

Factors Affecting the Outcome of Bronchiectasis in Pediatric Patients

*Nemat Bilan¹, Mitra Aghakhani², Farhad Niafar³

¹Pediatric Pulmonologist.

² Pediatrician, Tabriz University of Medical Sciences, Tabriz, Iran.

³ Internist, Faculty of Medicine, Tabriz University of Medical Sciences, Tabriz, Iran.

Abstract

Introduction

Bronchiectasis is a common problem in children and early diagnosis can lead to early treatment and prevent of its complications. This study was aimed to evaluate factors effectiveness on outcome of bronchiectasis in children.

Methods and Materials

In an analytical cross-sectional study, 347 children with bronchiectasis underwent the study. The patients were diagnosed based on chronic suppurative cough and Computerized tomography(CT) scan findings.

Results

Disease etiology was asthma in 55.6%, Gastroesophageal reflux disease (GERD) in 7.8%, Cystic fibrosis (CF) in 4.8%, other causes in 11.2% and idiopathic in 20.6%. All cases complained of chronic cough. The most common sign was daily sputum production (79.1%) and common symptoms were ral/crackle in 47.1% and wheezing in 25.4%. Mean treatment period was 32.82 ± 11.56 months. At the end of follow-up, complete improvement occurred in 35.6%, partial improvement in 40.9% and no improvement in 23.5%.

Conclusion

In children with chronic cough and crackle in physical examination, consideration of bronchiectasis could be helpful in early diagnosis and complementary evaluations and treatment initiation. Treatment of the underlying disease could prevent the occurrence and increase the response to treatment of bronchiectasis.

Keywords: Bronchiectasis, Etiology, Children, Outcome.

*Couresponding Author:

Nemat Bilan, MD, Tabriz Children's Hospital, Sheshgelan St, Tabriz, Iran; Fax: +9841135262280. E-mail: bilannemat@yahoo.co.uk Received date: Jul 5, 2014 ; Accepted date: Nov 12, 2014

Introduction

Bronchiectasis remains as an important health problem in both, developed and developing countries (1). It results in impaired quality of life and mortality if left untreated (2). Bronchiectasis is not a disease. primary but an anatomical abnormality which is caused by various factors. According to the literature, the primary etiologic factor cannot be found in 30-74% of the patients (3-5). Early diagnosis based on detailed medical history and radiological confirmation is important in order to establish a treatment focused on the underlying cause. For this purpose, an orderly and systematic diagnostic evaluation is required (1).

The management of bronchiectasis includes medical interventions as well as adjunctive therapies and finally surgery. The therapeutic modalities are provided following goals: with the aggressive treatment of infections, treatment of the promotion underlying disease. of mucociliary clearance, promotion of normal growth, avoidance of toxins, identification management and of complications, and treatment of the chronic inflammation to retard disease progression (2,6). Bronchiectasis is still a challenge to the pediatric chest physicians in many developing parts of the world and it remains a frequent problem being the final common pathway of several different lower respiratory tract insults such as cystic fibrosis, immunodeficiency, ciliary dyskinesia. There are unanswered questions about childhood bronchiectasis, mainly on etiology and treatment which require more research (7).

It seems that bronchiectasis is a neglected disorder, with no adequate diagnosis and treatment

(3). There are few studies published about bronchiectasis, clinical findings and response to treatment in children. This study was aimed to evaluate factors effective on the outcome of bronchiectasis in children.

Methods and Materials

This is a cross- sectional and analytic study performed on 374 pediatric patients with bronchiectasis presenting to Children Hospital and Outpatient Pediatric Clinics of Tabriz University of Medical Sciences since 2006 till 2010. Inclusion criteria were having bronchiectasis documented manifestations (including bv clinical chronic suppurative cough), and findings on CT scan, which was reported by one radiologist and repeated every 6 to 12 months. Exclusion criteria were concurrent medical disorders, congenital anomalies, previous medications. The patients and were treated for 2-3 years, using steroid bronchodilator inhalers. sprays. and continuous low- dose oral antibiotic. Then, according the clinical findings and CT scan results the patients were classified in three groups including Recovered. Partially recovered, and Non- recovered:

• The children in "Recovered group" were those in whom the clinical findings including suppurative cough) have been relieved, and control CT scan showed the improvement of radiologic findings in comparison with the basic CT, and the medication was stopped.

• The children in "Partially recovered" group continued receiving the decreased dose of medication because of partial improvement in suppurative cough.

