hypogammaglobulinemia, sweat chloride test and delta F508 chromosomal analysis for cystic fibrosis, nasal mucosal brush biopsy under electron microscopy and/or semen analysis for Primary Ciliary Dyskinesia (PCD).
7) Other investigations such as RA factor in suspected cases of rheumatoid arthritis, specific investigation in suspected cases of ankylosing spondylitis, ulcerative colitis, and Marfans syndrome.
8) Arterial blood gas analysis
9) Urine albumin with or without 24 hours urinary proteins.

Results:

The age group of patients included in this study ranged from 6-60 years, the mean age being 34.8 years. There were 31 males and 19 females. All patients had symptoms of cough with expectoration; dyspnoea was present in 92% of cases and haemoptysis at the time of presentation or in the past in 44% of cases. Fifty percent of patients had fever at the time of presentation and 50% of cases had associated sinusitis. (Table1). A family history of similar respiratory complaints could be obtained in 6% of patients, 20% of patients had history of sibling death in childhood either secondary to a respiratory illness or unknown cause. Twenty-four percent of patients gave a history of consanguineous marriage in their parents. Three patients (6%) were smokers, of which only 1 had evidence of centrilobular emphysema on HRCT.

Examination findings revealed digital clubbing in 84% of cases. On respiratory system examination, 44% of patients had rhonchi, and all patients had coarse crackles, of which 56% were bilateral whereas. Abnormalities in chest X-ray were seen in 36(72%) cases whereas 28% patients had normal X-rays of the chest. HRCT of the thorax identified unilateral disease in 52% of cases and bilateral disease in 48% (Table 1). All patients had cystic bronchiectasis, whereas 6 patients (12%) had associated changes of cylindrical bronchiectasis. None had varicose bronchiectasis on HRCT (Table 1). Sixty-eight percent of patients had areas of diffuse air trapping on HRCT with or without worsening of the same on expiratory scans.

The etiology of bronchiectasis could be evaluated in all cases (Table 1). Twenty-seven (54%) patients had predated their chronic symptoms to childhood viral exanthematous illness that was followed by pneumonia or a lower respiratory tract infection. Of these exanthems, three patients reported childhood varicella, two cases reported measles, and the rest did not report specific viral exanthems, but had a history of fever with rash followed by a lower respiratory tract infection preceding their symptoms. Seven (14%) of the cases had histories of symptoms of bronchiectasis occurring post treatment for a documented sputum positive pulmonary tuberculosis. Four (8%) patients had bilateral central bronchiectasis associated with Allergic Bronchopulmonary Aspergillosis (ABPA), having histories of asthma,

atopy, bilateral rhonchi on examination, elevated serum Immunoglobulin E and positive serum specific Immunoglobulin E against *Aspergillus fumigatus*. Four (8%) patients had bilateral bronchiectasis secondary to primary tracheo-bronchomegaly (Mounier Kuhn syndrome), identified on HRCT as tracheal diameter of more than upper limit of normal mean ± S.D. (i.e. 21.8mm in men and 19.4mm in women). Four patients (8%) had bronchiectasis secondary to PCD (3 bilateral, 1 unilateral), of which one patient had Kartagener's syndrome (triad of sinusitis, bronchiectasis and situs inversus). Three other patients had sinusitis, bronchiectasis and nasal mucosal biopsies displaying ultrastructural ciliary abnormalities. The most common ultra-structural ciliary abnormality observed was absence of outer dynein arms, followed by abnormalities of the microtubule configuration. One patient had bronchiectasis due to cystic fibrosis, diagnosed by elevated levels of sweat chloride (> 60mEg/L) tested by pilocarpine iontophoresis, with recurrent lower respiratory tract infections. However chromosomal analysis did not reveal an abnormal mutation of the Delta-F508 mutation. Bronchiectasis distal to an anatomical obstruction to the airways was seen in one case of a mediastinal dermoid cyst causing a left lower lobe collapse with bronchiectasis. One patient had developed bilateral bronchiectasis secondary to recurrent lower respiratory tract infection due to Job's syndrome, a neutrophil chemotactic factor defect, one patient had bilateral bronchiectasis in association with acromesomelic dysplasia, a developmental skeletal anomaly. Spirometry demonstrated obstructive abnormality in 25 (50%) patients of which 14 (28%) had good bronchodilator reversibility (BDR; i.e. post bronchodilator improvement FEV1 of more than 200 ml and 12%). Ten patients 20% had restrictive pulmonary abnormality, 10 patients 20% had mixed obstructive and restrictive abnormality, whereas five (10%) patients had a normal spirometry (Fig. 1). Of those, who demonstrated obstructive pulmonary abnormality, 19 (76%) had evidence of small airway disease on HRCT. Nine (18%) patients had pulmonary hypertension, of which 7 patients had cor-pulmonale (i.e. dilated right ventricle and right atrium). Of those patients having cor-pulmonale, 6 patients had associated changes of small airway disease on HRCT. Seven (14%) patients with no hypertension, diabetes mellitus or primary renal disease had evidence of proteinuria on urine analysis that suggested secondary renal amyloidosis. However, confirmation of the same by renal biopsy was not possible. All patients were systematically followed up for 3 years. Nine (18%) patients developed respiratory failure during the 3 year follow up period of which three had Type I and six had type II respiratory failure. Of these 9 patients, seven patients required hospitalization and 2 could be managed on outpatient basis.

