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Original Article

Prospective Study of Bronchial Asthma

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Abstract

Bronchial asthma is an atopic disease characterized by chronic airway inflammation and hyper-responsiveness. Severe acute asthma is a medical emergency and sometimes difficult to treat. This prospective study was done at Dhaka Medical College Hospital from January 1997 to January 1998. Total 30 patients of bronchial asthma were included in this study. Diagnosis was established on the basis of symptoms, evidence of airflow obstruction and its reversibility by bronchodilator therapy. The age range was 18 to 80 years with a mean 36.64±4.91. Of them, 63% were male and 37% were female. It revealed that all patients had classical triad of dyspnoea, wheeze and cough. Almost all patients (80%) had some precipitating agents for their attack. Regarding treatment of severe acute asthma - Nebulized salbutamol is superior to conventional intravenous aminophylline, as p value of nebulized salbutamol group is <0.001 which is significant. So, severe acute asthma should be managed with nebulized salbutamol instead of intravenous aminophylline.

Key words: Bronchial asthma, Bronchodilator, Nebulized salbutamol, Aminophylline.

Introduction :

Bronchial asthma is characterized by chronic airway inflammation and increased airway hyper-responsiveness leading to symptoms of wheeze, cough and dyspnoea. Prevalence of asthma is increasing steadily over the late part of the last century in countries with a western life style and is also increasing in developing countries[1]. Asthma affects 7% of the population and 300 million worldwide[2]. Public attention in the developed world has recently focused on asthma because of its rapidly increasing prevalence, affecting up to one in four urban children[3]. The association between atopy and asthma suggests that sensitization of exposure to allergen is an important risk factor. Many agents are responsible for triggering asthma attack such as house dust, mite, pollen and cold etc. Multiple environmental as well as genetic determinants are implicated. Hygiene hypothesis proposes that decreased infections in early life bias the immune system towards an allergic phenotype[1]. Asthma is caused by environmental and genetic factors[4], which can influence how severe asthma is and how well it responds to medication[5]. Some environmental and genetic factors have been confirmed by further research, while others have not been. Dietary intake may be important. Milk fat and anti-oxidants may protect against the development of asthma in children. But in another study early exposure to cow's milk protein has been linked to the development of atopy and asthma. Asthmatic inflammation appears to be coordinated principally by activated CD4+T-lymphocyte of the Th_2 type phenotype characterized principally be the production of the cytokines interleukin IL-4, IL-13 and IL-5[4]. The cytokine IL-5 acts on eosinophil while IL-4 and IL-13 up regulate adhesion molecules in the capillary endothelium of the bronchial mucosa resulting in increased adhesion of eosinophils to the endothelium.

Severe acute asthma is a life threatening medical emergency. In many areas of our country patients are treated by intravenous aminophylline but recent modalities of treatment - nebulization with salbutamol and ipratropium bromide are more effective. There are still more than 1,300 deaths reported each year in UK. Severe disease, inappropriate medical treatment and adverse behavioral and psychological factors have all been implicated in fatal attack of asthma. Airway inflammation and acute bronchial spasm can be induced by a variety of triggers in susceptible individual. Respiratory tract inflammation particularly viral are the most common cause of acute attack of asthma but other triggers include allergens, air pollutants, exercise, foods, drugs, emotion, Gastro Esophageal Reflux Disease (GERD), menstruation and pregnancy[6].

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Materials and methods:

Total 30 patients of bronchial asthma aged 18 years and above admitted into Dhaka Medical College Hospital (DMCH) in calendar year 1997-1998 were included in this study. Diagnosis was established by lung function tests and reversibility test by bronchodilator (inhaled salbutamol). Detailed history, physical findings and investigations were recorded as per protocol. Twenty patients of severe acute asthma having Peak Expiratory Flow Rate (PEFR) between 0-150 L/min were included in the study. Among them 10 patients were treated with intravenous (IV) aminophylline in bolus (6 mg/kg body weight over 15-20 min) followed by maintenance dose of 0.5 -0.8 mg/kg/hr and 10 patients with nebulized salbutamol 5 mg every 6 hrs. PEFR measured 8 hourly for 3 days and recorded.

Observations and results:

The age range was 18 to 80 years with mean age is 36.64±4.91. (Table I). Among them 19 (63%) were male and 11(36%) were female with a male: female ratio 1.7:1 (Figure I). Total 12 (40%) patients were smoker and 18 (60%) were non-smoker (Figure II). Out of 30 patients, 22 (74%) had positive family history of asthma (Figure III).

