

Original articles

Deaths from asthma in New Zealand

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SUMMARY We report the first complete population based study of childhood deaths due to asthma. All deaths ascribed to asthma in New Zealand children aged 0–14 were investigated as part of a two year national study of mortality from asthma. The 16 children who died from asthma all developed asthma by the age of 4; 15 had a family history of asthma, and 12 had associated atopic disorders. Disturbed psychosocial relationships were evident in eight families. Seven children died in less than three hours from the onset of their final attack. All children died outside hospital. Mortality from asthma in Maori children (3.14 per 100 000) was five times that of European children. With hindsight, factors which if avoided could have led to a different outcome were identified in eleven cases. The circumstances surrounding these deaths were similar to those described for adults with asthma; this study, however, underlines the importance of parental care and knowledge in the management of children with asthma. Inadequate long term medical care, underassessment of severity by family and doctors, failure of the family to call for help when required, and inadequate responses of medical services contributed to the fatalities. Excess beta₂ sympathomimetic dosage or overreliance on home nebulisers were uncommon. Most childhood deaths from asthma should be prevented by increased family awareness, better assessment of severity, improved long term treatment, and rapid access to emergency medical care.

Asthma is a rare cause of childhood mortality. However, an increase in mortality in young New Zealand asthmatics noted first in 1977 and continuing over the next five years caused concern.¹ The increase brought the New Zealand mortality rate for childhood asthma to over three times that of the United Kingdom.² Interaction between newly available sustained release theophylline and inhaled beta agonist was suggested as a possible reason for the rising mortality.³ Few series of childhood asthma deaths have been reported, and, apart from one community based study of 30 deaths over a period of 20 years,⁴ all have been hospital based populations.^{5–8}

This paper reviews all deaths from asthma in children below the age of 15 identified during a two year national study of mortality from asthma in New Zealand.⁹

Methods

Deaths in all age groups, certified as due to or related to asthma, between 1 August 1981 and 31 July 1983 inclusive in people usually resident in New Zealand were investigated.⁹ The criterion for inclusion in the study was the use of the words 'asthma', 'asthmatic', or 'asthmaticus' in Part I of the death certificate or on a coroner's report. Cases were identified by monthly regional searches of death certificates and a quarterly search of all certificates at the office of the Registrar General of Births, Deaths, and Marriages. Within four months of death an appropriate family member, the general practitioner, and any specialists consulted were interviewed and hospital records reviewed.

Ethnicity was taken as that stated by the relative interviewed. Data on the ethnicity and age distribu-

tion of the population were obtained from the 1981 New Zealand census data.¹⁰

Questionnaires, interviewers' comments, hospital records, and autopsy data where available were reviewed by two physicians, who assessed the characteristics of the asthma, the quality of care received during the year before death, and, where applicable, the care sought and received in the last episode. They sought to ascertain whether, bearing in mind all the information available, a fatal outcome could have been avoided.

Results

Over the two years 329 deaths were verified as due to asthma;⁹ 17 of these were of children aged under 15.

Certification of cause of death. In seven children the general practitioner certified death as due to asthma, and in 10 the coroner reported death as due to asthma. Autopsies were performed on 11 children. One institutionalised child certified as dying of pneumonia, methaemoglobinaemia, and asthma was found on investigation not to have had asthma at the time of death; the word 'asthma' more properly belonged in Part II of the death certificate as an accompanying condition not contributing directly to death, and this child was excluded from study. No cases came to knowledge that were not detected by search of death certificates, and no other children were coded by the National Health Statistics Centre as dying of asthma during the study. One of the 17 children who died from asthma also suffered from cystic fibrosis, and is excluded from the remainder of this report.

Demographic data. New Zealand children belong to three major ethnic groups. Eight European (three girls), one Pacific Island Polynesian boy, and seven Maori (indigenous Polynesian) children (five girls) died of asthma during the two years; the annual mortality rates for these racial groups were 0.59, 1.36, and 3.14 per 100 000, respectively. The deaths in younger age groups were predominantly of Maoris, including all the children aged under 10 who died. The mean age at death of Maori children was 8.6 years (range 2.1–14.8 years) and of European children 12.0 years (range 10.3–14.7 years). Eight of the 16 deaths occurred in the central area of New Zealand's North Island, which has a concentration of Maori people greater than the national average.