Management of respiratory failure included nasal oxygen in 4 of the 7 hospitalized cases (57%), whereas 3 (42.8%) required non-invasive ventilation for correction of hypoxemia. None of them required mechanical ventilation. Out of these 7 hospital admissions for respiratory failure, 2 patients had associated right heart failure.

Table 1: Demographic Data, Etiology, Symptoms, X-ray Findings of Study Subjects

	Age (Range)	6-60 years	
	Sex (Male / Female)	31/19	
Sr. No.	**Variable**	**Number**	**Percentage**
1.	**Symptoms**		
	a. Cough	50	100
	b. Dyspnoea	46	92
	c. Hemoptysis	22	44
	d. Fever at the time of presentation	25	50
	e. Sinusitis	25	50
2.	**Associated Antecedent Illnesses of Potential Etiology for Bronchiectasis**		
	a. Post infectious (with clinicoradiological correlation)	27	54
	b. Post tuberculous sequelae	7	14
	c. Allergic bronchopulmonary aspergillosis	4	8
	d. Primary Tracheobronchomegaly	4	8
	e. Primary Ciliary Dyskinesia (PCD)	4	8
	f. Kartageners syndrome	1	12.5
	g. Others (sinusitis, bronchiectasis and ultrastructural ciliary defects)	3	87.5
	h. Cystic fibrosis	1	2
	I. Post obstructive	1	2
	j. Jobs syndrome	1	2
	k. Acromesomelic dysplasia	1	2
3.	**Type of Bronchiectasis Identified on HRCT**		
	a. Cystic	50	100
	b. Cylindrical	6	12
	c. Varicose	0	0
	d. Air trapping on HRCT	34	68
4.	**Radiology**		
	a. Abnormal chest X-ray	36	72
	b. HRCT abnormality	50	100
	c. Unilateral disease	26	52
	d. Bilateral disease	24	48

