Bronchial asthma in Nigerian children

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SUMMARY Clinical and laboratory studies on bronchial asthma in 200 Nigerian children who were seen during a 2½-year period in Ibadan are described. Contrary to reports that the condition is rare in African children, after pulmonary tuberculosis, asthma is the next most common chronic chest disease in Ibadan. While many features of the disease are similar to those seen elsewhere, skin tests indicate that ascarsis is the most common antigen associated with asthma, followed by Dermatophagoides pteronyssinus.

Bronchial asthma, a common childhood chronic respiratory problem in the West (Godfrey, 1974), is reported to be rare among African children (Wesley et al., 1969; Godfrey, 1975; Warrell et al., 1975). While studies by Mitchell (1970), Sofowora (1970), Anim and Edoo (1972), and Buchanan and Jones (1972) showed that the condition is common in African adults, Wesley et al. (1969) reported that bronchial asthma is extremely rare among Bantu children in South Africa. In Nigeria, Warrell et al. (1975) reported that asthma is rarely seen in children living in the savanna region of the country. Our experience at this hospital in Ibadan during the last few years indicates that the condition is far more common in children here than these reports suggest. This paper gives the results of studies on 200 asthmatic children seen at this hospital during a 2½-year period (June 1974 to December 1976).

Materials and methods

The patients in this study were referred to the chest clinic mainly from the general outpatient department of the hospital but some came from private clinics and hospitals in Ibadan and nearby. Diagnosis of asthma was based on a history of at least three attacks of breathlessness and wheezing (Blair, 1969), often associated with cough, and usually supported at the time of initial examination or during subsequent follow-up, by the auscultatory finding of widespread respiratory rhonchi. In some patients who presented in acute attacks, the diagnosis was further supported by the demonstration of at least 20% increase in the peak flow (measured with a Wright's peak flow meter) after the administration of a bronchodilator.

Laboratory investigations carried out on the patients included packed cell volume, white blood count—total and differential—stool microscopical examination, chest x-rays, and in a few children, the radiology of the paranasal sinuses. Skin sensitivity tests were done by the prick method, using a selected range of commercially prepared allergen extracts (Bencard). The weal diameter of the reaction recorded was the difference in mm between it and the control. Weals of at least 2 mm in diameter were regarded as positive (Pearson, 1973).

The children were classified into three groups according to the severity of the disease. This grading system was modified from one described by Dawson et al. (1969). The severe group consisted of patients with more than 10 acute attacks a year with or without complete clinical recovery between attacks. The moderate group had 5 to 10 attacks a year and usually showed complete or partial freedom from wheezing between attacks, while the mild group had 1 to 5 attacks a year with complete clinical recovery between attacks.

Results

Age at presentation and sex incidence. The children were aged between 10 months and 13 years; 97 (49%) of them were less than 5 years. The boy/girl ratio was 1.6:1.

Age of onset. As shown in the Figure, 125 (63%) children had their first attacks before age 3 years.

Referral diagnoses. Diagnosis of asthma or asthmatic bronchitis was made before referral in 136 (68%)
cases, while 18 (9%) were referred as cases of pulmonary tuberculosis, 15 as cases of bronchopneumonia, and 5 (3%) were thought to be cases of acute bronchitis or bronchiolitis. Other referral diagnoses included chronic bronchitis, chronic chest infection, and lobar pneumonia. In 15 cases, no diagnosis had been entertained at the time of referral.

**Early feeding.** 166 (98%) of 170 children whose early feeding histories were reliably ascertained were breast fed from birth. This is similar to the rate of 99% in 385 Ibadan women from all social classes who breast fed (Omololu, 1972). 145 (85%) of the same 170 children were fed on artificial milk, generally in addition to the breast milk; in 55% of them this was from the first week of birth. This proportion of those fed on artificial milk contrasts with 78% (range 45% in illiterate women to 99% in ‘elites’) of the women in Omololu’s series who fed their children partially or wholly on artificial milk.

**Family and social histories.** Table 1 shows that there were 127 (63·5%) patients who came from the upper socioeconomic classes (classes I, II, and III). This compares with an estimated 20% of these three classes attending hospital for other respiratory complaints. There was a positive family history of allergy in 79 (40%) of the 200 patients. 73 of these were siblings or parents suffering from asthma, but 5 parents had allergic rhinitis, and one sibling suffered from eczema. 78 (39%) of the 200 children were attending school, and of these, 14 were often absent from school because of asthmatic attacks. 74 (38·9%) of 190 families had domestic animals or pets—such as chickens, dogs, cats, or goats.

