Analysis of Clinical Profile of Childhood Bronchial Asthma in the Asthma Clinic of a Tertiary Care Medical College Hospital

Menon Narayanankutty Sunilkumar[1], Vadakut Krishnan Parvathy[2]

ABSTRACT

Introduction: Asthma is a chronic inflammatory disease of airways that is characterised by increased responsiveness of the tracheobronchial tree to a multiplicity of stimuli. Prevention plays a pivotal role in reducing its morbidity and mortality. This study was conducted to understand the various risk factors and its spectrum in children with asthma.

Materials and methods: A prospective analysis of children attending the Asthma clinic of a tertiary care medical college hospital. A detailed history was taken using the proforma having special reference to age, sex, age of onset, symptomatology and triggering factors.

Results: Among the 100 children with asthma in the study 4% were less than 1 year of age and 32% in 4-6 years. 35% were female children. Parental asthma was seen in 70% of the children. 69% had severe lower respiratory tract infection below 2 years. Among the total cases, 36% had environmental tobacco exposure and 11% were low birth weight. The triggers found were infection (54%), allergy (47%), exercise-induced asthma (34%) and 9% had both infection and allergy. The mild intermittent, mild persistent and moderate persistent asthma were 22, 17 and 61%, respectively. None of them had severe persistent asthma. 42% were on inhaled steroids. Regular follow up was seen in 56% of patients with 26% having good compliance.

Conclusion: A good compliance with knowledge of asthma, regularly visiting asthma clinic, control of environmental triggers and co-morbid conditions, seeking early medical care and early treatment can reduce morbidity and mortality up to significant level.

Key words: Asthma, Lower respiratory tract infection, Allergy, Atopy, Steroids, Tracheobronchial

INTRODUCTION

Asthma is a chronic inflammatory disease of airways that is characterised by increased responsiveness of the tracheobronchial tree to a multiplicity of stimuli. Childhood bronchial asthma (CBA) is the most common chronic disease in the industrialized nations and its prevalence is increasing throughout the world. There is a worldwide variation in the prevalence of asthma. The largest study group which has conducted a study on the prevalence of CBA by International Study of Asthma and Allergies in Childhood.[1] The reported prevalence of CBA according to the study was about 25%. It varies from region to region in the world as seen in subsequent studies.[2]

Various environmental changes play a major role in the CBA epidemic.[3] The causes are multifactorial and in a country like ours with a vast diversity and increasing incidence of CBA the study on this major health issue in children is worthwhile. There is no permanent cure of asthma. Hence, prevention plays a pivotal role in reducing its morbidity and mortality. So a thorough analysis and sincere approach to the various issues in children with asthma can alleviate their symptoms to a greater extent. This study was conducted in the Asthma clinic of the Department of Paediatrics with an aim to understand the various risk factors and its spectrum in these children.

MATERIALS AND METHODS

A prospective analysis of children attending the Asthma clinic of Department of Paediatrics, Amala Institute of Medical Sciences, Thrissur, Kerala, India were included in the study. A detailed analysis was conducted with the proforma having special reference to age, sex, age of onset, symptomatology and trigger factors. Detailed examination and relevant investigations and was categorized and managed. Children with congenital heart diseases and structural lung diseases were excluded from the study. A written consent was obtained from the subjects or from their relatives. The study design was approved by Institutional Ethics Research Committee.

RESULTS

Total of 100 children (35 males and 65 females) were included in the study (Figure 1). Among the subjects of age ranges from 7 months to 18 years, 4% were less than 1 year of age, 30% were in the age group 1-3 years, 32% in 4-6 years, 23% in 7-10 years and 11% above 10 years group (Table 1). Of the various risk factors, parental asthma was seen in 70% of the children; 69% had severe lower respiratory tract infection less than 2 years; 36% had environmental tobacco exposure and 11% were low birth weight (Table 2). The trigger was infection (54%) and allergy (47%), 9% had both infection and allergy (Table 3). 34% had exercise induced asthma. Among the various allergens acting as triggers, cotton bed and pillow used were the most common (63%) (Figure 2).

The first attack of asthma was at less than 1 year of age in

[1]Assistant Proffessor, [2]Professor and HOD, Department of Pediatrics, Amala Institute of Medical Sciences, Amala Nagar, Thrissur - 680 555, Kerala, India

Corresponding author: Menon Narayanankutty Sunilkumar, Assistant Professor, Department of Pediatrics, Amala Institute of Medical Sciences, Amala Nagar, Thrissur - 680 555, Kerala, India

How to cite this article: Menon Narayanankutty Unilkumar, Vadakut Krishnan Parvathy. Analysis of clinical profile of childhood bronchial asthma in the asthma clinic of a tertiary care medical college hospital. International Journal of Contemporary Medical Research 2016;3(2):489-492.

