

Persistent cow's milk allergy with attacks of anaphylaxis

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Abstract

The most common food protein causing allergy in infants and young children is cow's milk, but persistence of this allergy into adulthood is rare. It is estimated that 2-3% of the general population suffers from cow's milk allergy (CMA), but reports of anaphylaxis in adulthood are limited. This case report discusses persistence of CMA with several anaphylaxis episodes in a 12-year-old girl who had allergic reactions from 6 months of age. She is also unable to tolerate milk from all available animal sources including goat and sheep. Skin prick test was strongly positive at 10 mm at 7 years of age, with specific IgE cow's milk >100 kUA/L when she presented with anaphylaxis episodes 5 years later. Patient-specific management plans need to be in place to avoid and manage anaphylaxis, especially for patients who do not have easy access to healthcare.

Keywords: Anaphylaxis, cow's milk allergy, immunoCAP

Introduction

Cow's milk allergy (CMA) is the most common food allergy which most children outgrow by 3 years of age. [1] Persistent CMA is a difficult condition where no treatment is available. This case report describes the difficulty that parents face when children have persistent food allergy with the added fear of anaphylaxis without access to emergency medical care.

Case report

A 12-year-old girl attended the allergy clinic for evaluation of severe allergic reactions. She was exclusively breastfed until 6 months of age. She developed redness around the mouth, coughed, and cried after every feed with several formula feeds and cow's milk. A diagnosis of extensive food allergies was made by the family doctor and advised to avoid milk, egg, wheat, soy, shellfish, and nuts. No investigations were carried out and the baby remained on this restricted diet.

Her parents started introducing wheat (rotis), fishes (hilsa, rui), and nuts in her diet at age of 7 years. She did not react to these foods but developed perioral redness, coughing after eating biscuits, cookies, and cakes. Skin prick tests were positive for egg (3 MM), shrimp (2 MM), wheat (flare only), cow's milk (10 MM

extensive flare ++). Polyvalent home dust and mixes (grasses, trees, and weeds) were negative. She was asked to avoid milk, egg, shrimp, and wheat. Lung function test excluded obstructive airways disease.

Since she had no reactions with fried egg or to wheat products, parents continued to give her egg and wheat in her diet. At 11 years old, she was able to tolerate egg (fried and boiled), shrimp, wheat, and soybean without any problems. She however had several minor reactions (perioral redness with itching) to biscuits, cakes, ice creams, curd, and sweets including serious reactions (urticaria, lip swelling, vomiting) to butter, chocolates, and Indian cheese (paneer). Her father mentioned that she was unable to breathe on 2 occasions requiring intramuscular steroids that happened when she had food at either religious festival or marriage ceremonies. Her parents tried her on goat and sheep milk including ghee (clarified butter) but had caused facial rash, coughing, and abdominal pains.

One-week before, she attended the clinic, and she had another severe allergic reaction with generalized urticaria, lip, and tongue swelling with coughing bouts followed by vomiting and apneic spell on eating fish topped with curd. She had tolerated the same fish without milk on several occasions. There was no history of rhinitis or asthma, eczema or drug allergies. There was no family history of allergies or angioedema.

On examination, she was thinly built and weighed 40 kg, height 146 cm, and blood pressure 96/76 mmHg. Physical examination was unremarkable.

Full blood count, liver function tests, thyroid profile (thyroid-stimulating hormone, fT4, fT3), and C4 level were normal. Pulmonary function test was normal, with no evidence of reversibility. Total IgE, specific IgE, and mast cell tryptase were performed using ImmunoCAP on Phadia 100 fluoro enzyme immunoassay (Thermo Fisher Scientific, Uppsala, Sweden). Results showed an elevated IgE at 665 kU/L, specific IgE against cow's milk (f2 ImmunoCAP) was >100 kUA/L (Grade 6), and negative specific IgE against common aeroallergens was <0.35 kUA/L (Grade 0). Mast cell tryptase level was normal at 2.94 ng/ml (reference range, <11.5 ng/ml) excluding the possibility of systemic mastocytosis.

A diagnosis of persistent CMA with anaphylaxis was made. She and her father were counseled regarding anaphylaxis, and an emergency management plan was provided to her. Given the unavailability of injectable epinephrine pens in India, the management plan consisted of oral corticosteroids, antihistamines and advised to visit a hospital/clinic with emergency facilities to ensure optimal treatment has been provided.

Discussion

This case highlights the importance of taking a detailed clinical history during the assessment of food allergies or anaphylaxis related to foods in children. The tests to diagnose food allergy may not always be possible or indeed required for every patient, but this should be considered by the physician, especially when multiple food items are being asked to be avoided for extended periods of time. European and US guidelines are clear on this: (1) a pediatrician with adequate training in allergy only should carry out oral food challenges as potential for misdiagnosis is high, and (2) challenges should be done in a hospital setting so that the patient can be adequately managed in the event of anaphylaxis. [1],[2]

The study by Skripak et al. on 807 children with IgE-mediated CMA showed rates of resolution were 19% by age 4 years, 42% by 8 years, 64% by 12 years, and 79% by 16 years. Children with persistent CMA had high specific IgE levels at all age groups (>50 kUA/L in 30%), especially in those with coexistent allergic rhinitis (40%) and asthma (49%). [3] A cow-milk specific IgE >100 kUA/L with anaphylaxis episodes at 12 years means our patient has little chance of outgrowing the allergy, and therefore, counseling is important so that the patient understands the nature of this disorder. Not only reading labels of packaged foods is important but also ensuring that the correct question is asked (i.e., is there milk in this food?) or this

information is sought for before consuming foods that may not obviously seem to contain animal milk proteins.

Animal models of allergy also provide clues to development of tolerance such as the research by Kerperien et al. on induction of cow's milk tolerance with short-chain oligosaccharides in whey-sensitized mice, [4],[5] but reports to the contrary also exist when children had developed anaphylaxis with similar short-chain oligosaccharides. [6] Nilsson et al. published data on 5 children with severe milk allergy (cow milk-specific IgE 30-160 kUA/L) who were desensitized using prolonged treatment with omalizumab (anti-IgE) injections and oral challenge performed but only after the in vitro basophil activation studies (against casein) had become negative. [7] The authors recognize the significant risks associated with such studies as with peanut immunotherapy trials where children required hospitalization. [8] Such trials can be done only in centers with adequate expertise and significant amount of funding is in place to cover treatment costs of biological therapies such as omalizumab including recourse to intensive care unit facilities when required for serious reactions. No attempt at oral immunotherapy to cow's milk was made in our patient.

Conclusion

Persistent food protein allergy may cause life-threatening allergic reactions and inability to develop tolerance. Patient-specific management plans should be developed with a list of 'strictly avoid' items and how to treat allergic reactions and anaphylaxis episodes.

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Conflicts of interest

There are no conflicts of interest.

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