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Bacterial infection and atopic eczema

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SUMMARY One hundred and ninety children with atopic eczema were studied prospectively for two and a half years. The mean period of observation was 13 months. Seventy six children (40%) had between them 164 episodes of exacerbation of eczema due to bacterial infection, and in 52 (32%) infection recurred within three months of a previous infection. Twenty five episodes (15%) led to admission to hospital. *Staphylococcus aureus* was recovered in 97% of episodes, in combination with β haemolytic streptococci in 62%. Physical signs suggesting infection were pustules, crusting, and a weeping discharge, but these signs alone are not diagnostic, and an exacerbation was only attributed to infection if there was a response to anti-infective treatment. Exacerbation of atopic eczema due to bacterial infection is common, the physical signs of infection are not always clear, and there is a case for a trial of oral antibiotics in any child with troublesome atopic eczema.

There is a high rate of skin colonisation with *Staphylococcus aureus* in atopic eczema. For example, Leyden *et al*[1] recovered *S aureus* not only from more than 90% of isolates taken from chronic eczematous lesions, but also from approximately 70% of isolates taken from unaffected areas of the skin. By contrast, Noble[2] found *S aureus* on the skin in fewer than 5% of isolates taken from various sites in 382 normal children. The high frequency of *S aureus* carriage in clinically normal skin in patients with atopic eczema may be contamination due to continual dispersion from skin lesions or colonisation. Impaired neutrophil chemotaxis,[3] and greater adherence of *S aureus* to keratinised epithelial cells of atopics[4] may contribute to the high rate of skin colonisation.

It is often held that while the skin in atopic eczema is usually colonised with *S aureus*, bacterial skin infections are uncommon, and less frequent than might be expected in a disease characterised by scratching and excoriation.[5] Observation of children with atopic eczema suggests, however, that in this disorder the frequency of bacterial skin infection is higher than previously suspected. This report describes a prospective study of bacterial infections in children with atopic eczema.

Patients and methods

Between January 1982 and July 1984, all children with atopic eczema referred to the Department of Child Health at Booth Hall Children's Hospital were studied prospectively for evidence of skin infection. One hundred and ninety patients, aged 7 weeks to 17 years (median 3 years) were observed for 2480 patient months, with a mean of 13 months.

The presence of pustules, a purulent discharge, crusting combined with weeping (Figure (a)), crusting alone (Figure (b)), or sudden appearance of weeping, were taken as physical signs of infection. Children were seen as outpatients at intervals no longer than three months, and their parents were asked to bring the child if there was sudden or unexpected deterioration of the eczema or if any of the above signs were noticed. Episodes of infection due to Herpes simplex,[6] Herpes zoster, and Coxsackie A have been excluded. Nine episodes of infection treated by the general practitioner and not seen at hospital were also excluded.

The criteria for 'infection' comprised (a) one of the above signs, together with (b) response to oral antibiotic or topical antiseptic treatment (see below). Episodes that did not respond to this treatment were classified as 'possible infections'. A recurrent infection was defined as one occurring within three months of a previous infection.

The following oral antibiotics were given in a dosage of: (a) phenoxymethylpenicillin, flucloxacillin, fusidic acid, or erythromycin—under 10 years 125 mg four times daily; over 10 years 250 mg four times daily: (b) cefadroxil: under 5 years 250 mg twice daily; over 5 years 500 mg twice daily.

