CASE REPORTS

Anaphylactic shock in an infant after feeding with a wheat rusk. A transient phenomenon

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Summary

A 10-week-old male infant presented with a history of recurrent diarrhoea and vomiting and in a state of collapse. After much fruitless investigation he was challenged with a wheat rusk and developed life-threatening anaphylactic shock followed by watery diarrhoea due to monosaccharide intolerance. This immediate type hypersensitivity to wheat rusk was transient, the infant being able to tolerate dietary wheat at the age of 14 months.

Introduction

Allergy to food antigen may be mediated by a number of immune mechanisms, most commonly by immediate hypersensitivity (Bleumink, 1979). Anaphylactic shock from this cause is, however, rare but has been described especially after ingestion of milk, egg, fish, nuts and shellfish. Although rare, allergic shock is a serious risk because it is life threatening.

Gastrointestinal intolerance to wheat protein has been described in infants as a cause of an enteropathy (Walker-Smith, 1970; Nusslé *et al.*, 1978). Antibodies to wheat protein are found in infants with coeliac disease, cystic fibrosis and IgA deficiency (Ferguson, 1976). Immediate hypersensitivity to wheat is very uncommon although Goldstein and Hainer (1969) reported gastrointestinal and pulmonary symptoms in a 16-year-old boy exposed to wheat; allergic shock caused by a cereal mix has been observed (in a 49-year-old man), but was thought possibly to be due to a contaminant (Golbert, Patterson and Pruzansky, 1969). Recently, May and Bock (1978) reported no clinical immediate type allergic reactions to wheat in a group of patients with food allergies.

The authors now describe a case in order to draw attention to the following points: because of the rarity of the problem the cause of the life-threatening illness was not immediately obvious; like other food intolerances in infancy the problem was transient; severe watery diarrhoea and temporary

monosaccharide intolerance were complicating problems and their pathogenesis is unclear.

Case report

The first child of Greek Cypriot parents was born uneventfully at term weighing 3.5 kg. He was breast fed for 2 weeks but then changed on to an adapted cow's milk formula (SMA). He first presented to Queen Elizabeth Hospital for Children, Hackney, with acute gastroenteritis at the age of 3 months. He did not settle on out-patient treatment and required admission 3 days later, pale and ill but not dehydrated. Stool bacterial and viral pathogens were not found. He settled after 2 attempts at regrading on to an adapted cow's milk formula. Three weeks later, he presented with the sudden onset of vomiting and explosive diarrhoea one hour after a feed with a wheat rusk and was re-admitted. He was shocked but not dehydrated and was rapidly resuscitated with plasma and i.v. fluids. Investigation for infection and metabolic disorders, including adrenogenital syndrome, aminoacid and organic acid abnormalities, galactosaemia and lactic acidosis were negative, urea and electrolytes were normal. Hb, 12 g/dl, WBC $18.6 \times 10^9/l$, neutrophils 61%, lymphocytes 34%, monocytes 5%, platelets normal. Hb electrophoresis normal; yeast opsonification normal, IgG 13.53 g/l (high), IgA 1.39 g/l (high), IgM 1.26 g/l (high), IgE 17 i.u./ml (slightly raised). Radio-allergosorbent test (RAST) was not performed because of only slight elevation of IgE. A small intestinal biopsy was morphologically normal.

Detailed dietary history revealed that the infant had been fed a wheat-containing feed at 2 months of age but this was discontinued after 2 weeks because of loose stools. No further solids had been given until the day of the second admission.

After numerous investigations had excluded other causes of collapse and because of the suspicion of gastrointestinal food allergy, the infant was given a small amount of rusk orally as a challenge whilst under close observation. Two hr after

ingestion he vomited and developed watery diarrhoea, bronchospasm and shock. He was rapidly resuscitated with adrenaline, antihistamine and i.v. fluids. Twenty-four hr later, following the introduction of oral glucose electrolyte mixture he developed severe watery diarrhoea and was found to be monosaccharide intolerant. This complication responded to a period of total bowel rest followed by regrading orally from electrolyte mixture plus 1% glucose and 1% fructose on to a glucose electrolyte mixture and then an adapted cow's milk formula.

He remained well on a wheat-free diet. When aged 14 months, total IgE was <15 i.u./ml and RAST to wheat was 0, to oats 0, rye 0, egg white 0, milk 1. He was challenged at home by his mother with a wheat-containing diet, without clinical effect. He is now well and thriving, at 22 months on a normal diet, with bread and lots of spaghetti.

Discussion

This patient developed both typical systemic and gastrointestinal symptoms of a Type 1 reaction to wheat rusk (Gell and Coombs, 1975). The anaphylactic shock was life-threatening in severity. It is likely that sensitization occurred when he was fed a wheat rusk at the age of 2 months. Total IgE was only slightly elevated and RAST was therefore not performed by the laboratory. Twelve months later, RAST was negative for cereals, egg and milk. RAST is, however, known not to correlate well with clinical food allergy giving negative results in approximately 25% of patients allergic to egg and >60% of patients allergic to milk (Editorial, 1980).

Gastrointestinal intolerance to food proteins, particularly cow's milk, soy and wheat, has been observed in infants and causes an enteropathy (Visakorpi and Immonen, 1967; Walker-Smith, 1970; Ament and Rubin, 1972). It is thought that the enteropathy is caused by immunological hypersensitivity mechanisms other than immediate type. The only changes due to immediate type hypersensitivity described have been slight mucosal oedema (Ferguson, 1976). However, mixed reaction, e.g. allergic shock in an infant with cow's milk-sensitive enteropathy fed cow's milk, have been reported (de Peyer and Walker-Smith, 1977).

In the present patient, small intestinal mucosal biopsy showed no enteropathy. The mechanism of the allergic shock and the temporary monosaccharide intolerance was, therefore, most likely a Type 1 reaction. The means by which a Type 1 reaction can damage the monosaccharide carrier mechanism without causing histological tissue damage is unknown.

Damage to the monosaccharide carrier mechanisms by a Type 1 reaction has not previously

been described. Lifschitz (1977) has stressed that severe mucosal damage is usually associated with monosaccharide intolerance.

From the clinical viewpoint it is important to remember such an anaphylactic reaction as a cause of collapse in an infant, particularly, perhaps, where gastrointestinal symptoms are evident. The management of such an infant obviously requires removal of the offending foodstuff from the diet. The infant must adhere strictly to the exclusion diet, although the length of time for which this is necessary is unclear. By analogy with cow's milk protein intolerance it would seem likely that the infant would lose his hypersensitivity by the age of 2 years (Walker-Smith, 1979). At the age of 14 months, cereals were re-introduced into the diet of this infant without clinical relapse. The fall in total IgE and the negative RAST influenced the authors in the decision to challenge him. This severe anaphylactic sensitivity to wheat rusk was therefore transient in nature.

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