• The children in "Non- recovered" group did not have any improvement in clinical findings and CT scan findings.

The required data were including patients' age, sex, symptoms and signs, imaging results, medical history, family history, risk factors, medications, duration of treatment, and response to the therapy. The were collected from patients' data medical records using prepared questionnaire, and then were compared between the three groups. The study was confirmed by Regional Ethic Committee, and the personal information of all patients was handled as secret.

The collected data were analyzed by SPSS-16 statistical software by using descriptive methods (including abundance, percent, average and standard deviation). The qualitative variables were compared by chi- square, and the quantitative variables were compared by One- way ANOVA test. Also, independent t- test was applied for comparison of quantitative variables in dual groups. The p- values less than 0.05 were considered as statistically significant.

Results

of 374 children Α total with bronchiectasis were studied. The patients had the age of 2 to 17 years (average: 8.61±3.36; median: 9). Of all the patients studied, 240 (64.2%) were male and 134 (35.8%) were female. Medical history was positive in 51 (13.6%) including preterm birth in 22 (5.9%), recurrent pneumonia in 16 (4.3%), cleft lip in 4 (1%), pulmonary lobe collapse in 3 (0.8%), asphyxia in 3 (0.8%), congenital metabolic disorders in 2 (0.5%), and pulmonary lobectomy in 1 (0.3%). Familial history of respiratory disease was positive in 65 (17.4%).

The etiology was idiopathic in 77 (20.6%) and known in 297 (79.4%), including asthma in 208 (55.6%), GERD in 29 (7.8%), Cystic fibrosis (CF) in 18 (4.8%), Chest physiotherapy (CP) in 9 (2.4%),

pneumonia in 4 (1.1%), hypotonia and repeated aspiration in 3 (0.8%), Chronic liver disease (CLD) in 5 (1.3%), Down syndrome in 5 (1.3%), esophageal atresia in 2(0.5%), obesity in 2 (0.5%), and pulmonary abscess, achalasia, congenital myopathy, bone deformity and unilateral paresis of vocal cords, each in one case (0.3%).

Symptoms were as following: cough in 374 (100%), Chronic suppurative cough (CSC) in 296 (79.1%), CSC with recurrent pneumonia in 14 (3.7%), CSC with dyspnea in 30 (8%), CSC with repeated aspiration in one (0.3%), CSC with tachypnea in 3 (0.8%), and with hemoptysis in 11 (2.9%). Also, there was hemoptysis with pneumonia in 8 (2.1%), recurrent pneumonia in 6 (1.6%), dyspnea in 4 (1.1%), and pneumonia with pulmonary collapse in one (0.3%).

The clinical findings were as following: rales (as only sign) in 176 (47.1%), wheezing in 95 (25.4%), rales and wheezing in 24 (6.4%), coarse crackle in 75 (20.1%), and clubbing in 4 (1.1%).

CT scan showed the evidences of bronchiectasis including thickening and dilatation of bronchi and/or cystic changes in all patients.

The Medications used in studied were: antibiotic+ seretide in 216 (57.8%), antibiotic+ flixotide+ salmeterol in 91 (24.3%), antibiotic+ seretide+ anti-reflux in 49 (13.1%), and antibiotic+ seretide+ treatment for CF in 18 (4.8%). The duration of the treatment was 4 to 66 months (mean= $32.82\pm11.56m$, median= 30m).

In follow up, the symptoms and signs were relived in 227 (60.7%), but did not improve in 147 (39.3%). Imaging revealed the relief in 140 patients (37.7%) and the decrease in disease severity in remaining 234 (62.6%).

It was reported the need for repeated presentation to emergency ward and readmission in 82 cases. The presentation to emergency ward and readmission was reported 1 to 7 times (mean= 1.41 ± 0.87 , median= 1). The total of visits of children by physician was occurred 5 to 60 times $(\text{mean}=22.44\pm10.24, \text{median}=20).$

At the end of follow up, the patients' outcome were as following: complete recovery and termination of the treatment in 133 (35.6%), partial recovery and continuing the medications with reduced dose in 153 (40.9%), and not recovery and continuing the medications with the same dose in 88 (23.5%).

Figure.1 shows the mean age of studied patients and the disease outcome. As seen in the figure, the patients without the response to the therapy are younger than other groups

(p=0.88). Table.1 shows the relation of patients' sex and duration of medication with the disease outcome.

