Growth in atopic eczema

A A Massarano, Sally Hollis, J Devlin, T J David

Abstract

Growth was studied in 68 children aged 2-12 years with atopic eczema. Height SD scores were significantly correlated with the surface area of skin affected by eczema. The mean height of 41 patients with less than 50% of their skin surface affected (group I) was normal (mean SD score -0.11). The 27 children with more than 50% of their skin affected (group II) were significantly shorter (SD score -0.83) and were also short allowing for their parental target height. The predicted heights were also normal in group I but were lower than expected in group II. Regression analysis suggested that height was most dependent on parental height. The extent of the disease had a significant additional effect, whereas dietary treatment and treatment with topical steroids had only marginal additional effects. The growth of children with eczema affecting less than 50% of the skin surface area appears to be normal, and impaired growth is confined to those with more extensive disease.

(Arch Dis Child 1993; 68: 677-679)

The growth of children with atopic eczema has attracted little attention. In one cross sectional study of 89 patients aged 1-16 years requiring regular hospital attendance, the height of nine (10%) children was below the third centile.¹ When corrected for mid-parental height, the height of 16 (23%) of 69 patients aged 2-9 years was below the tenth centile.¹ Those with short stature tended to have extensive eczema, to use more potent topical steroids, and to have more severe asthma, but these trends were not statistically significant. A postal questionnaire study which relied on home measurement of 128 patients with atopic eczema up to 16 years of age found the height of patients to be significantly less than that of their unaffected friends.²

The aim of this study was to examine the growth of children with eczema confined to the age of the mid-childhood period of growth^{3,4} which avoids the confounding variability of the

age of puberty. An attempt was also made, using data routinely collected in all children with atopic eczema who regularly attend our unit, to assess which factors could be implicated in any growth impairment.

Patients and methods

Sixty eight patients with atopic eczema (46 boys, 22 girls) who regularly attended the university department of child health and who fulfilled the diagnostic criteria of Hanifin and Rajka⁵ were studied. All were prepubertal and aged 2-10 years for girls or 2-12 years for boys. None had evidence of any chronic non-atopic disease which would limit growth; one had well controlled insulin dependent diabetes. Sixteen of the patients had been included in a previous study.¹

The height of all patients was measured on a Harpenden stadiometer by a single observer (AAM) using standard techniques.⁶ Bone ages⁷ were assessed in patients over 6 years of age by a single observer (AAM); predicted final height was calculated by the TW2 method.⁷ To compare heights of children of different ages and different sexes the height and predicted height SD scores, representing the number of SDs away from the mean, were calculated from standard data.⁸ Heights were also related to parental height for comparison with charts allowing for mid-parental height.⁹ Preferably both parents were measured; at least one parent was measured in 57 (84%) cases. If one or other of the parents was not available, the height was taken from recall or by a measurement at home. The parental target height was calculated as the mean of the parental heights after either adding 12.6 cm to the maternal height for a boy, or after subtracting 12.6 cm from the paternal height for a girl. The parental target height SD score was then calculated.⁸

The percentage of skin surface area affected by erythema, vesicles, and crusts was routinely recorded at each outpatient visit. In this study we used the maximum figure ever recorded for each patient. All patients had received topical corticosteroids, which were scored according to the *British National Formulary* classification¹⁰ as category 4 (mildly potent), category 3 (moderately potent), category 2 (potent), or category 1 (very potent). Systemic corticosteroids were scored for analysis as 0.

Exclusion diets were noted and scored as follows: 1, normal diet, but including avoidance of additives, or milk free if soya based milk formula used as substitute; 2, avoidance of one or two key foods, such as eggs, wheat, or milk (if not substituted with an alternative milk formula); 3, avoidance of three or more key foods; 4, a few food diet¹¹ (those who rapidly returned to a normal diet within a few weeks were scored as 2); and 5, elemental diet.¹²

Department of Child Health, University of Manchester

A A Massarano

S Hollis

J Devlin

T J David

Correspondence to:

Professor T J David,
University of Manchester
Department of Child Health,
Booth Hall Children's
Hospital, Charlestown Road,
Blackley, Manchester
M9 2AA.

Accepted 8 December 1992

Table 1 Details of 68 patients with atopic eczema separated into two groups by the maximum percentage surface area of skin affected

	Below 50% (group I) (n=41)	Over 50% (group II) (n=27)
Boys/girls	27/14	19/8
Median surface area (%)	18	75
Median (range) age (years)	5.3 (2.3-11.6)	7.0 (3.0-11.9)
Median (range) duration of eczema (years)	5.0 (2.2-11.2)	6.1 (3.1-11.8)
No (%) previously treated with systemic steroids	0 (0)	5 (19)
Median treatment score	4 (4-2)	3 (4-0)
Asthma		
No (%) with asthma	30 (73)	19 (70)
Median asthma score	1 (0-3)	1 (0-3)
Diet		
No (%) with restricted diet	17 (41)	24 (89)
Median diet score	1 (1-5)	5 (1-5)
No (%) with elemental diet	2 (5)	17 (63)

Height SD score and surface area affected by eczema.