Characteristics of asthma and its management (Table). In six cases asthma had developed during the first year of life and in the remainder by the age of 4. Eczema was present in 10 and hay fever without eczema in two. All except one child had a family history of asthma, involving first or second degree relatives. During the year before death asthma was considered by the general practitioner to be improving in five children, unchanged in four, but worsening in seven. Even when at their best in the last year, four were breathless on mild exertion and two breathless even on dressing. Only five children had ever had pulmonary function tests performed by their general practitioner. Of the 14 children aged 8 and over and considered old enough to use peak expiratory flowmeters, 10 had never done so, and only two were using meters regularly at home.

Ten of the 16 children had had two or more emergency visits to their general practitioner or

Table Patient characteristics and circumstances of death

Sex	Age at death (yrs)	Race*	Regular treatment**	Nebuliser use at home	Duration of last attack (h)	Place of death	Emergency calls in last year	Hospital admissions in last year	Severity: assessment by general practitioner
F	2	Maori	—	—	2	Home	0	1	Severe
M	3	Maori	β,Cr	Regular	84	Home	6	1	Severe
F	8	Maori	β,Th,Cr	—	0.5?	Relative's home	Unknown	0	Moderate
F	8	Maori	β,Th,Cr	—	0.2	Relative's home	4	3	Severe
F	10	Maori	β,Th	—	0.1	Car	6	1	Moderate
M	10	Eur	β,Cr,Bc	Occasional	16	Home	3	0	Moderate
F	10	Eur	β,Th,Cr,Bc	—	0.2	Home	0	0	Moderate
F	11	Maori	β,Th,Cr,Ip	Regular	3.5	Car	5	4	Severe
M	11	PIP	β	—	0.7	Taxi	5	0	Moderate
M	11	Eur	β,Th,Cr	—	14	Home	3	0	Moderate
F	11	Eur	β	—	72	Home	5	0	Mild
M	12	Eur	β,Th,Bc,S	Regular	12	Friend's home	10	7	Severe
M	12	Eur	β,Cr,Bc,S	Occasional	0.1	Home	4	1	Moderate
M	13	Eur	β	—	0.6	Home	0	0	Mild
F	14	Eur	β,Th,Cr,Ip,S	Regular	10	Home	20	16	Severe
M	14	Maori	β,Bc?	—	48	Camp site	0	0	Moderate

*Race: PIP=Pacific Island Polynesian; Eur=European.

**Treatment: β=Beta₂ agonist; Th=Theophylline; Cr=Cromoglycate; Bc=Beclomethasone; Ip=Ipratropium; S=Oral steroids.

accident and emergency department within the last year. Twelve children had been admitted to hospital with asthma, eight in the year before death. In only five cases was there evidence of a crisis plan for dealing with an asthmatic emergency, the instruction being to contact their doctor, use a nebuliser, or seek direct hospital admission. Four children, however, had never required emergency general practitioner or accident and emergency visits or hospital admissions.

All except one were regularly inhaling a beta₂ agonist drug, seven used cromoglycate, seven beclomethasone, and three daily or alternate day corticosteroids. Four children used nebulisers regularly at home for the administration of a beta₂ agonist drug, and a further two used a nebuliser occasionally for severe episodes. Only one child using a home nebuliser had a peak flowmeter. Compliance with treatment with drugs was noted by the general practitioner in 14 children of school age as good in seven children, satisfactory in a further two, and poor in five. The specialist reviewing physicians considered that the regular treatment supervised by the general practitioner was inadequate in five cases and that in four cases advice given or follow up arranged by a specialist physician or paediatrician seemed inadequate.

Characteristics of families. Stressful psychosocial circumstances and disturbances of relationships were identified in eight families. Comments made by general practitioners included 'poor family care of children', 'mobile family', 'alcoholic mother', 'neglected child', 'non-compliant family', and 'fatalistic attitude'. In 12 families at least one parent smoked regularly. Two mothers had died of asthma.