As seen in the table, the male patients have better response to the therapy than females (p=0.06). Also, the duration of medication was not significantly different in three groups (p=0.16). Also (Table.2) shows the effect of some variables on response to the therapy in studied patients.



Table 1 : Patients' sex and duration of treatment in three studied groups								
Variables	Complete	Partial	No	p-value				
	Recovery	Recovery	Recovery					
Male	100 (25.40%)	95 (23.80%)	60 (14.97%)	>0.05				
Female	42 (10.16%)	70 (17.11%)	38 (8.56%)	>0.05				

34.43±10.47

31.35±9.83

Duration of medication (m)

alue

>0.05

32.90±12.8

Variables	Complete	Partial	No	P- value				
	Recovery group	Recovery group	Recovery group					
History of previous disease	19 (14.3%)	19 (12.4%)	13 (14.8%)	0.84				
Familial history of respiratory disease	19 (14.3%)	34 (22.2%)	15 (13.6%)	0.12				
Know etiology for bronchiectasis	110 (37%)	122 (41.1%)	65 (21.9%)	0.27				
Unknown etiology (<i>idiopathic</i>)	23 (29.9%)	31 (40.2%)	23 (29.9%)					
With Asthma	82 (39.4%)	98 (47.1%)	28 (13.5%)	<0.001				
Without Asthma	51 (30.7%)	55 (33.1%)	60 (68.2%)	<0.001				
With GERD	17 (58.6%)	7 (24.1%)	5 (17.2%)	0.02				
Without GERD	116 (33.6%)	146 (42.3%)	83 (24.1%)	0.02				
With CF	1 (5.6%)	5 (27.8%)	12 (66.7%)	<0.001				
Without CF	132 (37.1%)	148 (41.6%)	76 (21.3%)	10.001				
GERD: Gastroesophageal reflux; CF: Cystic fibrosis								

Table 2: the effect of some variables on response to the therapy in studied patients

As seen in table 2, the response to medication was better in patients with asthma than patients without it (p<0.001). The presence or absence of known etiology for bronchiectasis was not effective on the response to the medication (p=0.27).

The rate of complete recovery was more in patients with GERD than patients without it (p=0.02). The response to medication was lower in patients with asthma than patients without it (p<0.001).

The imaging findings were bronchial thickening and dilatation and other signs of bronchiectasis, and the response to the therapy was not significantly different between various findings in imaging (p=0.69).

(Table.3) compares the outcome of various medications scheduled for studied patients. The complete and partial recovery was reported at least level in CF patients with bronchiectasis (cystic fibrosis bronchiectasis).

The set of							
Medication	Complete	Partial	No				
	Recovery	Recovery	Recovery				
Seretide	70 (32.4%)	104 (48.1%)	42 (19.5%)				
Salmetrol+Flixotide	29 (31.9%)	33 (36.2%)	29 (31.9%)				
Seretide & Medication for GERD	33 (67.3%)	11 (22.4%)	5 (10.2%)				
Seretide & Medication for CF	1 (5.6%)	5 (27.8%)	12 (66.7%)				
GERD: Gastroesophageal reflux; CF: Cystic fibrosis; *Antibiotics were used in all cases.							

Discussion

We evaluated factors effective on the outcome of bronchiectasis in 374 children with the clinical and radiographic diagnosis of bronchiectasis. Bronchiectasis is a morphological term used to describe abnormal irreversibly and permanent dilated (Ectasia) and often thick walled bronchi (8-15). The bronchial wall is typically thickened by an inflammatory infiltrate of lymphocytes and macrophages (14). Bronchiectasis has a variety of causes and traditionally been viewed has as а that is irreversible, often condition progressive and associated with significant morbidity and mortality (15). It has recently been demonstrated that patients with bronchiectasis have a progressive decline in lung function (14).

The prevalence of bronchiectasis has decreased significantly in industrialized countries, but is still commonplace in developing (3,16). countries Although morbidity and mortality from bronchiectasis seem to be declining in Western countries, the condition is still one of the most common reasons for morbidity in developing countries (17).

Extremes of age and smoking/chronic obstructive pulmonary disease may be

important considerations. There are а variety of different pathogens involved in bronchiectasis, but a common finding despite the presence of purulent sputum is pathogenic failure to identify any microorganisms. The bacterial flora appears to change with progression of disease (14). Bronchiectasis is a pathologic description of lung damage characterized bv inflamed and dilated thick-walled bronchi. These findings may result from a number of possible causes and these may influence treatment and prognosis (18).