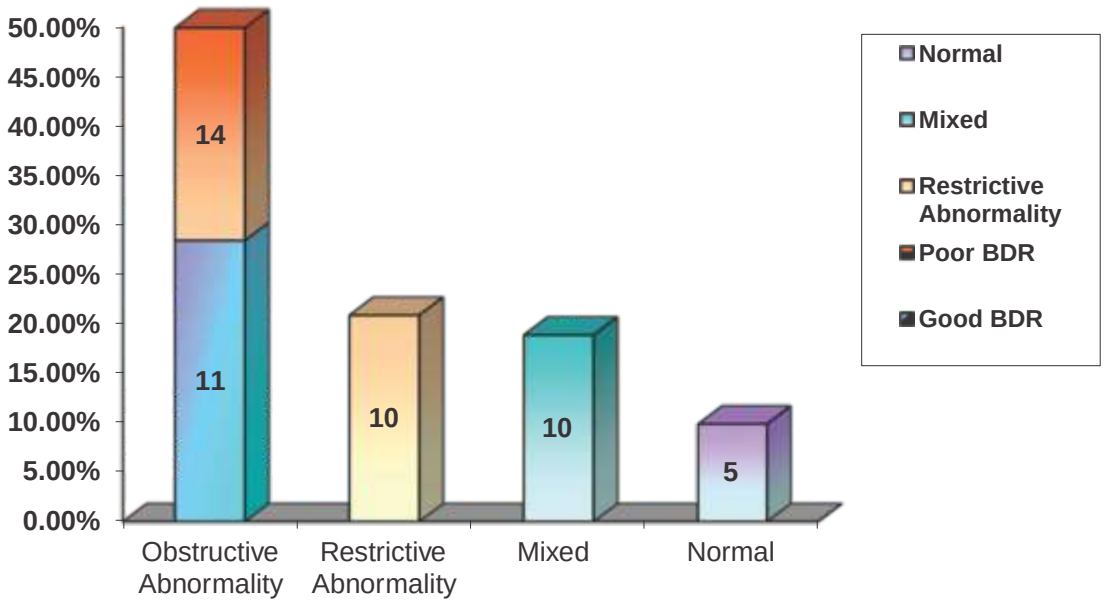

Fig. 1: Spirometry Pattern in Bronchiectasis

Discussion:

Bronchiectasis is a progressive airway disease with its pathogenesis enrooted in the Coles hypothesis denoting the vicious cycle of airway inflammation, colonization dilatation and remodeling [5]. We have reviewed 50 patients with bronchiectasis confirmed on HRCT, characterized the manifestations of the disease and evaluated the causative factors. The patients studied were evaluated on outpatient basis that followed up on their own due to respiratory symptoms or were referred by other physicians; hence this study does not provide data on the prevalence of bronchiectasis. The patients were drawn from a population base that is Indian (Nonwhite Caucasian) with a high incidence of HIV and pulmonary tuberculosis, which must be considered when extrapolating the results to other groups. In comparison with most of the previous studies in bronchiectasis having a predominance of female subjects, we have identified more male patients. Bronchiectasis caused by infection occurred predominantly in middle aged to elderly populations, while those associated with congenital defect occurred in younger patient. The mean age of presentation was 34.8 years as compared to 52 and 57 years as seen in other studies [4, 6].

Chronic productive sputum was seen in all patients as the hallmark of the disease either on a daily basis or only during episodes of upper respiratory tract infections. None of the patients had the so-called 'dry bronchiectasis'. Dyspnoea was present in 92% cases in our study as compared to 71% of patients observed [6]; haemoptysis was seen in 44% cases which occurred in about 63% of bronchiectasis patients in the comparative study. Digital dubbing was observed in 84% cases which were strikingly in contrast to the comparative study [6]. Forty five percent patients had wheezing which was identical to the earlier study [6]. Crackles on respiratory system examination were observed in all cases in our study i.e. 100%

(56% bilateral, 44% unilateral) as compared to 86% of the patients in the compared study [6].

Radiology plays a pivotal role in clinching the diagnosis after a clinical suspicion. On chest radiographs bronchiectasis displays distinctive patterns like tram tracks, parallel line opacities, ring shaped opacities and tubular structures [7]. However HRCT edges forward, owing to its higher sensitivity and assisting to radiologically pigeon hole the subtypes [7]. HRCT was diagnostic in all our cases (100%) of bronchiectasis in keeping with the high sensitivity (84-97%) and specificity (82-99%) of this tool as compared to chest X-ray, which was abnormal in 72% cases and normal in the rest. However in a comparative study done between chest radiography and high resolution CT in 84 patients, a normal chest radiograph almost always excludes relevant bronchiectasis and no further investigation seemed necessary [8]. There was a significant linear relationship between the severity of bronchiectasis on HRCT and abnormalities as seen on the chest radiograph [8]. All patients had evidence of cystic bronchiectasis in our study (100%); twelve percent had associated cylindrical bronchiectasis on HRCT, while none showed the varicose type. In an attempt to assess accuracy of HRCT in the differentiation of specific disease, post infectious bronchiectasis is seen predominantly in lower lobes (85%), post tuberculosis in upper lobes (71.4%), bilateral changes in tracheobronchomegaly, proximal or central bronchiectasis in allergic bronchopulmonary aspergillosis and cystic fibrosis. Of patients who had PCD, 50% had bilateral disease whereas 50% had unilateral bronchiectasis. In a retrospective study of 82 patients [9] who had a specific diagnosis of bronchiectasis proven by appropriate clinical and laboratory criteria, bilateral upper lower predominant bronchiectasis was seen most commonly in patients with cystic fibrosis and allergic bronchopulmonary aspergillosis, unilateral upper lobe predominance in patients with tuberculosis and lower lobe predominance in patients after childhood viral infection. Thus, the pattern and distribution of abnormalities revealed by HRCT in patients with bronchiectasis are influenced by the underlying cause. Sixty eight percent demonstrated significant areas of decreased attenuation on expiratory CT due to air trapping (mosaic perfusion) commonly seen in severe bronchiectasis and predominantly post infectious bronchiectasis. Such areas in lobes can be seen without overt bronchiectasis and may suggest that small airway disease may precede the development of bronchiectasis [10].