**Precipitating and associated factors.** There was a positive history of precipitating factors in 155 (78%) of cases (Table 2), leaving 45 cases where there was none.

**Presenting symptoms.** The predominant symptom was breathlessness during attacks noted in all 200 children. Cough and wheezing were the next most common, both occurring in 96% of children. Other common and frequent symptoms included sweating (most profuse on the forehead during acute attacks), anorexia and loss of weight, which occurred while the attack lasted and abated soon after it ceased. Symptoms of chest pains and headaches were given by patients older than 5 years. Sputum, mixed with

<table>
<thead>
<tr>
<th>Social class</th>
<th>Profession</th>
<th>No. of cases</th>
<th>% of total</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Academic professionals, senior administrators, owners of large business</td>
<td>40</td>
<td>20</td>
</tr>
<tr>
<td>II</td>
<td>Nonacademic professionals</td>
<td>25</td>
<td>12·5</td>
</tr>
<tr>
<td>III</td>
<td>Clerical workers, skilled workers, and artisans</td>
<td>62</td>
<td>31</td>
</tr>
<tr>
<td>IV</td>
<td>Semiskilled workers</td>
<td>40</td>
<td>20</td>
</tr>
<tr>
<td>V</td>
<td>Unskilled workers and petty traders</td>
<td>32</td>
<td>16</td>
</tr>
<tr>
<td>VII</td>
<td>Students</td>
<td>1</td>
<td>0·5</td>
</tr>
</tbody>
</table>

**Table 2 Major precipitating and associated factors in the history of 200 cases of bronchial asthma**

<table>
<thead>
<tr>
<th>Factor</th>
<th>No. of cases</th>
<th>% of total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Night-time</td>
<td>87</td>
<td>43·5</td>
</tr>
<tr>
<td>Exercise</td>
<td>60</td>
<td>30</td>
</tr>
<tr>
<td>Rainy season</td>
<td>47</td>
<td>23·5</td>
</tr>
<tr>
<td>Cold season</td>
<td>18</td>
<td>9</td>
</tr>
<tr>
<td>Specific foods</td>
<td>16</td>
<td>8</td>
</tr>
<tr>
<td>Fever</td>
<td>16</td>
<td>8</td>
</tr>
<tr>
<td>Cold bath</td>
<td>15</td>
<td>7·5</td>
</tr>
<tr>
<td>Cold water drink</td>
<td>10</td>
<td>5</td>
</tr>
<tr>
<td>Excitement</td>
<td>9</td>
<td>4·5</td>
</tr>
<tr>
<td>Fan</td>
<td>9</td>
<td>4·5</td>
</tr>
<tr>
<td>Dry season</td>
<td>7</td>
<td>3·5</td>
</tr>
<tr>
<td>Dust</td>
<td>5</td>
<td>2·5</td>
</tr>
</tbody>
</table>

Figure  Age of onset in 200 children with asthma.
streaks of blood (often slight and infrequent), was present in 8 (4%) patients.

Physical signs

Physical growth

Weight and height were recorded in 169 patients at the first visit to the clinic, and weight alone was recorded in 28 others. The figures were plotted on centile charts for British children (Tanner et al., 1966). According to Janes (1970), the weights and heights of élite or privileged Nigerian children of both sexes compare favourably with those of British children, hence the use of British centile charts. 63 (32%) of the 197 children had weights <10th centile, while 151 (77%) were <50th centile. The corresponding figures for heights were 40 (24%) of 169 <10th centile and 109 (65%) <50th centile.

Respiratory signs

No abnormal auscultatory signs were present in 102 (51%) of the 200 cases at the initial visit. There was audible wheeze in 30 of the remaining 98 patients, and the other children had abnormal signs only on auscultation. These abnormal auscultatory signs consisted of inspiratory and expiratory rhonchi in 59 children, expiratory rhonchi alone in 23, inspiratory rhonchi alone in 8, and combined rhonchi and crepitations in the remaining 8 patients. There was a barrel-shape chest deformity in 19 (10%) of the 200 patients.

Other signs

10 children (5%) had flexural eczema which had been present from infancy in 2 of them and was quiescent in 7 others; vernal conjunctivitis was present in 19 (9.5%) patients.

Severity of asthma

58 (29%) of the 200 patients were classified as having severe asthma, while 30 (15%) had a moderate type, and in 112 (56%) of them it was mild.