Table I: Distribution of patients according to age

Age in years	Number of Patients (%) n =30
18 -29	8 (26.7)
30 -49	13 (43.3)
>49	9 (30)

Fig I: Distribution of patients according to sex

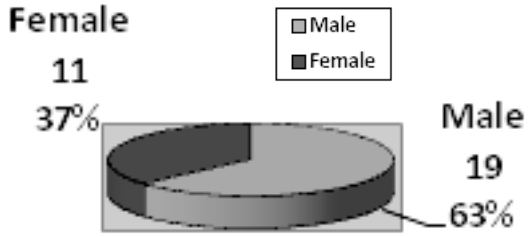


Fig II: Distribution of patients according to smoking habit

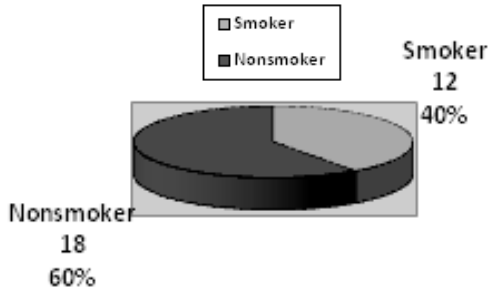
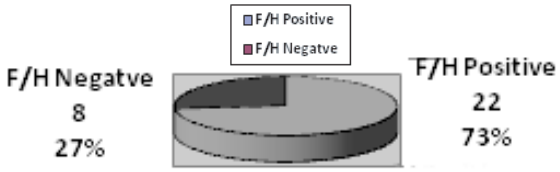


Fig III: Distribution of patients according to positive family history



Almost all patients had classical triad of dyspnoea, wheeze and cough. Expectoration of scanty mucoid sputum was recorded in 60% cases. Chest tightness or discomfort was noted in 33% cases. Nocturnal or early morning exacerbations of symptoms were noted in 76% cases. Seasonal variation present in 25 (78%) cases, 66% patient's asthma worse in winter and 16% patient's in summer. Fever was present in 26% cases. Pulsus paradoxus, cyanosis was present in 33% cases. Heart rate >100/min and respiratory rate >30/min was found in all the cases and all patients were unable to talk in one sentence. Total 33% patient had associated allergic rhinitis, 10% had eczema and 10% had both the conditions. Blood examination showed eosinophilia in 13% cases and chest radiology showed pneumothorax in 6% cases. Sputum culture and sensitivity negative in all the cases but sputum eosinophilia was present in 33% cases. Thirty six percent patients had episodic disease, 43% chronic and 12% had mixed type of asthma (Table II)

Table II: Distribution of patients according to clinical presentation

Characteristics	Number (Percentage)
Symptoms:	
Dyspnoea	30 (100)
Wheeze	30 (100)
Cough	23 (76)
Sputum	18 (60)
Chest discomfort	10 (33)
Nocturnal symptoms	23 (76)
Seasonal variation	25 (78)
Worse in winter	20 (66)
Worse in summer	05 (12)
Signs:	
Respiratory rate >30/min	30 (100)
Heart rate>100 b/min	30 (100)
Inability to talk in a sentence	30 (100)
Pulsus paradoxus	10 (33)
Cyanosis	10 (33)
Use of accessory muscles of respiration	30 (100)
Fever	11 (36)
Pneumothorax	02 (6)
Associated allergy - Rhinitis	10 (33)
Eczema	03 (10)
Both	03 (10)
Investigations:	
Blood eosinophilia	04 (13)
Chest X -ray pneumothorax	02 (06)
Sputum eosinophil	10 (33)
Sputum C/S	Negative
Clinical type:	
Episodic	11 (36)
Chronic	13 (43)
Mixed	04 (13)
1st attack	2 (6)
Episodic (18-30 yrs)	6 (55)
(31-49 yrs)	4 (36)
Chronic (18 -30 yrs)	1 (7)
(31-49 yrs)	7 (53)
(Above 49 yrs)	5 (38)

Almost all patients (80%) had some precipitating factor for their asthma. 25(83%) house dust, 23(76%) cold air, 6(20%) pollen, 7(23%) fumes, 6(20%) food, 1(3%) exercise and no external allergen in 6(20%) (Table III).

Table III: Precipitating factors of asthma

Allergen	Number (Percentage)
House dust	25 (83)
Cold air	23 (76)
Fumes	07 (23)
Pollen	06 (20)
Food (Beef, Egg, Prawn, Hilsa fish)	06 (20)
Exercise	01 (3)
None	06 (20)

Table IV shows nebulized salbutamol is clearly superior to IV aminophylline in severe acute asthma with a p value <0.001 and <0.02 respectively.

Table IV: Comparative study on nebulized salbutamol versus intravenous aminophylline in severe acute asthma.

No.of patients	Nebulized salbutamol group PEFR L/m			Intravenous aminophylline group PEFR L/m		
	Pre - treatment	Post treatment	p value	Pre - treatment	Post treatment	p value
1	10	188.44	<0.001	120	208	<0.02
2	50	288.44		60	174.44	
3	150	311.11		80	133.55	
4	30	175.55		70	103.88	
5	50	235		100	150	
6	80	250		60	130	
7	110	323.33		50	111.11	
8	30	186.66		60	148.33	
9	90	252.22		70	112.44	
10	60	244.44		70	118.88	

Discussion:

In this study, most of the patients were in 3rd to 4th decades of life. Bronchial asthma occurs at all ages but predominantly in early life[7]. This discrepancy may be due to the age range of our patients (18-80 yrs) where children were not included. Male: Female ratio is 1.7:1. Male to female ratio in childhood asthma is 2:1 which equalizes by age 30 years[8]. This apparent numerical superiority of the males could be due increased attendance of male patients to have treatment from hospital. It also reflects the general tendency of our women to refrain from or inability to seek medical help.