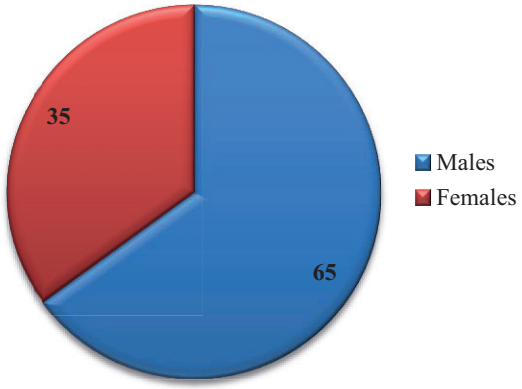

Figure-1: Distribution of sex of the study subjects

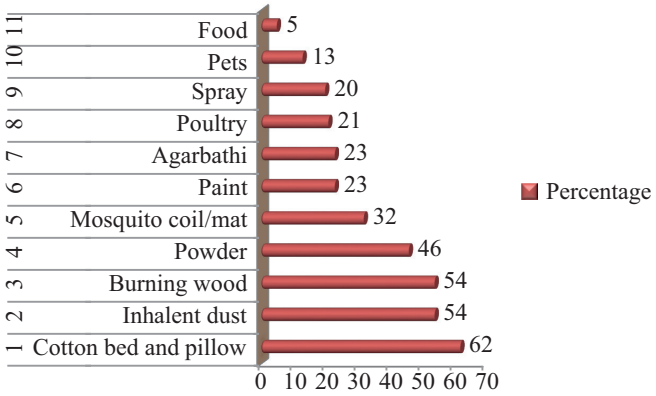

Figure-2: Distribution of various allergens

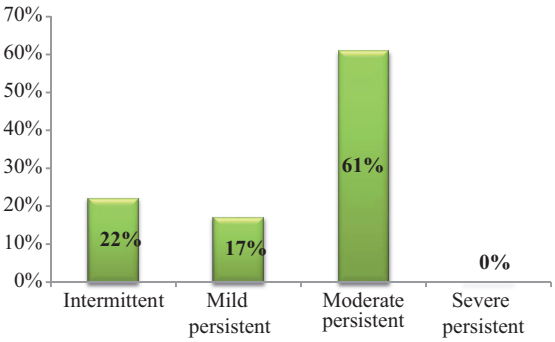

Figure-3: Distribution of asthma severity

52% of patients. 22% had mild intermittent asthma, 17% had mild persistent and 61% moderate persistent asthma (Figure 3). None of them had severe persistent asthma. 42% were on inhaled steroids. Regular follow up was seen in 56% of patients. Good compliance was seen only in 26% of the study population.

DISCUSSION

Asthma is a leading cause of chronic illness in childhood and most often starts early in life and has variable courses and unstable phenotypes which may progress or remit over time.[1,2] CBA is strongly associated with alleregy, especially in developed countries.[1-3] The gradual increase in the incidence of CBA over the past few years and the geographic variation in its prevalence rates and the magnitude is directly linked to the environmental changes that play a major role in

Age group	Percentage
Less than 1 year	4
1 to 3 years	30
4 to 6 years	32
7 to 10 years	23
More than 10 years	11

Table-1 Distribution of age

Sl. No	Risk factor	Percentage
1.	Parental asthma	70
2.	Lower respiratory infections	69
3.	Tobacco exposure	36
4.	Low birth weight	11
5.	Atopy	43
6.	Allergic rhinitis	13

Table-2: Risk factors for asthma

Sl No	Trigger	Percentage
1	Infection	54
2	Allergens	47
3	Mixed	9
4	Exercise	34
5	Drugs	0

Table-3: Various triggers for asthma

the CBA epidemic.[4] Many national and international studies have also thrown light into the epidemiology of CBA.[5]

Wheeze in preschool children may result from a number of different conditions around half of preschool wheezers become asymptomatic by school age irrespective of treatment. Abnormal lung function has been documented by Lowe et al.[6] in their study in preschool children with persistent wheezing as young as age 3 years. Martinez et al.[7] identified the spectrum of CBA in the first six years of life.

Among the 100 children with asthma in the study 4% were less than 1 year of age, 30% were in the age group 1-3 years, 32% in 4-6 years, 23% in 7-10 years and 11% above 10 years group. About 34% children were in the preschool age group. Martinez et al.[7] identified that evenlthough some 50% of preschool children in their cohort have wheezing, only 10%–15% have a diagnosis of "true" CBA by the time they attain the school age.