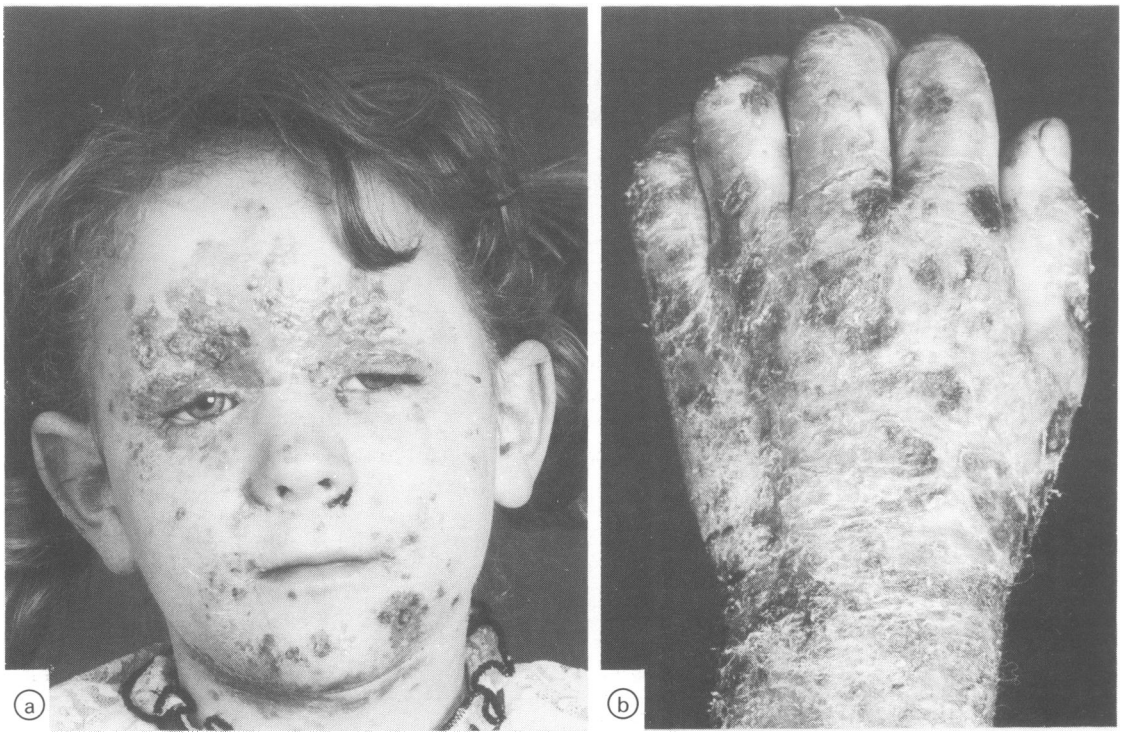

Figure *A 6 year old child with atopic eczema. The crusting plus weeping on the face (a), and the dense crusting on the hand (b), suggest the presence of bacterial infection.*

Antibiotics were always given in tablet or capsule form because of the possibility of adverse reaction to colouring agents or preservatives present in liquid preparations. A combination of phenoxymethylpenicillin plus flucloxacillin was the first choice for oral antibiotic treatment. Either antibiotic was used alone only for recurrent infections where eradication of a single organism was intended. Cefadroxil was used in cases where it was suspected that the family would not comply with four times daily dosage of two antibiotics. Erythromycin or fusidic acid were used in cases of penicillin allergy. A combined topical corticosteroid-antiseptic ointment was used in the presence of chronic lichenified patches of eczema. Topical treatment with an antiseptic, povidone-iodine 10% ointment, was used where a purulent discharge was present. Topical treatment alone was given only where it seemed that the infection was localised.

Results

One hundred and sixty four episodes of infection and 20 episodes of possible infection (as defined above) occurred in 76 (40%) of the 190 patients. Twenty six patients were infected at their first visit. Twenty five episodes (15%) of infection led to admission to hospital. Three children were admitted twice for this reason. Twenty three admissions were because of the severity and extent of the infected lesions, and two admissions were because of systemic illness associated with the infection. The bacteriological results and details of treatment are given in the Table.

Recurrent infections. Fifty two (32%) of the 164 infections were recurrences within three months of a previous infection, 30 of the 52 occurring within one month of the previous infection. Attempts to prevent further recurrences comprised the topical application to the inside of the nostrils (four times daily for two weeks) of chlorhexidine hydrochloride 0·1% and neomycin sulphate 0·5% cream (21 infections), and the addition of 25 ml of chlorhexidine gluconate 1·5% and cetrimide 15% solution to each bath. There are insufficient data to assess the efficacy of either procedure.

Table *Infections and possible infections: bacteriology results and treatment*

	Infections (n=164)	%	*Possible infections (n=20)*	%
Bacteriology				
Skin swab results	*Staphylococcus aureus* + β haemolytic streptococci	62*†	*S aureus* + β haemolytic streptococci	62
	S aureus alone	35*	*S aureus* alone	38
	β haemolytic streptococci alone	3		
Lancefield grouping of β haemolytic streptococci	Group A	68‡	Group A	100
	Group B	13		
	Group C	8		
	Group G	10		
	Group L	1		
Resistance of *S aureus*	Penicillin	70	Penicillin	100
	Erythromycin	18		
	Tetracycline	12		
	Fusidic acid	1		
	Gentamicin	1		
Treatment				
Oral antibiotics for 14 days		94		81
	Phenoxymethylpenicillin + flucoxacillin	63	Phenoxymethylpenicillin + flucoxacillin	82
	Flucoxacillin	14	Flucoxacillin	6
	Cefadroxil	8	Phenoxymethylpenicillin	6
	Phenoxymethylpenicillin	6	Phenoxymethylpenicillin + fusidic acid	6
	Fusidic acid + erythromycin	3		
	Fusidic acid	3		
	Erythromycin	3		
Topical steroid/antiseptic		33		40
	Clioquinol 3%/hydrocortisone 1% ointment	25	Clioquinol 3%/hydrocortisone 1% ointment	40
	Chlorquinaldol 3%/hydrocortisone 17-butyrate 0·1% oint.	6		
	Clioquinol 3%/flurandrenolone 0·0125% ointment	2		
Topical antiseptic	Povidone-iodine ointment	21	Povidone-iodine ointment	5