Investigations

Skin sensitivity tests. Skin tests were performed on 139 children using between 10 and 30 allergen extracts. The results were negative in 46 (33%) of the children. Table 3 shows the 10 most common allergens to which the remaining 93 patients were sensitive. Ascariis antigen topped the list of positives, followed by *Dermatophagoides pteronyssinus* and house dust.

Chest x-rays. The findings in 190 cases are shown in Table 4. 99 (52%) of the 190 had normal films or a slight linear increase in lung markings. Only 4 (2%) showed evidence of hyperinflation (most of the films were not taken during acute attacks). It is noteworthy that 11% of the patients had increased lung markings and increased hilar shadows mimicking hilar lymphadenopathy.

Paranasal sinus x-rays. These were done in 32 patients, 22 (69%) of whom had normal sinususes while 8 (25%) showed thickening of the mucosa; clouding of the sinuses was evident in only 2 children.

Blood eosinophilia and stool parasites. The mean eosinophil count in 168 children was 1166/mm³ (1.1 × 10⁹/l) (range 0–5562/mm³; 0–5.5 × 10⁹/l), compared with a mean count of 449/mm³ (0.4–10⁹/l) (range 0–1425; 0–1.4 × 10⁹/l) in a group of 50 control children. In all, 67 (40%) of the 168 had eosinophil counts >1000/mm³ (1.0–10⁹/l). In relation to the total white blood count, the mean percentage was 13% (range 0–42%), compared with a mean of 5% (0–23%) for the controls. 87 (52%) of the 168 had percentages >11%.

Stool specimens from the children were examined at the same time that the eosinophil counts were done. Helminths, mainly the ova of ascaris and trichuris, were present in the stools of 120 of these patients. The eosinophil count (mean 1362/mm³;
1.3 x 10^6/l) in asthmatic children with stool parasites was higher than the counts in those without parasites (mean 787/mm^3; 0.7 x 10^6/l). However, in a group of 35 controls with stool parasites, the eosinophil count (mean, 510/mm^3; 0.5 x 10^6/l) was lower still, suggesting that both the atopic state and helminthiasis contributed to the high eosinophil count in the patients with asthma.

**Tuberculin skin test.** These were carried out in 183 of the children. 102 (57%) of these had negative tuberculin reactions while 75 (41%) and 6 (3%) had grades 1 and 2 respectively. In none of the children was there any other evidence of tuberculosis.

**Haemoglobin electrophoresis.** 179 children had genotype determinations. Of these, 140 had the normal A genotype while 39 (22%) had abnormal genotypes (AS, 31; AC, 6; SC, 1; SS, 1). This proportion of abnormal Hb electrophoretic pattern is similar to that in the general population.

**Discussion**

Most of the features of asthma in the present series are similar to those described in children elsewhere. The male preponderance is in keeping with the findings of Dawson et al. (1969), and Davis (1976). 63% of our patients had their first attacks of wheezing before age 3 years, similar to the findings of Wesley et al. (1969), but higher than the 44% reported by Dawson et al. (1969) and the 32% reported by Williams and McNicol (1969). Other similarities with the situation in the UK or Australia are found in the proportion of patients with a family history of allergy and in the common precipitating factors. Contrasting with these similarities, there are certain dissimilarities, notably the finding that the ascaris antigen was the most common allergen to which the skins of the children were sensitive. This sensitivity bore no consistent relationship with the presence of ascaris ova in the stool. Although the full significance of this finding is not clear, none of the 35 controls who has so far undergone skin tests with the ascaris antigen has had a positive reaction. Another dissimilarity is the proportion of children showing negative skin reactions to a battery of between 10 and 30 antigens. In the present series, 33% of the children tested did not react to any antigen, including *D. pteronyssinus* and grass pollen. This is in contrast to the 20% for asthmatic children in Melbourne (Williams and McNicol, 1969) and the 7% quoted by Goofre (1974) in the UK.