Forty nine patients also had asthma (table 1). Treatment of coexisting asthma was graded as follows: 1, bronchodilators only; 2, controlled with regular inhaled corticosteroids or cromoglycate; and 3, difficult to control despite prophylaxis and bronchodilators.

Height SD scores were compared with the surface area of eczema by Spearman's rank correlation. Height SD scores and predicted height SD scores were compared with the parental target height SD scores by the Wilcoxon signed rank test. Between groups comparison was by the Mann-Whitney U test. Sex ratios were compared by χ^2 analysis. Regression analyses, comparing height SD score as the dependent variable with several explanatory variables, was performed using the SPSS program.¹³ Data are presented as either median (range) or mean (SD).

Results

The median age of the 68 patients was 6.2 years (range 2.3–11.9) and the median surface area affected by eczema was 30% (range 2–95). Forty one patients were either receiving or had received an exclusion diet of whom 19 had received an elemental diet. Five had previously been treated with either prednisolone or corticotrophin. Forty nine of the patients had coexisting asthma.

The figure shows that there was a highly significant correlation between height SD score and surface area of eczema (Spearman's correlation coefficient $r_s=0.42$; $p=0.003$). There appeared to be two groups of patients; the height

Table 4 Regression analysis: dependent variable height SD score analysed with various explanatory variables

Variable	r^2 (%)
Parental target height SD score	23.6
Extent (surface area) of eczema	17.7
Diet score	12.2
Total duration of eczema	11.0
Treatment score	9.2
Asthma score	3.3

SD score of those with less extensive disease was within the normal range, whereas that of the severely affected patients was either in the lower half of or below the normal range. The 68 patients were therefore separated into two groups (see table 1). Forty one had less than 50% of their body surface area affected by eczema (group I). Twenty seven had more extensive disease (group II). The ages and sex ratios were similar ($U=460$, $p=0.27$; and $\chi^2=0.016$, $p=0.9$ respectively). Group II patients had significantly higher treatment ($p<0.01$) and diet ($p<0.0001$) scores, but similar asthma scores.

Table 2 gives auxological data for the two groups of patients. The height of group I patients was normal; their height SD score was almost identical with that of their parents. Group II patients were short; their height SD scores were significantly less than those of their parents ($t=53$; $p=0.001$) and they were also shorter than group II patients ($U=278$; $p=0.0007$).

Of those in group I the heights of only two (5%) were below the third centile allowing for mid parental height. In these two children, although the area of eczema was limited, the skin lesions were particularly severely inflamed. Of the 27 patients in group II the height of eight (30%) was below the third centile allowing for their parents. Four of the eight had received an elemental diet and one had also received systemic steroids. Two patients in group I (5%) and none in group II had a height above the 97th centile allowing for the parents' height.

Of the 36 children aged 6 years or older, the bone age was estimated in 35 (all 19 in group I and 16/17 in group II). The bone age was mildly retarded in the two groups. Bone age retardation was greater in group II, but the difference was not statistically significant ($U=225$; $p=0.09$). When calculating predicted heights (table 3) those of the group II patients were not significantly less than the mid-parental heights ($p=0.08$). The predicted heights were, however, below those of the group I patients despite having taller parents ($U=111$; $p=0.18$).

Table 2 Mean (SD) height of patients in the two groups

	Below 50% (group I)	Over 50% (group II)	p Value
Height SD score	0.11 (1.04)	-0.83 (1.06)	0.001
Parental height SD score	0.08 (0.73)	0.08 (0.88)*	0.64
Bone age retardation (years)	0.5 (1.2)	1.1 (1.0)	0.09

* $p<0.001$ compared with height SD score of group II patients.

Table 3 Predicted mean (SD) height of patients aged at least 6 years in the two groups

	Below 50% (group I)	Over 50% (group II)	p Value
Boys/girls	14/5	11/5	
Parental height SD score	0.08 (0.68)	0.23 (0.77)	0.08
Predicted height SD score	0.25 (0.94)	-0.25 (1.11)	0.08

REGRESSION ANALYSES

The dependent variable of height SD score was analysed with several explanatory variables, namely parental target height, surface area, treatment score, diet score, asthma score, and duration of disease (table 4). The variation in height SD score was best explained by parental target height ($r^2=0.24$) and this variable was then included for further comparisons. Surface area had the second largest effect (additional $r^2=0.13$; $p<0.001$), the combination of parental height and surface area explaining 36% of the variation in height. Further combination with treatment

score (additional $r^2=0.03$; $p=0.07$) and diet score (additional $r^2=0$) had only a marginal additional effect and no effect respectively. Asthma score and duration of disease had no additional effect.