The etiology of bronchiectasis is heterogeneous and can sub grouped into post infectious, post obstructive, post aspiration, immunological, congenital, COPD associated and idiopathic [11]. The etiology of bronchiectasis could be evaluated in all 50 patients and a comparison was made with a similar study of 150 cases to evaluate for the causative factors in bronchiectasis [4]. In our study, 54% of bronchiectasis was due to post infectious etiology (post viral exanthematous illness followed by pneumonia or lower respiratory tract infection) of which 11% were post varicella and 7% after childhood measles. Eighty two percent of patients had no definite viral illness but dated their symptoms to an episode of fever with rash followed by lower respiratory tract infection preceding chronic symptoms. In the comparative study, respiratory tract symptoms began at a young age in many, 40% were younger than 10 years of age at onset of symptoms and no cause was identified in 60% of these. Of the post-infectious causes in their study, 48% had a history of pneumonia, 21% had pertussis and 13% had

childhood measles, all which predated the onset of chronic lower respiratory tract infections [4]. Fourteen percent of patients developed bronchiectasis as post-tuberculous sequelae, as compared to 2% in the compared study [4]. Eight percent had ABPA with central bronchiectasis in our study whereas ABPA accounted for around 10% of cases of bronchiectasis in their series [4]. Another 8% had bilateral bronchiectasis due to primary tracheobronchomegaly (Mounier Kuhn syndrome) which was not seen in the comparative study. 8% of cases were due to PCD of which 1(25%) was identified as Kartageners syndrome and remaining 75% demonstrated ultrastructural ciliary abnormality on nasal mucosal brush biopsy the commonest being absence of outer dynein arms. In the comparative study, three patients (2%) were identified with ciliary defects of which 1 had Kartageners syndrome, two had absent or uncoordinated ciliary movement with ultra-structural abnormalities on electron microscopy of which one had Young's syndrome secondary to childhood mercurial poisoning (pink disease) known to affect ciliary function. Humoral Immunodeficiency (antibody deficiency and, or a defective antibody response) was defected in 8% of cases in their study however no patient had the same in ours.

One patient (2%) had bronchiectasis due to cystic fibrosis in comparison with an incidence of 5% based on elevated sweat chloride levels and 6% based on CF gene mutations. One patient developed bronchiectasis due to recurrent lower respiratory tract infections secondary to Job's syndrome characterized by recurring bacterial infections of the skin and sinopulmonary tree. A study on based on experience of 4 patients with Job's syndrome suggested that recurrent respiratory infections and bronchiectasis are common pulmonary complications of the syndrome [12].

Post obstructive bronchiectasis was seen in 1 case involving the left lower lobe due to extrinsic compression by a mediastinal dermoid cyst. One patient had bilateral bronchiectasis in association with acromesomelic dysplasia a developmental skeletal disorder. Bronchiectasis in skeletal dysplasias is postulated to be secondary to dys-gammaglobulinemia leading to severe pulmonary infections [13]. Thus in the series of 150 cases studied earlier, one or more cause was identified in 70 patients (47%), whereas no definite cause could be established in 80 patients (53%) [4].