The incidence of asthma in European children has been estimated as 0.4% in Helsinki (Eriksson-Lihr, 1955), 4.8% in Aberdeen (Dawson et al., 1969), and 9.9% in Birmingham (Morrison Smith et al., 1971). In Africa, a school survey in Nigeria showed a rate of 2.4% (Sofowora and Clarke, 1969), and Carswell et al. (1977) reported a rate of 7.8% in a group of Tanzanian children. These estimates, and those of the present series, suggest that bronchial asthma is as common a condition in Ibadan children as it is in European or Australian children. Yet elsewhere in Africa the condition is reported to be rare. In Kenya, the youngest patient reported by Wasunna (1968) in a series of 210 cases was 12. Mitchell (1970) also from Kenya reported that no child under 12 was seen from over 1200 asthmatic patients who attended the Kenyatta National Hospital, Nairobi, and Rees et al. (1974), reporting on 124 asthmatic cases from the same hospital, recorded only 18 patients under 21 years. In South Africa, Wesley et al. (1969), comparing the incidence of asthma in Durban children of three racial groups, concluded that bronchial asthma was extremely rare among Bantus. Warrell et al. (1975) reported that only one child was admitted with severe asthma during a period of 4 years in Zaria, Northern Nigeria, an area where the paediatric outpatient attenders number approximately 96000 children each year. It thus appears that while the prevalence of asthma among adults is similar in many parts of Africa, there are wide variations in its prevalence in childhood. This variation may be attributed to the fact that the children in different areas of Africa are not exposed to adverse environmental precipitating factors—such as artificial feeding, atmospheric pollution, and other allergens—in the same way and at the same ages. Another possible explanation may be the difficulty in differentiating bronchial asthma from other forms of chronic respiratory disorders affecting children in Africa.

The fact that the diagnosis of bronchial asthma in environments such as ours depends mainly on its clinical features (provocation tests cannot easily be carried out), makes it inevitable that mistakes in diagnosis are common. Bronchial asthma can easily be confused with pulmonary tuberculosis, a common condition in African children, with the cardinal symptoms of cough, fever, anorexia, loss of weight, and night sweats, which also occur with bronchial asthma. (Even haemoptysis was present in 4% of the cases in this series of asthma.) 9% in the present series of asthmatic children were diagnosed as pulmonary tuberculosis before referral, and 2 of these were treated with antituberculous drugs for a prolonged period before the correct diagnosis was made. To avoid misdiagnosis, as great an emphasis should be placed on the history of recurrent attacks of breathlessness as on wheezing, for it is much easier to obtain a history of breathlessness than of wheezing in Africa where there are often no
vernacular equivalents of the word, and where to elicit such symptoms the examiner must be able to mimic the wheezing sound.

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References


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Dr R. H. Dobbs, formerly Professor of Paediatrics in Zaria, Northern Nigeria, was invited to comment on this article.

This paper illustrates the difficulties in assessing the many environmental factors—social, ethnic, and climatic—that interact to bring out clinical manifestations in atopic children.

Climate is undoubtedly crucial and it is made clear, both in the text and in Table 2 of this paper, that in Ibadan, less than 100 miles from the south coast, the rainy season is an important precipitating factor of asthmatic attacks, whereas in Zaria, 100 miles from the northern frontier, where the dry season often extends throughout 9 or 10 months of the year, childhood asthma scarcely exists, and those few cases that are encountered there, are in the neighbouring densely urban town of Kaduna, and confined to the few rainy months.

Social class appears also to have much bearing on the incidence of childhood asthma and its delayed appearance until early adolescence. As recorded, in Ibadan 64% of patients were drawn from the relatively wealthy classes I to III, in contrast to only 20% attending with other respiratory illnesses. The almost universal reliance on breast feeding, and the late onset of cows’ milk supplements in the poor may be an important factor in this delay, although the data are not sufficiently clear to draw conclusions.

It may be added that it is now thought that the protective factors in breast milk are more important than any immunologically deleterious action of cows’ milk. But perhaps of even greater importance is the enormously high infant mortality rate, which may well lead to the elimination of a large proportion of the undernourished, immunologically deficient, and atopic infant population of the poorer classes, with the rich paying for their Darwinian survival with the development of childhood allergy.

The high frequency of positive skin tests to ascaris is of great interest, being in conformity with previous studies. This does not necessarily mean that ascaris antigen is responsible for triggering the asthmatic state. Indeed it has been suggested that, on the contrary, because of high levels of IgE antibody to
ascaris, most cell-binding sites may be blocked for antibody to other antigens, and thereby protect against asthma. This concept might seem to be countered by the author’s statement that ‘None of the 35 controls . . . has shown positive reactions (to ascaris)’, an observation that is hard to understand in view of the worm infestation of African children in general.

A final point relating to the incidence of asthma in developing countries, although not discussed in this paper, is worth consideration—the rarity of asthma in young children of some localities and social classes is in contrast with its development as adolescence approaches. This is illustrated by the need to set up asthma clinics in Northern Nigeria; these are found to deal almost entirely with adult asthmatics. It seems that environmental factors can, with the passage of time, induce atopic disorders.