Correspondence

Elimination diets in eczema

Sir,

We read with interest the paper by T J David on anaphylactic shock during elimination diets for severe atopic eczema. We recently observed a similar case. This girl had developed severe diarrhoea at 5 weeks of age while being fed an adapted cows’ milk formula (Preaptamil, Milupa). During a milk free diet her symptoms subsided, but diarrhoea recurred when a full amount of adapted cows’ milk formula was again given. The infant stayed well and lost little weight. Three such challenges were carried out, but after the third the severe diarrhoea persisted even when lactose-free soya protein milk was given. At age 7 weeks the baby was fed a lactalbuminhydrolysate milk formula (Alfaré, Nestlé) and on this regimen she quickly recovered and gained weight.

Although we believed that she suffered from cows’ milk protein intolerance, it was thought that the symptoms might also have been due to a postdiarrhoeal lactose intolerance. At the age of 16 weeks, therefore, after nine weeks of receiving Alfaré, reintroduction of cows’ milk formula was attempted. The baby was given 150 ml adapted cows’ milk formula (Preaptamil, Milupa) at 11 am. The feed was well tolerated and she stayed asymptomatic. The next feeds were Alfaré at 2 pm and 5 pm. Suddenly, 9½ hours after the cows’ milk feed, she was found by the nurse nearly dead, grey and cyanotic, with gasping respirations, and vomited milk was seen around the mouth. She was resuscitated and treated in the intensive care unit without further complications. No milk was found in the trachea and the chest film showed no aspiration pneumonia. Subsequently, however, spastic tetraplegia developed.

At the time we were uncertain about the aetiology of the shock, because we had never seen delayed anaphylactic shock in cows’ milk intolerance. Having read the paper by David we feel that the aetiology in our patient might be similar to that of the patients reported.

Close observation might lead to a more frequent recognition of delayed anaphylactic shock and sequelae in patients challenged after long term elimination diets.

U GORIU
University of Graz,
and I MUTZ
Children’s Hospital,
Loeben,
Austria

Dr David comments:

There is a striking resemblance between this important report, case 4 of the original paper, and the patient mentioned in the first paragraph of the discussion of the paper. The intervals between milk exposure and collapse were 9½ hours, 10½ hours, and 8 hours respectively. The delay in onset of anaphylaxis adds a new dimension to the difficulties of food challenges. So far the problem seems to apply only to challenges with cows’ milk, though there is no reason why it should not occur with other foods. The hazards of delayed anaphylactic reactions to foods have not been clearly recorded before, though it is presumably this kind of disaster that formed the basis for the recommendation that babies having cows’ milk challenges should be kept under observation on the ward for 9 to 12 hours. Given the lack of warning of anaphylaxis, and the very small risk, it is difficult to know what should comprise ‘close observation’. Mere hospital admission is plainly no guarantee of safety. For cows’ milk challenges we try to ensure that either a nurse or the mother is with the child constantly throughout the 12 hours of inpatient observation which we insist upon, but there are often insufficient nurses. Catastrophic cases such as these are rare. Nevertheless I suspect some under reporting, and I would urge colleagues to publish (or pass to me) details of similar cases.

References


Sir,

It is not clear what is special about children with either active atopic eczema or a history of atopic eczema, that makes them react in the manner described by David in his interesting and important paper.

Of the four boys who developed anaphylaxis, two (cases 1 and 3) showed urticaria as part of the picture and one of these (case 1) showed contact urticaria also; but only one of the four (case 2) seems to have had worsening of his atopic eczema with ingestion of the particular anaphylactic allergen. The negative radio-allergosorbert test and low total IgE values for cases 3 and 4 are interesting but only add to the puzzle; perhaps case 4 did not have atopic eczema at all. Measurement of serum IgG4 antibodies against dietary allergens may be helpful in some cases of reactions to foods.

Repeated ingestion of a dietary allergen may result in the development of allergy and the appearance of a diffuse erythematous rash, urticaria, or even anaphylaxis. Urticaria is not atopic eczema and any atopic eczema already present may not necessarily worsen with exposure to the allergen producing urticaria. It is possible, however, that the widespread itching that accompanies urticaria will tend to make existing eczema more itchy and thus more prone to scratching, and may tend to localise atopic