Almost all patients presented with dyspnoea, wheeze and cough which indeed constituted the clinical hallmark of disease. Expectoration of sputum was present in 60% of patients. Cough and sputum during an attack of asthma don't necessarily indicate respiratory infection rather these result from inflammation in the bronchial wall. Nocturnal and early morning aggravation of symptoms was present in about 73% patients. This also correlates with the finding that morning dipping is a characteristic feature for asthma[9]. About 10 (33%) patients complained of tightness or discomfort in the chest. The character of this pain was not indicative of any definite structural involvement. The discomfort might be produced by fatigue of the respiratory muscles due to excessive work. The incidence of fever on admission was 36%. An underlying bacterial infection could not be substantiated in most of them as sputum C/S was negative in all the cases. This correlates with the fact that viruses not the bacteria precipitate asthmatic attacks[7]. A precipitating factor was recognized in more than half of the patients. Most are incriminated dust (83%), cold air (76%), Pollen (20%). In one study Barrio et al found that 12 out 21 or 57% patients had some precipitating factors for their attack[8]. Our study also correlates with this study.

Regarding clinical types 36% had episodic disease 43% had chronic disease and 13% mixed type. Episodic asthma was more common among young adult and chronic and mixed asthma in older age group. This is in keeping with the fact that younger patients tend to have extrinsic asthma with intermittent symptoms and older people have intrinsic asthma with perennial symptoms[4]. In this series 50% suffered from other allergic conditions such as Rhinitis (33%), Eczema (10%) and both (10%). Total 74% have a family history of allergic diathesis. Bronchial asthma is the most common allergic disease which runs in family. This also coincides with the findings reported by Mullick et al in their series of 100 cases (age range from 0 to 60 years and above) where bronchial asthma was the most allergic condition in the family, followed by rhinitis, eczema and urticaria in diminishing order[9]. Most of the hospital admission for asthma occurred in winter and it is proved that respiratory tract infections are also common in winter which precipitates asthma. Cold air is also a common trigger factor. Blood examination showed that 13% had eosinophilia which may indicate the proportion of attacks have an allergic basis. Estimation of IgE level and skin prick test would be more specific for detection of allergic cases.

In this study it was found that p value of nebulized salbutamol group was <0.001 which indicates that the result is highly significant. On the contrary p value of intravenous aminophylline group was <0.02 which indicates that the result is less significant. This shows that nebulized salbutamol is the first line drug for the treatment of severe acute asthma. This correlates with guidelines of British Thoracic Society for the management of severe acute asthma where they advocate nebulized salbutamol along with corticosteroid as the treatment of choice[10].

Conclusion:

Many precipitating factors are responsible for triggering the attack. In this study we found that dust, cold air, pollen and food are mostly responsible. No new allergen is detected. Most of the patient was presented with the complaints of dyspnoea, wheeze, cough and chest pain. Clinical types are of chronic, episodic and mixed variety. Episodic type associated with allergic rhinitis and Eczema. Severe acute asthma is a life threatening form of bronchial asthma. This can be best managed by Nebulized salbutamol, as nebulized salbutamol is the drug of choice in the treatment of severe acute asthma.

References

1. Innes JA, Reid PT. Respiratory Disease. In: Boon NA, Colledge NR, Walker BR, Hunter JAA, editors. Davidson's principles and practice of Medicine, 20th ed, Elsevier; 2006. p.670-78.

2. Christopher HF. Asthma. N Engl J Med. 2009; 360:1002-14.

3. Lilly CM. Diversity of asthma: evolving concepts of pathophysiology and lessons from genetics. J. Allergy Clin. Immunol. 1998;115(Suppl 4):526-31.

4. Martinez FD. Genes, environments, development and asthma: a reappraisal. Eur Respir J. 1998; 29(1):179-84.

5. Choudhry S, Seibold MA, Borrell LN. Dissecting complex diseases in complex populations: asthma in latino americans. Proc Am Thorac Soc. 1994; 4(3):226-33.

6. Szentivanyi A, Ali K, Calderon EG, Brooks SM, Coffey RG, Lockey RF. The in vitro effect of Imunnoglobulin E (IgE) on cyclic AMP concentrations in A549 human pulmonary epithelial cells with or without beta adrenergic stimulation. J. Allergy Clin Immunol. 1993; 91:379.

7. Mc.Fadden JER. Asthma. In: Brounwald E, Isselbachere KJ, Wilson JD, editiors. Harrison's principles of internal medicine, 13th ed. New York: Mc.Graw Book Company;1994. p.1047-52.

8. Barriot P, Rion B. Prevention of fatal asthma. Chest 1987; 92:460-6.

9. Mullick RNB, Mullick SB. Heredity in bronchial asthma. Journal of Indian Med. Assoc.1987; 85:44-7.

10. Harrison B. Acute severe asthma in adults. Medicine International Bangladesh edition 1995; 9(31):298-301.