Sex affects the development of CBA as the growth of the child progresses. The incidence and prevalence of CBA are greater among boys than among girls until the age of 13–14 years. Studies through puberty have shown a greater incidence of CBA among adolescent and young adult female and a greater proportion of males children with remission of CBA.[8,9] Nicolai et al.[9] have attributed these changes in prevalence and severity to events of puberty. In the present study, 35% were female children and there was a male predominance as is seen in other studies.[8,9]

The spectrum of risk factors in the causation of CBA is many. New-onset CBA can occur at any age, without any prior illness or disease process. There are many risk factors for CBA such as allergic sensitization, decreased lung function in infancy, family size and structure, socio-economic status,

antibiotics and infections, and sex and gender. In our study we analysed the risk factors such as parental asthma in these children, severe lower respiratory tract infection less than 2 years, environmental tobacco exposure, low birth weight, atopy and allergic rhinitis.

Parental asthma especially maternal asthma has been postulated as a risk factor for the development of CBA.[10] Wickens et al.[11] in their study in New Zealand observed even paternal asthma as a risk factor of CBA. Mrazek et al.[12] predicted the development of early-onset asthma in genetically at-risk children and in low birth weight babies. The repeated lower respiratory tract infection may affect the paediatric population who are already at risk for CBA due to family history or atopy. Children born to mothers with allergy or asthma have a relatively persistent maturational defect in Th1 (T helper 1) cytokine synthesis in the first year of life, which may play a role in the development of persistent or severe viral infections. Friedlander et al.[13] postulated that severe viral infection of the lower respiratory tract in genetically susceptible infants who are already sensitized to inhalant allergens may lead to deviation toward Th2 (T helper 2) responses promoting asthma.

Environmental triggers may affect asthma differently at different times of a child's life. Exposure to environmental tobacco smoke also consistently worsens asthma symptoms and is a risk factor for severe asthma[3] Atopy as a risk factor for asthma is less common with increasing age but occasionally it is the dominant trigger and allergic rhinitis is a concomitant risk factor.[1-5,14] The majority of children ie 34% with persistent wheezing (in whom CBA was diagnosed) experienced their first symptoms before age 3. Of the various risk factors, parental asthma was seen in 70% of the children; 69% had severe lower respiratory tract infection less than 2 years; 36% had environmental tobacco exposure and 11% were born of low birth weight.

The triggers of an asthma could be an infection as manifested by the child developing a fever, signs of upper respiratory tract infection such as a running nose cough and develops wheeze. Various studies highlight the point that there is ongoing interactions of genes with environmental exposures (including allergens, air pollution, environmental tobacco smoke and diet) which modulate the host response to infections. A controversy exists whether the occurrence or timing of childhood infection is pathogenic or protective for the development and long-term outcome of CBA and allergy and of nonallergic wheeze phenotypes.[15,16] Exercise induced CBA is known and is an important entity not to be missed.[1,2,5-7,16] Specific drug treatments (e.g., β-blockers, nonsteroidal anti-inflammatory drugs) or, in women, the use of hormone replacement therapy, occupational exposure to sensitizing agents or irritants can cause adult asthma.[17] The trigger was infection in 54% and allergy in 47% of children, 9% had both infection and allergy as triggers. 34% had exercise induced asthma. None of the children were on any drugs to cause CBA.

In childhood, airway hyperresponsiveness is more common and more severe among males due to the exposure of various allergens.[17,18] Among the various allergens included in the proforma use of cotton bed and pillow, exposure to inhalent dust, burning wood at home and exposure to the smoke, powder, exposure to mosquito coil/mat being used as a repellant, agarbathi, paint, poultry,spray, pets and food as a cause for CBA were asked in detail.

Among the allergens cotton bed and pillow were the most common (seen in 63%) allergen in the study leading onto an asthma attack. The exposures to these allergens are a major cause of CBA and rapid efforts to minimise the contact with these allergens can alleviate the symptoms of CBA.[17-19]

The children were grouped according to the severity of CBA as per the standard guidelines on CBA and managed accordingly.[20] The first attack of asthma was at less than 1 year of age in 52% of patients in the study. Accordingly based on the severity of CBA, 22% had mild intermittent asthma, 17% had mild persistent and 61% moderate persistent asthma. None of them had severe persistent asthma. 42% of the children were on inhaled steroids. A regular follow up was seen in 56% of patients. A good compliance was seen only in 26% of these children.

ACKNOWLEDGEMENT

The authors acknowledge the help of Dr Ajith TA, Professor, Biochemistry, Amala Institute of Medical Sciences, Amala Nagar, Thrissur, Kerala, India during the preparation of the manuscript.