Circumstances of fatal episode. Four deaths occurred in January during the summer holiday season. Eight children died after a gradual increase in symptoms of asthma over hours or days. Seven died in less than three hours from the reported onset of their wheezing, and one child was found dead. In only six instances had the general practitioner or his deputy been telephoned. One child died minutes after the doctor was telephoned, two were seen by their doctors and advice was given, but no visit was made in three cases. With one exception, the doctor called was the child's usual practitioner. All children died outside hospital. There was no clustering of times of death; nine of the 16 died between 1800 and 0600 hours.

In two cases excessive treatment with drugs may have contributed to the fatal outcome. A 14 year old child with worsening asthma used a full beta₂ agonist

inhaler over two days, but used it only three times during the three hours before death. Autopsy revealed severe mucous plugging. An 8 year old child with increasing nocturnal wheeze was given four or five tablets (thought to be sustained release theophylline) over eight hours by a relative, but died from severe asthma. An autopsy was not performed.

Four of the six children with home nebulisers had used these during the last few hours of life when asthma was increasing in severity. Medical advice was obtained by telephone in two of these cases, but other appropriate treatment was not given. Overreliance on a home nebuliser, which was used up to two hourly for 10 and 16 hours, seemed to lead to delay in beginning more aggressive treatment in these two cases. The use of a home nebuliser did not adversely affect management of the other two children. Delays in obtaining help in these were due to failure to appreciate the severity of the attack or reluctance to call for help.

Because of the rapidity of deterioration, the deaths of two children were regarded as unavoidable. A two year old known to have asthma, apparently well apart from an upper respiratory tract infection, was found dead two and a half hours after being put to bed. Autopsy showed mucous plugging of bronchi as well as acute bronchiolitis. A 12 year old with moderately severe asthma well controlled with daily oral steroids and appropriate inhaled drugs died within five minutes of the onset of the attack. His peak expiratory flow rate one and a half hours earlier had been normal, although he then had symptoms of influenza. The physicians classified the deaths of three other children who had precipitous attacks as possibly unavoidable.

In the remaining 11 children avoidable factors contributed to the fatal outcome. In four children long term treatment was inadequate due either to attitudes of the family (one strongly antagonistic to any treatment with drugs, the other accepting asthma with resignation and using minimal treatment) or the medical advisers (two cases). In one instance the family did not call the general practitioner during several hours when asthma was increasing. Difficulties in communication with a general practitioner through a receptionist led to undue delay and death in one case. One child who had recently graduated from a paediatric to an adult respiratory service no longer sought direct hospital admission. She had survived previous severe attacks when admitted directly to hospital. One family delayed calling their general practitioner, then took the child to hospital by car as previous use of an ambulance had resulted in a request for payment. The child died in the car; oxygen might have been life saving. In four cases the response of the medical

service and the assessment of asthma seemed inadequate. Advice was given by telephone without the doctor seeing the child, or treatment given was inappropriate to the severity of the asthma. One child was seen by two different locums over three days and on all three occasions received a nebulised beta₂ agonist only. No child received additional treatment with steroids before death. No child for whom a doctor was consulted was referred for urgent hospital admission. Two children lived over 20 km from a medical centre, but remoteness from care contributed to only one death.

Discussion

The mortality rate from asthma in New Zealand children is approximately three times that of the United Kingdom.² Possible reasons for this include a higher prevalence of asthma, greater severity, or poorer management. There are reports that the prevalence of asthma in New Zealand is higher than that in the United Kingdom.¹¹⁻¹³ Formal comparisons of severity of asthma have yet to be undertaken, but physicians and paediatricians employed in the United Kingdom before coming to New Zealand consistently state that asthma in New Zealand is more severe and less responsive to treatment.¹⁴ That all childhood deaths from asthma in our study occurred outside hospital may reflect a greater severity and a shorter time course of fatal asthma.

