

Clinical characteristics of childhood asthma

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Summary

A prospective study was undertaken of 298 asthmatic children attending a paediatric allergy clinic in Bloemfontein. A detailed history was obtained, and skin tests were performed. Male predominance and an early age of onset were confirmed. Symptomatic allergic rhinitis was an extremely common finding. A family history of allergy was obtained in over 90% of cases. Respiratory tract infections, changes in the weather and exercise were found to be the most common asthma-inducing factors. Common allergens were grass pollen (63%) and animal danders. House-dust mite (25%) and dietary allergens (less than 20%) were relatively uncommon. The failure to recognise the symptoms of asthma in children, particularly young children, was identified as an ongoing problem.

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Asthma is a common condition among children, causing widespread, and sometimes severe, chronic or recurrent morbidity. Treatment based on the use of effective medication, aided by the avoidance of trigger factors and, occasionally, desensitisation, is straightforward and effective. The vast majority of childhood asthmatics can lead a normal, virtually symptom-free life until such time as their asthma improves spontaneously in their mid-teens.¹

One of the major stumbling blocks in achieving this ideal is the underdiagnosis and misdiagnosis of asthma in children. The current concept of asthma is much broader than in the past. Recent workers²⁻⁶ are of the opinion that doctors, particularly those in general practice, often fail to recognise all but the most obvious cases.

A study was undertaken to highlight the clinical characteristics of childhood asthma and to examine some of the possible reasons for the underdiagnosis of this condition, and consequent failure to treat it successfully.

Patients and methods

Two hundred and ninety-eight asthmatic children attending the Paediatric Allergy Clinic at Universitas Hospital, Bloemfontein, were studied prospectively. The diagnosis of asthma was based on the presence of a history of recurrent wheeziness, supported by one or more of the following findings: a positive family history of atopy or asthma; the presence of positive skin-prick or laboratory tests for allergy; the demonstration of reversible airways obstruction; and a positive therapeutic response to anti-asthma medication.

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The clinical data were recorded on a standardised questionnaire subsequent to the patient's initial visit to the clinic. The age and sex of the patient was noted, as well as the age at which the child first experienced a wheezing episode.

The occurrence of other atopic symptoms (eczema; hayfever; angio-oedema; urticaria; reactions to food; bee-sting; drugs; etc.) was noted, as well as the presence or absence of a family history of asthma or any of these other conditions in first- and second-degree relatives.

Specific trigger factors of asthma enquired about included: upper airway infections (defined as fever with rhinorrhoea, pharyngitis, tonsillitis or sinusitis); exercise; weather changes; allergens (environmental, animal and food); and irritants such as paint, perfume and aerosol sprays.

The severity of the patient's asthma during the preceding 12 months was graded according to the following categories: grade I — outgrowing the condition — usually an older child showing a definite decrease in the frequency and severity of attacks; grade II — mild — one attack every 1 - 2 months, promptly relieved by inhalation of β_2 -adrenergic stimulants; grade III — moderate — significant attacks once or twice a month, or more frequent mild wheezing, responding well to daily medication with β_2 -adrenergic stimulants and/or sodium cromoglycate; grade IV — severe — significant attacks at least twice a week, occasionally requiring hospitalisation, and usually requiring inhaled corticosteroids in addition to other medication; grade V — only with exercise; and grade VI — only with infection.

Height and weight were measured, and the child was examined for rhinitis, eczema, chest deformity, and wheeze. Skin-prick testing with 0.5% phenol saline (negative control), 1% histamine (positive control) and several common allergens was performed on 189 patients. Patients under 6 years of age were excluded from skin testing. The following allergenic extracts (Bencard) were used: pollens of Bermuda grass, maize, mixed South African grasses, privet, acacia, mixed South African shrubs and mixed South African trees, dog hair, cat hair, feathers, *Dermatophagoides pteronyssimus* (house-dust mite), dietary wheat, milk, egg, fish, orange and chocolate. Reactions were measured at 20 minutes. Any weal greater than that of the negative control was taken as a positive reaction.³

Results

Of the 298 children studied, 197 were boys (66.1%). The ratio of boys to girls was 2:1. The median age at onset of wheezing was 3 years with a range of 1 month - 11 years 4 months. The age distribution is shown in Fig. 1.