Discussion

Impaired growth is a recognised complication of many chronic diseases during childhood, including asthma.^{14,15} In contrast with the latter, however, the poor growth of some children with atopic eczema has received scant attention. As in two previous reports on the subject,^{1,2} this study used patients who attended a tertiary referral centre and who were therefore a highly selected group.

In contrast with the previous studies we have been able to show that the growth of children with eczema is statistically significantly related to the severity of disease. The height of patients in whom eczema affected less than 50% of their body surface area was normal, compared with standard data and with their parents. Those with eczema which affected more than 50% of their body surface area, however, had notable growth impairment. The mean height SD score of -0.83 in patients in this group implies that their heights were centred around the 25th rather than the 50th centile. Eight of the 27 (30%) patients in the latter group had a height less than the third centile when corrected for their parents' height.

The previous finding of more generalised short stature in a group of children with atopic eczema compared with control children² is probably explained by the inclusion of a small number of severe cases. In addition, the overall figure of height SD score in their study of -0.45 was less than the figure of -0.26 found in the present study, and the former may have been due to the inclusion of adolescent patients with pubertal delay. Delay in puberty would also explain the previous finding of a relatively lower sitting height than subischial leg length in patients with atopic eczema.¹ It is known that there is less growth in the spine than there is in the legs in patients with pubertal delay.¹⁶

The regression analysis showed that apart

from parental heights, the largest influence on height SD score was the surface area of skin affected by eczema. The reason for this association is unclear. There appeared to be no independent effect of asthma, and little effect of strength of steroid preparation or use of exclusion diets. Any effect of the latter two may have been masked by the fact that the most severely affected patients tended to receive the strongest steroid preparations and the most stringent diets.

Our data suggest that children with atopic eczema which affects less than 50% of the skin surface area have a normal height, but that those with more extensive disease may have impaired growth. The cause of this impaired growth has not been identified.

- 1 Kristmundsdottir F, David TJ. Growth impairment in children with atopic eczema. *J R Soc Med* 1987; 80: 9-12.
- 2 Pike MG, Chang CL, Atherton DJ, Carpenter RG, Preece MA. Growth in atopic eczema: a controlled study by questionnaire. *Arch Dis Child* 1989; 64: 1566-9.
- 3 Karlberg J. A biologically orientated mathematical model (ICP) for human growth. *Acta Paediatr Scand Suppl* 1989; 350: 70-94.
- 4 Sorva R, Lankinen S, Tolppanen E-M, Perheentupa J. Variation of growth in height and weight of children, II. After infancy. *Acta Paediatr Scand* 1990; 79: 498-506.
- 5 Hanifin JM, Rajka G. Diagnostic features of atopic dermatitis. *Acta Derm Venereol Suppl (Stockh)* 1980; 92: 44-7.
- 6 Brook CGD. *Growth assessment in childhood and adolescence*. Oxford: Blackwell, 1982.
- 7 Tanner JM, Whitehouse RH, Cameron N, Marshall WA, Healy MJR, Goldstein H. *Assessment of skeletal maturation and prediction of adult height (TW2 method)*. New York: Academic Press, 1975.
- 8 Tanner JM, Whitehouse RH, Takaishi M. Standards from birth to maturity for height, weight, height velocity and weight velocity: British children, 1965; Part II. *Arch Dis Child* 1966; 41: 613-35.
- 9 Tanner JM, Goldstein H, Whitehouse RH. Standards for children's height at ages 2-9 years allowing for height of parents. *Arch Dis Child* 1970; 45: 755-62.
- 10 British Medical Association and the Royal Pharmaceutical Society of Great Britain. *British national formulary*. Number 23. London: British Medical Association and the Royal Pharmaceutical Society of Great Britain, 1992: 393.
- 11 Devlin J, David TJ, Stanton RHJ. Six food diet for childhood atopic dermatitis. *Acta Derm Venereol (Stockh)* 1991; 71: 20-4.
- 12 Devlin J, David TJ, Stanton RHJ. Elemental diet for refractory atopic eczema. *Arch Dis Child* 1991; 66: 93-9.
- 13 Norusis MJ. *SPSS/PC+V 3.0 update manual*. Chicago: SPSS, 1988.
- 14 Cohen MB, Abram LE. Growth patterns of allergic children. *J Allergy* 1948; 19: 165-71.
- 15 Falliers CJ, Szentivanyi J, McBride M, Bukantz SC. Growth rate of children with intractable asthma. *J Allergy* 1961; 32: 420-34.
- 16 Buckler JM. *A longitudinal study of adolescent growth*. London: Springer Verlag, 1990: 199.