REFERENCES

1. International Study of Asthma and Allergies in Childhood (ISAAC) Steering Comxmittee. Worldwide variation in prevalence of symptoms of asthma, allergic rhinoconjunctivitis, and atopic eczema: ISAAC. Lancet. 1998;351:1225–32.
2. Asher MI, Montefort S, Bjorksten B, Lai CK, Strachan DP, Weiland SK, et al. ISAAC Phase Three Study Group. Worldwide time trends in the prevalence of symptoms of asthma, allergic rhinoconjunctivitis, and eczema in childhood: ISAAC Phases One and Three repeat multicountry cross-sectional surveys. Lancet. 2006;368:733–43.
3. Lødrup Carlsen KC. The Environment and Childhood Asthma (ECA) Study in Oslo: ECA-1 and ECA-2. Pediatr Allergy Immunol. 2002;13(Suppl 15):29–31.
4. Eder W, Ege MJ, von Mutius E. The asthma epidemic. N Engl J ed. 2006;355:2226–35.
5. Johnston NW, Sears MR. Asthma exacerbations. Epidemiology. Thorax. 2006;61:722–8.
6. Lowe LA, Simpson A, Woodcock A, Morris J, Murray CS, Custovic A. Wheeze phenotypes and lung function in preschool children.Am J Respir Crit Care Med. 2005;171:231–7.
7. Martinez FD, Wright AL, Taussig LM, Holberg CJ, Halonen M, Morgan WJ. Asthma and wheezing in the first six years of life. The Group Health Medical Associates. N Engl J Med. 1995;332:133–8.
8. de Marco R, Locatelli F, Sunyer J, Burney P. Differences in incidence of reported asthma related to age in men and women. A retrospective analysis of the data of the

European Respiratory Health Survey. Am J Respir Crit Care Med. 2000;162:68–74.
9. Nicolai T, Illi S, Tenborg J, Kiess W, v Mutius E. Puberty and prognosis of asthma and bronchial hyper-reactivity. Pediatr Allergy Immunol. 2001;12:142–8
10. Wright AL, Holberg CJ, Taussig LM, Martinez F. Maternal asthma status alters relation of infant feeding to asthma in childhood. Adv Exp Med Biol. 2000;478:131–7.
11. Wickens K, Crane J, Kemp T, Lewis S, D'Souza W, Sawyer G, et al. A case-control study of risk factors for asthma in New Zealand children. Aust N Z J Public Health. 2001;25:44-9.
12. Mrazek DA, Klinnert M, Mrazek PJ, Brower A, McCormick D, Rubin B, et al. Prediction of early-onset asthma in genetically at-risk children. Pediatr Pulmonol. 1999;27:85–94.
13. Friedlander SL, Jackson DJ, Gangnon RE, Evans MD, Li Z, Roberg KA, et al. Viral infections, cytokine dysregulation and the origins of childhood asthma and allergic diseases. Pediatr Infect Dis J. 2005;24(Suppl):S170–6.
14. Jaakkola MS, Ieromnimon A, Jaakkola JJ. Are atopy and specific IgE to mites and molds important for adult asthma? J Allergy Clin Immunol. 2006;117:642–8.
15. Lemanske RF, Jr, Busse WW. Asthma: factors underlying inception, exacerbation, and disease progression. J Allergy Clin Immunol. 2006;117 (Suppl Mini-Primer):S456–61.
16. Subbarao P, Becker A, Brook JR, Daley D, Mandhane PJ, Miller GE, et al. CHILD Study Investigators. Epidemiology of asthma: risk factors for development. Expert Rev Clin Immunol. 2009;5:77–95.
17. Kogevinas M, Zock JP, Jarvis D, Kromhout H, Lillienberg L, Plana E, et al. Exposure to substances in the workplace and new-onset asthma: an international prospective population-based study (ECRHS-II) Lancet. 2007;370:336–41.
18. Takkouche B, Gonzalez-Barcala FJ, Etminan M, Fitzgerald M. Exposure to furry pets and the risk of asthma and allergic rhinitis: a meta-analysis. Allergy. 2008;63:857–64.
19. Arruda LK, Sole D, Baena-Cagnani CE, Naspitz CK. Risk factors for asthma and atopy. Curr Opin Allergy Clin Immunol. 2005;5:153–9.
20. Hollenbach JP, Cloutier MM. Childhood Asthma Management and Environmental Triggers.Pediatr Clin North Am. 2015;62:1199-214.

Source of Support: Nil; **Conflict of Interest:** None

Submitted: 29-12-2015; **Published online**: 19-01-2016