The pathogenesis of bronchiectasis is incompletely understood. The most commonly proposed pathophysiological mechanism is the "vicious cycle theory" whereby an initial insult damages the respiratory tract resulting in impaired mucociliary clearance. This leads to chronic bacterial infection associated with а persistent inflammatory response producing fibrotic changes. The initial trigger is often infective although other factors be considered, must also predispose particularly those that to bronchial and pulmonary infection including immunodeficiency and anatomical abnormalities of the airways (8).

Identification of predisposing causes may facilitate prevention of further bronchial damage (19). Twiss et al. reported etiology to be unknown (idiopathic) in 54% of cases, post infectious in 22%, as a oncological of disease result or chemotherapy in 11%, aspiration 6%, and immunodeficiency primary 6% (9). Idiopathic bronchiectasis was found in 13 patients (14%) (20).Nikolaizik and Warner (19) reviewed 41 cases of bronchiectasis who presented with chronic productive cough, and no cause was found in 37% of their study group. Forty- eight percent of studied subjects from a New Zealand despite cohort. extensive investigations, had no known cause for bronchiectasis (21, 22).their In the present study, the etiology was unknown (idiopathic) in 77 (20.6%). This discrepancy may be due to the differences in referral patterns and study populations in the studies.

Unlike the results obtained in this study, the etiologies of bronchiectasis, reported by a study in New Zealand were as following: infections (22%), immunosuppression/ chemotherapy (17%), aspiration (6%), and idiopathic (54%) (9).

Nikolaizik and Warner suggested that of 4000 children referred for respiratory disease, 41 (1%) had chronic suppurative lung disease not due to cystic fibrosis. Further investigations showed congenital malformations in six (15%), primary ciliary dyskinesia syndrome in seven (17%), 11 had immunological abnormalities (27%), and two bronchiectasis due to aspiration (5%). Therefore the underlying cause for the disease was found in 63% (19).

Bronchiectasis is a relatively common complication of lower respiratory tract infections (16,23). It is a major problem of children in developing countries (24). In Karadag et al. study, the mean age of the patients was 7.4 ± 3.7 years at presentation (17).

The underlying etiology of bronchiectasis is distinguishable in about 70% and is considered idiopathic in remaining (8,9,19). Regarding the initial etiologic factor and risk factors, the patients with bronchiectasis have variable outcomes (15.25).the present In study. the underlying etiology was determined in 77.4% including asthma (55.6%), GERD (7.8%), and CF (4.8%). Anomalies, anatomic disorders, congenital and brain disorders constituted less than 12% of etiologies. In the study of Lai et al. the previous lower respiratory tract infection, asthma, and primary immunodeficiency were the most common causes, with unknown etiology in 31% (26).

In Kim et al. study the underlying etiologies identified in 85.8% of patients, included bronchiolitis obliterans (32.6%), childhood respiratory infection (20.6%), interstitial lung disease (17.3%), immunodeficiency (8.6%), and primary ciliary dyskinesia (4.3%) (20). This diversity may be due to effects of geographical and racial differences in the occurrence of various disease.

The relation between asthma and bronchiectasis has been controversial with different prevalence of concurrent asthma and bronchiectasis (2.7% and 27%) even in a same region (27,28). In a report from Finland the prevalence of in patients hospitalized asthma for bronchiectasis was 19% (29). Possible which mechanisms by asthma and bronchiectasis predispose to each other include asthmatic obstruction contributing to development of bronchiectasis, sensitization and of airways with increased lability due to

microbial colonization of the ectatic bronchial tree (28). Misdiagnosis of asthma is common and the diagnostic process is further complicated by the fact that the co-existence of asthma is not uncommon (30). There is a delay from onset of bronchiectasis to diagnosis by HRCT scan.

Usually this may be due to the misdiagnosis of asthma in children with cough and no wheeze who had been labeled as having "cough variant asthma". It has been reported a potential inaccuracy of a diagnosis of asthma when based on the symptom of cough alone (8). In the present study, the most common known etiology was asthma in 208 (55.6%).

Cystic fibrosis (CF) is one of the most important causes, but a great variety of other causes makes non cystic fibrosis bronchiectasis a relatively frequent diagnosis (1). In Western countries, CF is the most common cause of bronchiectasis (20,31), and other causes include various respiratory infections such as pneumonia, pertussis, measles, and tuberculosis (20).