The ages of the children at the time of presentation ranged from 5 months to 14 years 11 months (median 7 years 1 month).

In addition to their asthma, 229 children (76.8%) complained of troublesome symptoms of allergic rhinitis, and 51 (17.1%) had suffered from eczema.

A family history of allergy in first- and/or second-degree relatives was positive in 272 cases (91%).

All patients' parents reported that upper respiratory infections could lead to wheeziness. The two other trigger factors mentioned most frequently were exercise and changes in the weather. Specific allergens were troublesome to approximately half of the patients (Fig. 2).

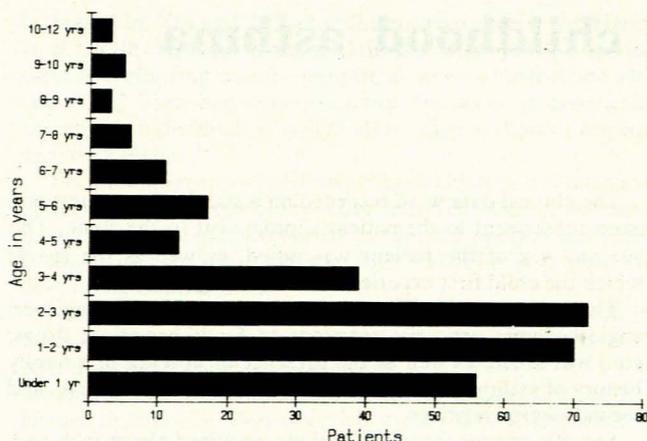


Fig. 1. Age of onset.

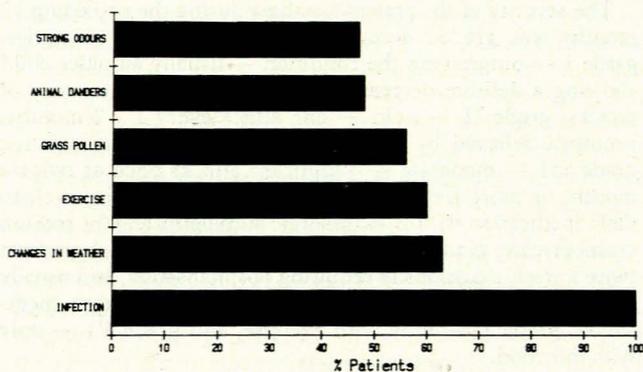


Fig. 2. Precipitating factors.

The numbers of patients falling into each of the 6 grades describing severity of their asthma are shown in Table I.

Grade	No. of children	%
I	32	11
II	81	27
III	124	41
IV	47	16
V	9	3
VI	5	2
Total	298	100

Of the 189 patients who were skin tested a total of 165 (87,3%) showed a positive skin prick test to one or more allergens (Table II). A total of 126 patients (69%) gave a positive response to grass or maize pollen, while only 63 patients (31%) showed no response to either of these.

Discussion

The findings in this study are contrary to the often expressed opinion that allergy is an affliction found mainly in the coastal

TABLE II. RESULTS OF SKIN-PRICK TEST IN 189 ASTHMATIC CHILDREN

Allergens	No. of patients with a positive skin test	%
Bermuda grass	103	56,3
Cat hair	101	55,2
Mixed SA grasses	96	52,5
Maize pollen	78	42,6
Dog dander	65	35,5
Mixed SA shrubs	58	31,7
House-dust	59	32,2
<i>D. pteronyssimus</i>	47	25,7
Acacia	44	24,0
Privet	40	21,9
Mixed SA trees	39	21,3
Feathers	39	21,3
Dietary wheat	38	20,8
Milk	35	19,1
Fish	33	18,0
Oranges	32	17,5
Chocolate	29	15,9
Egg	24	13,1