The New Zealand study⁹ is the first complete national study of mortality from asthma and has identified the largest reported community series of childhood deaths from asthma collected within a short period. We believe our case finding methods detected all childhood deaths from asthma over this period, but as we did not investigate every childhood death, it is possible that mortality from asthma could be even higher. In Avon county, England, 26 of 28 possible cases were detected by a search of death certificates.⁴ The present study provides a wider range of deaths than earlier series based on hospital treated patients,⁵⁻⁸ and more complete information on circumstances surrounding death, including treatment, than was available for cases dying in (37%) or out (63%) of hospital in Avon county.⁴

The dominance of Maoris in the under 10 age group and the fivefold increase in mortality from asthma among Maori children is a cause for particular concern. Prevalence rates for asthma in each ethnic group are not yet known, but differences in prevalence could not be great enough to account for a fivefold higher mortality in Maoris. The numbers of deaths are small and are drawn from a small population of 840 000 in this age group, but the rates

are consistent with the data from the much larger adult New Zealand population.⁹

The mean age of European children in our study was 12.0 years, 3.5 years older than in Avon county.⁴ Severe asthma is more prevalent in boys both in Britain⁴ and in New Zealand,¹¹ but in neither country is this reflected in the mortality rates. The reason for the apparent 'excess' mortality among girls is not evident.

Medical and family attitudes to treatment with drugs resulted in inadequate treatment in some cases, including instances where inhalers had deliberately not been replaced by a parent so that the child would learn to manage without, and where treatment was said to be unnecessary or inadvisable. Two children subjected to therapeutic nihilism on medical advice had within the month before their death been started on treatment with inhaled beta₂ agonist for severe asthma because of worsening symptoms, but had not been given other drugs, including treatment with steroids. In only two cases did we find evidence of apparently excessive drug use. Overreliance on a home nebuliser for administering bronchodilator drugs resulted in delay in beginning more adequate treatment in two other cases. It is of great concern that no child had been given additional treatment with steroids during the fatal attack. By contrast, six of 13 children in Avon county whose treatment was recorded were given steroids, although the dose was inadequate in four.

The disturbed family circumstances encountered in half of these children may have contributed to mortality. Neglect of children, care of children by other than immediate family members, and stressful home circumstances with ill or alcoholic parents seemed relevant. These family circumstances probably led to less attention being given to the child's asthma.

Assessment of severity of asthma can be difficult, but in many of these cases the assessments made by the families and their doctors seemed inadequate. The virtual absence of peak flow monitoring must have contributed to this lack of awareness of severity.

Four of the fourteen children aged 5-14 years died in January during the New Zealand summer. Exposure to allergen might possibly have been associated with increased mortality. Seasonal variation in childhood asthma admissions and deaths has recently been reported in the United Kingdom;^{15 16} heightened bronchial reactivity associated with spring pollens was suggested as a possible cause of summer deaths. Unlike the findings in New Zealand⁹ and British⁷ adults, and in those children in Avon county for whom time of death was known,⁴ there was no excess in nocturnal deaths in

New Zealand children, despite 14 of the 16 having more troublesome symptoms in the evenings or through the night.

Many of the factors associated with mortality from asthma in childhood were similar to those found in the British Thoracic Association study of mortality among adults with asthma¹⁷—namely, chronic undertreatment, lack of objective assessment of severity, and delay in calling help. Disturbed family circumstances may compound these problems in childhood where clearly parental care and knowledge are of greater importance.

At least 11 of the 16 deaths in children aged under 15 were associated with avoidable factors. Measures that we believe could lower the mortality rate from asthma among children include increased family awareness of asthma and understanding of a crisis plan for dealing with an emergency due to asthma; regular assessment of severity and control with peak flow monitoring; improved access of high risk children with asthma to medical care, including provision for direct hospital admission; and removal of financial barriers, including the cost of ambulances, general practitioner consultations, and peak flowmeters. Improved long term care, including education to accept regular treatment to control abnormal lung function as well as symptoms, may reduce mortality. While further studies are required to prove the benefits of both education and long term treatment this survey of childhood deaths from asthma suggests these are areas for particular action.

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