Bronchiectasis is one of the obvious manifestations in patients having CF years. Evaluation of children with for bronchiectasis has revealed the CF as a background cause in 3% to 6% (18). CF was underlying cause of bronchiectasis in 4.8%, which is compatible with the statistics indicating its prevalence as 3% to 6% (18). In the study of Karakoc et al. infection was the major cause of bronchiectasis. In patients. bronchiectasis 8 developed after tuberculosis or pneumonia, was associated with immune deficiency syndromes in 4 children, and with asthma in 4. Cystic fibrosis was diagnosed in 4 cases and ciliary dyskinesia in 3. In 10 patients, only one lobe was involved (16).

Non-CF bronchiectasis in childhood is still one of the most common causes of childhood morbidity in developing countries (17). Various factors have been identified as predisposing to the development of Non-CF bronchiectasis. Recurrent respiratory infections, immune deficiency, a retained foreign body, asthma, tuberculosis and primary ciliary dyskinesia are some of the more common risk factors (17).

Eastham et al. evaluated Non-cystic fibrosis (CF) bronchiectasis. Associations were previous pneumonic illness (30%),immunocompromised (21%), obliterative bronchiolitis (9%), congenital lung abnormality (5%), chronic aspiration (3%), eosinophilic esophagitis (2%), familial syndrome (2%),primary ciliary dyskinesia (1%), and right middle lobe syndrome (1%), while 18% were idiopathic (8). Banjar evaluated a total of 151 cases with Non-CF bronchiectasis of which. There is a period of 5 ± 3.2 years between the start of symptoms and the diagnosis of bronchiectasis. More than 2/3 of the patients had cough, tachypnea, wheezing, sputum production and failure to thrive. 60% had associated disease: Pulmonary diseases in 48(32%), immunodeficiency in 27(18%), CNS in 18(12%), cardiac in 12(8%) and asthma in 103(68%) of the patients. Sixty-eight (67%) were found to have sinusitis. Forty-nine (32%) developed GERD (32). In patients with Non-cystic fibrosis (Non-CF) bronchiectasis. immunodeficiency, aspiration and primary ciliary dyskinesia accounted for 67% of the cases. In 56% of children, the identification of a cause led to a specific change in management (21).

Bronchiectasis is currently nearly always diagnosed early by using High-resolution computed tomography (HRCT) scanning of the chest (8,14,15,31). So, the potential now exists for the much early detection and treatment of children with lesser degrees of bronchial dilation and bronchial wall thickening than was previously possible (8,15,31).

HRCT scanning is the most reliable noninvasive method for assessing the degree of bronchial wall dilatation, and thus bronchiectasis can be accurately diagnosed applying this technique (20,21). Characteristic findings of bronchiectasis on CT (33,34) include bronchial wall thickening with associated bronchial dilatation. When seen in cross- section, the internal diameter of the bronchus or bronchiole becomes larger than that of its accompanying pulmonary artery (a broncho arterial ratio greater than 1) resulting in the "signet ring" sign. The ectatic airway may be air-filled, fluidfilled, or have air-fluid levels (14,31,35). In the present study, CT scan was used diagnosis and for follow of up bronchiectasis, and showed one or more evidences of bronchiectasis in all cases including bronchial thickening, bronchial dilatation, or cystic images.

The immune system is less effective in young children and elderly adults which results in an increased incidence of infection in these two groups (36,37). Bronchiectasis has most commonly been described as commencing in childhood, particularly in the first five years of life, with chronic productive cough and unresolved infection (12,14,38). In the present study, the median age at the time of the diagnosis was 8.61±3.36 years. In Kim et al. study the median age at the time of the diagnosis of bronchiectasis was 7.6 years (range, 2 months to 18 years) (20).

In this study of 374 children with bronchiectasis, 240 (64.2%) were male. This

ratio is compatible with the study of Eastham et al. in which the male to female ratio was 2:1 (8). However, in the study of Banjar et al. in Saudi Arabia, 49.7% were male (32). Also, in the study of Lai et al. the male to female ratio was 1:1, and the mean was 12.1 years (range, 3.1 to 18.1 years) (21). Karakoc et al. evaluated 23 children with bronchiectasis (13 boys (57%) and 10 girls (43%), with a mean age of 8.45 ± 4.02 years) (16).

of The most common symptom bronchiectasis is productive cough with or without chest pain, hemoptysis, dyspnea, and failure to thrive (14,15,20). In the study of Banjar et al., more than two third had cough, tachypnea, and hemoptysis (32). These symptoms were common (41.4%) in another study (26). In our patients, the most common symptom was cough which was occurred in all cases and was productive in 79.1%. Other common symptoms were recurrent pneumonia, tachypnea, and dyspnea. Hemoptysis was seen in 5%. It is a common symptom in adults while is uncommon in children (23). The high frequency of hemoptysis in the study of Lai et al. in comparison with others may be due to the low number of patients with hemoptysis (n=31) enrolled by Lai et al. (26).