Spirometry is a serviceable tool in patients with bronchiectasis. Presence of reduced FEV1 adumbrates higher risk of frequent exacerbations, hospitalizations, accentuated mortality and a poor quality of life [14]. Spirometry in our study demonstrated predominantly obstructive abnormality in 50%, 29% having good broncho-dilator reversibility, restrictive patterns in 21% and mixed abnormality in 19%, normal lung functions in 10%. Of those who demonstrated obstructive pulmonary defect, 76% had evidence of small airway disease on HRCT. In a study of inspiratory and expiratory features on CT scan of 100 patients with bronchiectasis undergoing concurrent lung function tests, the extent and severity of bronchiectasis, the severity of bronchial wall thickening and the extent of decreased attenuation on the expiratory CT scan correlated strongly with the severity of airflow obstruction [10]. The closest relationship was seen between decreased FEV1 and the extent of decreased attenuation on the expiratory CT scan. An important finding of our study was that majority of the patients with pulmonary hypertension and cor-pulmonale had associated small airway disease (89% patients).

This is in contrast to the patient without associated airway disease, where only 1 patient developed cor-pulmonale. This may indicate that pure bronchiectatic changes rarely result in pulmonary hypertension whereas associated small airway disease significantly contributes to pulmonary hypertension and subsequent cor-pulmonale.

Secondary renal amyloidosis due to chronic suppuration as sequelae of bronchiectasis was suspected in 14% patients. In a retrospective evaluation of 40 cases for causes of secondary amyloidosis excluding cases with causes other than bronchiectasis, secondary amyloidosis was identified in 40% cases [15]. Of the 18% patients who developed respiratory failure during the follow up period Type II failure was seen predominantly and all (100%) who had evidence of small airway disease on HRCT. All of the hospitalizations were for respiratory failure, fifty seven percent of which could be managed by nasal moist oxygen rest 43% required noninvasive ventilation for correction of hypoxemia. Twenty two percent of these cases of respiratory failure had concomitant right heart failure. This indicates that respiratory failure is uncommon in bronchiectasis where symptoms are adequately controlled, high incidence being in patients who develop recurrent infective exacerbations or have changes of small airway disease. Bronchiectasis has been described as an orphan disease with a prevalence estimated to be low and decreasing, the true prevalence most likely is underestimated and less severe forms of bronchiectasis have been documented with the increasing use of HRCT. The lack of adequate vaccination campaigns and the persistence of tuberculosis in the developing countries are resulting in appearance of a constant number of new cases. We have investigated a sample of cases representative of the general population and evaluated the cause of bronchiectasis in all. In comparison with the previous study, we have identified more cases of post-infectious bronchiectasis due to respiratory tract infections or pneumonia following viral exanthematous illness in childhood. This was followed by post-tuberculous cases of bronchiectasis. The remaining cases were secondary to noninfectious or intrinsic causes, clearly stating that infectious causes of bronchiectasis are still predominant in the etiology. The clinical spectrum and the diagnosis by radioimaging did not differ. Small Airway Disease (SAD) identified on HRCT was predominantly seen in post-infectious bronchiectasis and contributed to the obstructive pulmonary defect on spirometry and to the development of pulmonary hypertension and cor pulmonale indicating that the association of SAD with bronchiectasis has a higher incidence of complications than the disease itself. Most of the patients were adequately controlled on chest physiotherapy and postural drainage requiring antibiotics for infective exacerbation's and oxygen with or without positive pressure ventilation for respiratory failure.

Patients who had a hereditary cause of bronchiectasis such as cystic fibrosis, PCD were managed conservatively. Surgery as a therapeutic option to remove the diseased part of the lung was strongly deferred in them due to the presence of extensive, bilateral disease in most and due to the progressive nature of the disease. These patients were offered genetic counseling to reduce the incidence of the disease in their family.

Conclusion:

Bronchiectasis is a heterogenous entity with varied etiologies and multifarious clinico-radiological patterns. Radiology plays a key role in the diagnosis. The prognosis is favorable with opportune diagnosis and therapy. Our study illustrated the common etiologies, varied clinical presentations, importance of radiology, common spirometry patterns and the affirmative prognosis in the disease with timely management.

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