areas of South Africa. Asthma is common in children from the Orange Free State, although it does differ in certain respects from asthma at the coast. An exceptionally high proportion of our patients (76,8%) suffered from symptoms of allergic rhinitis and required additional medication for this. This figure is at variance with the 34 - 63% found in studies from England^{3,7} and Kuwait.⁸ In contrast, a relatively small number of our patients (17,1%) had ever experienced eczema. The same English studies and one from South Africa⁹ have quoted figures of between 35% and 57%. Eczema also has been reported to be uncommon in studies of asthmatics in Kuwait⁸ and northern Nigeria.¹⁰ These differences may be of racial origin or related to climatic and other environmental factors.

The warm dry climate of South Africa's inland plateau supports the existence of vast tracts of natural grassland and cultivated cereals, all belonging to the grass (Gramineae) family. Pollination of the various species occurs over a period of at least 8 months — spring, summer and autumn.¹¹ This explains the high incidence of grass-pollen hypersensitivity found in this study. It also may account for the frequent occurrence of allergic rhinitis among the children studied.

On the other hand, house-dust mite hypersensitivity (25% positive) is relatively uncommon in and around Bloemfontein. This is unlike the situation in the Cape Peninsula, a coastal area of South Africa, where house-dust mite accounts for 65% of positive skin tests and grass pollens for only 7%.¹² House-dust mites thrive in conditions of high relative humidity,¹³ and many of the children in the present study who had a positive skin test to house-dust mite were found to have lived at the coast for a period of time.

Another group of allergens commonly giving rise to positive skin prick tests in this study were animal danders. Fifty-five per cent of children tested were positive to cat, 36% to dog and 21% to feathers. Findings vary considerably in other studies, perhaps reflecting the extent to which the population groups involved keep household pets.^{3,8}

Dietary allergens gave positive skin test results in 13 - 20% of subjects tested, supporting the hypothesis that inhaled allergens trigger asthma far more readily than ingested allergens. Conversely, dietary restrictions seldom are able to produce significant improvement in asthma.

The 2:1 male predominance found in this series is similar to the findings in many epidemiological studies of asthma in children.^{1,3,8}

The proportion of children in the group studied with onset of symptoms when under the age of 3 years was very high (67%). This confirms the findings of Williams and McNicol¹⁴ and of Loftus and Price,³ who all concluded that the more severe forms of childhood asthma begin at an early age. The converse is not always true, however, and the age of onset of symptoms cannot be used prognostically in isolated cases.¹⁵ What is clear is that true asthma can and does develop before the age of 2 years. Reluctance to diagnose asthma in a wheezy baby would appear to be misplaced.

Another significant finding in this study is the average time span of 4 years 1 month (range 1 month - 13 years 4 months) between the age at onset of wheezing and the age at presentation of these children at the Allergy Clinic. A general practice audit carried out by Levy and Bell⁶ found that an average of 16 - 20 consultations for respiratory problems were needed before the diagnosis of asthma was made. The reasons for this failure to recognise asthma need to be elucidated.

Fifty-seven per cent of the children in the group studied were regarded as having moderate-to-severe asthma requiring medication with two or more drugs. This correlates with the early age of onset. It should be noted, however, that the occurrence of such a high proportion of moderate-to-severe cases is not reflective of the situation in childhood asthma as a whole. Only children with obvious symptomatic asthma, often difficult to control, tend to be referred to specialist clinics.

The great majority of the children studied (91%) gave a family history of some form of atopic disease among first- and second-degree relatives. This is a high figure compared with the 40 - 57% quoted in other studies.^{3,7,16} The explanation may be that the population studied was largely drawn from Afrikaner stock with its own high incidence of inherited disorders.¹⁷ In the context of this study, the absence of a family history of allergy possibly may be regarded as a useful negative indicator in the diagnosis of equivocal cases of asthma.