The common signs of bronchiectasis are wheezing (39), crackle, coarse pulmonary sounds, clubbing, chest deformity, and cyanosis (14,15). Compatible with these findings, the most common signs in our patients were crackle in 47.1% and wheezing in 25.4%.

Lai et al. reported wheezing and crackle as the most common findings (26). Clubbing has reported in 20.7% (26), and 3% to 51% (7,24,39,40). We found clubbing only in 1.1%. The advancement in the diagnostic and therapeutic methods has led to the early diagnosis and control of the disease (8,14,15,31). In developed countries, the incidence of bronchiectasis has decreased due to improvements in socioeconomic conditions and effective treatment of bacterial pneumonia (including the development of broad spectrum antibiotics) (20). This has decreased the of clubbing prevalence as symptom find prolonged bronchiectasis in (7,24,39,40). This issue can explain the lower rate of clubbing in our study series.

Kim al. investigated et the characteristics, epidemiological clinical features, underlying etiologic factors, and distinct change in the management of patients with bronchiectasis. The median age at the time of the diagnosis of bronchiectasis was 7.6 years (range, 2 months to 18 years). Persistent coughing was the most common symptom. The etiologies identified underlying in 79 patients (85.8%) included bronchiolitis obliterans (32.6%), childhood respiratory infection (20.6%), interstitial lung disease (17.3%), immunodeficiency (8.6%), and primary ciliary dyskinesia (4.3%). In 53 children (67%), the identified cause led to a distinct and individualized change in management. The distribution of CT abnormalities had no correlation with the underlying cause of bronchiectasis (20).

The life expectancy of patients with bronchiectasis has improved tremendously, as a result of advances in therapy. Before the development of surgical resection and antibiotic treatment, the mortality of bronchiectasis was as high as 49%, in a follow up of 3 to 6 years. Even those who survive, usually have poor quality of life and incapacitating symptoms (41). The treatment of childhood bronchiectasis is medical in initial (10,42). In the study of Karakoc et al. the initial treatment was primarily medical, but in 2 patients whose medical therapy failed, pulmonary resection was carried out. Three patients died from severe pulmonary infection and respiratory failure (16). The goals of treatment are: a) controlling symptoms; b) improving quality of life; c) preventing disease progression. The mainstays of treatment are antibiotics and physiotherapy. Antibiotics are used to treat acute exacerbations and as prophylaxis to reduce the number and severity of exacerbations (15). It is aimed at controlling infection and improving bronchial hygiene with use of mucolytic agents, inhaled bronchodilators and corticosteroids, long-acting 2 agonists and antibiotics (41,43). In the study of Eastham et al. the total resolution of the changes in six, improvement in one, progression in five, and was unchanged in six at a minimum interval of 18 months (8).

In our study, 35.6% had complete recovery, 40.9% had relative recovery, and 23.5% had not recovery. The presence of underlying disease, asthma, GERD, and CF were effective on the response to the therapy.

The patients in Kim et al. study were treated by chest physical therapy and postural drainage with bronchodilator therapy, which may assist in mobilizing endobronchial secretions.

Furthermore, short-term antibiotic therapy was performed to reduce the volume and bacterial density of sputum (20).

If an underlying cause can be determined, this should be treated vigorously. Immunological problems require the expert assessment of the pediatric immunologist. A regular joint respiratory immunology clinic can be the ideal forum to manage such complex patients (15).

Conclusions

Early recognition and institution of treatment and vaccination proper of anti- bacterial available and anti-viral vaccines are encouraged prevent to progression of the disease. In children presenting with chronic productive cough and crackles, considering bronchiectasis can be helpful in early diagnosis and providing complementary evaluation and treatment.

Treatment of the underlying disease can prevent bronchiectasis and is effective on early treatment, and improves its therapeutic response. According to the study results, it is recommended to consider additional evaluations in all patients complaining of chronic cough and sputum, to exclude bronchiectasis or earlier onset of treatment to prevent complications.