*Combined with pneumococcus in one patient.
†Combined with *Pseudomonas aeruginosa* in one patient.
‡7% were resistant to erythromycin.

Nose and throat swabs. *Staphylococcus aureus* was recovered from 41% of all nose swabs. Beta haemolytic streptococci were recovered from 24% of all throat swabs and from 42% of those from patients with positive streptococcal cultures from the skin. In no patient did a nose swab indicate a staphylococcus when one was not also found in the patient's skin. In three patients who had only a staphylococcus in the skin, a throat swab detected β haemolytic streptococcus, and in two of these three this was a group C organism.

Adverse effects. Deterioration of the eczema was attributed to an oral penicillin in two patients, and the treatment changed to another antibiotic. The combination of two penicillins resulted in diarrhoea in two patients, causing the parents to stop treatment. The unpleasant taste of flucloxacillin powder led to a change to the antibiotic syrup in two patients. The antiseptic component of the topical antiseptic-corticosteroid combinations invariably caused yellow staining of clothes or hair.

Discussion

There are problems with the definition of 'infected eczema'.[7] While the discharge of pus strongly suggests infection,[8] other appearances are not so clearly diagnostic. The presence of weeping is unreliable; although it may be due to infection, it may also be the result of a deterioration due to allergy or simply to incessant scratching. The presence of bacteria on eczematous skin is almost universal,[9 10] and colonisation cannot be considered a criterion of infection. In this study, the definition of infection included response to anti-infective treatment, usually oral antibiotics. Oral antibiotics were chosen because of the widespread nature of many of the infections, and the severity of some. Topical antibiotics were not used because of the possibility of sensitisation and the difficulty of application to widespread lesions. All patients with physical signs suggesting infection were treated, so the possibility that some lesions might have recovered spontaneously cannot be excluded.

There were 164 episodes of symptomatic bacterial infection affecting 40% of 190 children with atopic eczema during a two and a half year period. This does not include those treated by the general practitioner or those with uncertain response to treatment. The high hospital admission rate partly reflects the severity of some of these cases. It is also partly attributable to the local policy of using short admissions to initiate treatment and teach parents how to continue the same management at home. As in any hospital based study, the patients were selected and contained an excess of severe or intractable cases, but in this group infection was common and caused substantial morbidity.

The importance of *S aureus* as a skin pathogen is well known, but the frequency of concomitant infection with β haemolytic streptococci, 62% in this series, is less well appreciated. This high incidence of streptococci underlines the need for treatment of both streptococci and staphylococci in infected eczema. Flucloxacillin has antistreptococcal activity, and ought to be adequate as a single agent to deal with both bacteria. Previous local experience of treatment failure with flucloxacillin alone, however, led to the addition of phenoxymethylpenicillin. Erythromycin was avoided except in cases of penicillin allergy, because of the high incidence (18%) of erythromycin resistant staphylococci. Cefadroxil was sometimes used, as a single agent to cover both pathogens and having the advantage of requiring only twice daily dosage. The role of topical antiseptics, topical antiseptic-corticosteroid preparations, antiseptics in the bath, and intranasal neomycin and chlorhexidine cream are not clarified by this study.

Thirty two per cent of the β haemolytic streptococci were not Lancefield group A. The data do not distinguish between the relative pathogenicity of different strains of streptococci in atopic eczema, nor do they prove the individual importance of either streptococci or staphylococci. Treatment was directed against both organisms. Obtaining bacteriology swabs from the skin was atraumatic, but taking nose and throat swabs was disliked. The results indicate that taking nose and throat swabs is unnecessary, for it did not detect organisms that could not be more simply recovered from the skin, and did not influence treatment.

This study shows that it is common for bacterial infection to cause exacerbations of atopic eczema. The signs of infection are not always clear, and there is a case[7] for a trial of oral antibiotic treatment in any child with troublesome atopic eczema.

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