Virtually all the children studied had experienced asthma associated with upper airways infection. The strong association between viral respiratory tract infection and asthma has been noted in several studies. Carlsen *et al.*¹⁸ found that rhinovirus and respiratory syncytial virus were most commonly implicated. The seasonal variation in asthma attacks was thought to correlate with the upsurge of viral respiratory tract infections during these months. Attacks may be triggered by virus-induced increases in bronchial hyperreactivity.³

Exercise-induced asthma was reported in 60% of cases, confirming the importance of this condition in children. Other

non-allergic phenomena that triggered asthma in a significant number of children (63%) were changes in the local weather conditions. In Kuwait⁸ 72% of patients complained that windy, dusty weather and 63% that increased humidity triggered their asthma. Rain and dust also were implicated in northern Nigeria.¹⁰

Exposure to specific allergens (pollen, dust, foods, animal danders) precipitated asthma attacks in half the patients studied. It has been shown by Cockcroft *et al.*¹⁹ that inhalation of allergens causes a sustained increase in bronchial hyper-reactivity in virtually all asthmatic children, even while not necessarily triggering attacks. They conclude that exposure to allergens makes asthma worse, and that allergens should be avoided wherever possible.

In conclusion, general practitioners attending to paediatric patients need to be aware that asthma should be diagnosed in any child who presents with recurrent wheeze. Asthma may occur at a very early age. Early diagnosis will enable such children to receive appropriate treatment for their asthma.

REFERENCES

1. Rackemann FM, Edwards MC. Asthma in children. *N Engl J Med* 1952; **246**: 815-823.
2. Konig P. Asthma: pediatric pulmonary disease and a changing concept. *Pediatr Pulmonol* 1987; **3**: 264-275.
3. Loftus GB, Price JF. Characteristics of pre-school asthma. *Clin Allergy* 1986; **16**: 251-257.
4. Drosti LM. Asthma: lung-function testing. *Hosp Med* 1988; **10**: 18-20.
5. Speight ANP, Lee DW, Hey EN. Underdiagnosis and undertreatment of asthma in childhood. *Br Med J* 1983; **286**: 1253-1256.
6. Levy M, Bell L. General practice audit of asthma in childhood. *Br Med J* 1984; **289**: 1115-1116.
7. Heese HdeV. On asthma in children. *S Afr Med J* 1961; **35**: 229-232.
8. Ellul-Micallef R, Al-Ali S. The spectrum of bronchial asthma in Kuwait. *Clin Allergy* 1984; **14**: 509-517.
9. Ordman D. Allergy in childhood: its pattern, control and significance in adult prophylaxis. *S Afr Med J* 1958; **32**: 377-380.
10. Abdurrahman MB, Taqi AM. Childhood bronchial asthma in northern Nigeria. *Clin Allergy* 1982; **12**: 379-384.
11. Ordman D. Pollinosis in South Africa. *S Afr Med J* 1947; **21**: 38-48.
12. Van Niekerk CH, Shore SC, Weinberg EG. The housedust mite and childhood asthma in the Cape Peninsula. *S Afr Med J* 1977; **52**: 74-75.
13. Ordman D. The incidence of 'climate asthma' in South Africa: its relation to the distribution of mites. *S Afr Med J* 1971; **45**: 739-743.
14. Williams J, McNicol KN. Prevalence, natural history and relationship of wheezy bronchitis and asthma in children - an epidemiological study. *Br Med J* 1969; **4**: 321-325.
15. Blair H. Natural history of childhood asthma. *Arch Dis Child* 1977; **52**: 613-619.
16. Walt F. Asthma in childhood. *S Afr Med J* 1955; **29**: 340-344.
17. Rosendorff J, Bernstein R, Macdougall L, Jenkins J. Fanconi anemia: another disease of unusually high prevalence in the Afrikaans population of South Africa. *Am J Med Genet* 1987; **27**: 793-797.
18. Carlsen KH, Orstavik I, Leegard J, Hoeg H. Respiratory virus infections and aeroallergens in acute bronchial asthma. *Arch Dis Child* 1984; **59**: 310-315.
19. Cockcroft DW, Ruffin RE, Dolovich J, Hargreave FE. Allergen induced increase in non-allergic bronchial hyperreactivity. *Clin Allergy* 1977; **7**: 503-513.