Conflict of interests: None

Acknowledgment

The authors are grateful to all children and staffs for their help and cooperation during the study period.

References

- Alvarez Caro F, Gómez Farpón A, Ruiz del Árbol Sánchez P, de Miguel Mallén MÁ, Alvarez Berciano F. Bronchiectasis in pediatrics, diagnosis approach and management. Arch Argent Pediatr. 2012;110(1):52-9.
- 2. Masekela R, Green RJ. The role of macrolides in childhood non-cystic fibrosis-related bronchiectasis. Mediators Inflamm 2012;2012:134605.
- 3. Habesoglu MA, Ugurlu AO, Eyuboglu FO. Clinical, radiologic, and functional evaluation of 304 patients with bronchiectasis. Ann Thorac Med 2011;6(3):131-6.
- 4. King PT, Holdsworth SR, Freezer NJ, Villanueva E, Holmes PW. Characterisation of the onset and presenting clinical features

of adult bronchiectasis. Respir Med 2006;100:2183-9.

- 5. Pasteur MC, Helliwell SM, Houghton SJ, Webb SC, Foweraker JE, Coulden RA, et al. An investigation into causative factors in patients with bronchiectasis. Am J Respir Crit Care Med 2000;162:1277-84.
- 6. Feldman C. Bronchiectasis: new approaches to diagnosis and management. Clinics in Chest Medicine 2011;32(3):535-46.
- 7. Dagli E. Non cystic fibrosis bronchiectasis. Paediatr Respir Rev 2000;1(1):64-70.
- 8. Eastham KM, Fall AJ, Mitchell L, Spencer DA. The need to redefine noncystic fibrosis bronchiectasis in childhood. Thorax 2004;59(4):324-7.
- 9. Twiss J, Metcalfe R, Edwards E, Byrnes C. New Zealand national incidence of bronchiectasis "too high" for a developed country. Arch Dis Child 2005;90(7):737-40.
- Sirmali M, Karasu S, Türüt H, Gezer S, Kaya S, Taştepe I, Karaoğlanoğlu N. Surgical management of bronchiectasis in childhood. Eur J Cardiothorac Surg 2007;31(1):120-3.
- 11. Barker AF. Bronchiectasis. N Engl J Med 2002;346:1383-93.
- 12. King PT, Holdsworth SR, Freezer NJ, Villanueva E, Holmes PW. Characterisation of the onset and presenting clinical features of adult bronchiectasis. Respir Med 2006;100:2183-89.
- 13. King PT, Holdsworth SR, Freezer NJ, Villanueva E, Gallagher M, Holmes PW. Outcome in Adult Bronchiectasis. COPD: Journal of Chronic Obstructive Pulmonary Diseases 2005;2:27-34.
- King PT. The pathophysiology of bronchiectasis. Int J Chron Obstruct Pulmon Dis 2009;4:411-9.
- 15. Fall A, Spencer D. Paediatric bronchiectasis in Europe: what now and where next? Paediatr Respir Rev 2006;7(4):268-74.

- 16. Karakoc GB, Yilmaz M, Altintas DU, Kendirli SG. Bronchiectasis: still a problem. Pediatr Pulmonol 2001;32(2):175-8.
- 17. Karadag B, Karakoc F, Ersu R, Kut A, Bakac S, Dagli E. Non-cystic-fibrosis bronchiectasis in children: a persisting problem in developing countries. Respiration 2005;72(3):233-8.
- Pasteur MC, Helliwell SM, Houghton SJ, Webb SC, Foweraker JE, Coulden RA, et al. An investigation into causative factors in patients with bronchiectasis. Am J Respir Crit Care Med 2000;162(4 Pt 1):1277-84.
- 19. Nikolaizik WH, Warner JO. Aetiology of chronic suppurative lung disease. Archives of Disease in Childhood 1994; 70: 141-2.
- 20. Kim HY, Kwon JW, Seo J, Song YH, Kim BJ, Yu J, et al. Bronchiectasis in children: 10-year experience at a single institution. Allergy Asthma Immunol Res 2011;3(1):39-45.
- 21. Li AM, Sonnappa S, Lex C, Wong E, Zacharasiewicz A, Bush A, et al. Non-CF bronchiectasis: does knowing the aetiology lead to changes in management? Eur Respir J 2005 ; 26(1):8-14.
- 22. Edwards EA, Metcalfe R, Milne DG, Thompson J, Byrnes CA. Retrospective review of children presenting with non cystic fibrosis bronchiectasis: HRCT features and clinical relationships. Pediatr Pulmonol 2003;36:87-93.
- Ferkol TW, Davis PB. Bronchiectasis and bronchiolitis obliterans. In: Taussig LM, Landau LI, Le Souef PN, Morgan WJ, Martinez FD, Sly PD editors. Pediatric respiratory Medicine 1999; St Louis, Mosby, 784-9.
- 24. Brown MA, Lemen RJ. Bronchiectasis. In: Chernick V, Boat TF, Kendig EL, ediors. Kendig's disorders of respiratory tract in children. 6th ed. Philadelphia: Saunders;1998. 538-52.
- 25. Baker A, Bardana E. Bronchiectasis: update of on orphan disease. Am Rev Respir Dis 1988; 137: 969-76.
- 26. Lai SH, Wong KS, Liao SL. Clinical analysis of bronchiectasis in Taiwanese children. Chang Gung Med J 2004;27(2):122-8.

- 27. Pang J, Chan HS, Sung JY. Prevalence of asthma, atopy, and bronchial hyperreactivity in bronchiectasis: a controlled study. Thorax 1989; 44, 948-51.
- Ip MS, So SY, Lam WK, Yam L, Liong E. High prevalence of asthma in patients with bronchiectasis in Hong Kong. Eur Respir J 1992;5(4):418-23.
- 29. Säynäjäkangas O, Keistinen T, Tuuponen T, Kivelä SL. Bronchiectasis in Finland: trends in hospital treatment. Respir Med 1997;91(7):395-8.
- 30. Chang AB, Redding GJ, Everard ML. Chronic wet cough: Protracted bronchitis, chronic suppurative lung disease and bronchiectasis. Pediatr Pulmonol 2008;43(6):519-31.
- 31. Kothari NA, Kramer SS. Bronchial diseases and lung aeration in children. J Thorac Imaging 2001;16(4):207-23.
- 32. Banjar HH. Clinical profile of Saudi children with bronchiectasis. Indian J Pediatr 2007;74(2):149-52.
- 33. Coleman LT, Kramer SS, Markowitz RI, et al. Bronchiectasis in children. J Thorac Imag 1995; 10: 268-79.
- Kang E, Miller RR, Muller NL. Bronchiectasis: comparison of preoperative thin-section CT and pathologic findings in resected specimens. Radiology 1995; 195: 649-54.
- 35. McGuinness G, Naidich DP. CT of airways disease and bronchiectasis. Radiol Clin North Am 2002;40:1-19.
- 36. English BK, Schroeder HW, Wilson CB. Immaturity of the fetal and neonatal immune system. In: Rich RR, Fleisher TA, Shearer WT, Kotzin BL, Schroeder HW, editors. Clinical Immunology, Principles and Practice. London, UK: Mosby; 2001. pp. 40.10-40.41.
- 37. Weksler ME, Szabo P. Aging and the immune system. In: Rich RR, Fleicher TA, Shearer WT, Kotzin BL, Schroeder HW, editors. Clinical Immunology; Principles and Practice. London, UK: Mosby; 2001. pp. 41.41-48.
- 38. Pasteur MC, Helliwell SM, Houghton SJ, et al. An investigation into causative

factors in patients with bronchiectasis. Am J Respir Crit Care Med 2000;162:1277-84.

- 39. Dagli E. Non cystic fibrosis bronchiectasis. Paediatr Respir Rev 2000; 1: 64-70.
- 40. Lewinston NJ. Bronchiectasis. In: Hilman BC, editor. Pediatric respiratory disease: diagnosis and treatment . Philadelphia: Saunders:1993.p. 222.
- 41. Al-Shirawi N, Al-Jahdali HH, Al-Shimemeri A. Pathogenesis, etiology and treatment of bronchiectasis. Ann Thorac Med 2006; 1(1), 41-51.
- 42. Caylak H, Genc O, Kavakli K, Gurkok S, Gozubuyuk A, Yucel O, et al. Surgical management of bronchiectasis: a collective review of 339 patients with long-term follow-up. Thorac Cardiovasc Surg 2011;59(8):479-83.
- 43. Redding GJ. Update on treatment of childhood bronchiectasis unrelated to cystic-fibrosis. Paediatr Respir Rev 2011;12(2):119-23.