

Case Report



Goat's milk allergy in a family following household sensitization to goat's milk soap

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Received: Jan 30, 2021

Accepted: Apr 19, 2021

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Conflict of Interest

The authors have no financial conflicts of interest.

Author Contributions

Conceptualization: AA. Data curation: AA. Investigation: AA, KR. Methodology: AA. Project administration: AA, KR. Writing - original draft: AA. Writing - review & editing: AA, KR.

ABSTRACT

Epicutaneous sensitization to food allergens is a recently recognised phenomenon. However, there is less understanding around necessary or sufficient cofactors that permit sensitization through this route. In this report, we describe the development of goat's milk allergy in members of a household with pre-existing atopic dermatitis, following use of a goat's milk soap marketed for sensitive skin. Soaps appear to be an effective vehicle for sensitization to food antigens and the risks are potentiated in those with compromised barrier function. Although such products may be marketed for atopic dermatitis, we would advocate for the avoidance of food-based skin care products in this patient group.

Keywords: Food allergy; Epicutaneous sensitization; Atopic dermatitis; Soaps

INTRODUCTION

Epicutaneous sensitization to food allergens is a recently recognised phenomenon. Food-containing soaps marketed for eczema can serve as particularly effective vehicles for sensitization as they desiccate and deliver food antigens to surfaces with already compromised barrier function. Here we describe the first report of multiple members of a household developing goat's milk allergy after exposure to goat's milk soap.

CASE REPORTS

Two brothers from the same household were sequentially referred to our allergy service after systemic allergic reactions to goat's milk cheeses. The first case, aged 17, experienced a generalised allergic reaction characterised by widespread urticaria, tongue and facial angioedema and shortness of breath within 15 minutes of ingesting halloumi chips derived from goat's milk. This reaction required Emergency Department attendance and treatment with intramuscular adrenaline. One week prior to this reaction, he experienced milder symptoms of throat pruritus upon ingestion of the same halloumi chips, however the symptoms did not progress further on that occasion. The patient continued to tolerate cow's milk products and confirmed that he last tolerated goat's milk cheese one month prior to his systemic reaction.

His past medical history was significant for honey bee anaphylaxis and atopic dermatitis. On specific questioning, it was ascertained that the household had started using a new goat's milk body soap for the preceding 12 months. The soap, which was marketed for sensitive skin and formulated with 0.5% goat's milk (*Ingredients: Sodium Palmate, Sodium Palm Kernelate, Aqua, Sodium Gluconate, Goat Milk Powder, Palm Acid, Glycerine, Butyrospermum Parkii, Sodium Chloride*), had been purchased to treat the atopic dermatitis that affected multiple members of the family. Prior to his systemic reaction, the patient reported the development of urticaria upon contact with the soap but still continued to use it intermittently. Investigations revealed normal acute and baseline tryptase (4.4 µg/L and 6.4 µg/L, respectively) and very high levels of specific immunoglobulin E (IgE) to goat's milk and sheep's milk protein (each >100 kU/L) (ImmunoCAP, Phadia, Uppsala, Sweden). As the patient had never reacted to goat's milk prior to experiencing local irritation on contact with the goat's milk soap, it was suspected that sensitization to goat's milk occurred through its application and the family were advised to discontinue its use.

Three months later, his brother, aged 22, was referred following a generalised reaction to goat's milk feta cheese. Within minutes of ingesting a gozleme containing feta and spinach, he developed throat pruritus and generalised urticaria. He received treatment in an Emergency Department with corticosteroids and antihistamines; adrenaline was not administered and serum tryptase was not measured at that time. His past medical history included atopic dermatitis and allergic rhinitis. Investigations demonstrated a high-normal baseline serum tryptase of 10.4 µg/L and very high serum specific IgE to goat's milk (>100 kU/L) and sheep's milk protein (94.2 kU/L). Cow's milk specific IgE was low positive at 0.42 kU/L but dairy was maintained in his diet. Although he had discontinued use of the goat's milk soap at the time of his brother's diagnosis, he also reported the occurrence of local erythema, urticaria and pruritus upon its application.

The boys' mother, who was the only other member of the household who had used the soap, also reported local symptoms (pruritus and erythema without urticaria) on contact with the soap after 12 months of daily application but had no pre-existing eczema. She continued to tolerate ingestion of goat and sheep's milk and there was no evidence of sensitization to either goat or sheep's milk protein on specific IgE testing. Of note, the identical twin of the younger brother, who also had atopic dermatitis but lived away from home and had not been exposed to the soap, had no symptoms on ingestion of goat and sheep milk products.

Written consent was obtained from the patients presented in this case in December 2020.

DISCUSSION

Epicutaneous sensitization to food antigens is now recognised as a mechanism in the pathogenesis of food allergy. Recognition of this phenomenon stemmed from the observation by Lack and others, that some children experienced clinical allergy on first ingestion of a food and were therefore potentially sensitized via an alternate route [1, 2]. This hypothesis was supported by a large epidemiological study, which examined risk factors for the development of peanut allergy and found that children with a positive peanut challenge were significantly more likely to have been exposed topically to products containing peanut oil [3]. Although this mechanism of sensitization appears established, there is less understanding around necessary or sufficient cofactors that allow sensitization through this

route. Murine models of adjuvant-independent epicutaneous sensitization have required prolonged, repeated antigen exposure using occlusive dressings applied to abraded skin [4]; whereas the use of an adjuvant abrogated the requirement for skin irritation and prolonged exposure [5]. Review of published case reports and small series have highlighted common factors involved in epicutaneous sensitization to food antigens, including prolonged, repetitive exposure (usually occupational) and the presence of underlying eczema or cutaneous irritation [2, 6-8]. Of particular interest was the outbreak of hydrolysed wheat protein (HWP) allergy affecting close to 2000 individuals in Japan between 2004–2010 linked to the use of a single brand of cosmetic soap containing HWP. This affected a number of individuals without underlying eczema and demonstrated that particular modes of antigen delivery (i.e., soaps and detergents) could be potent sensitizers [2, 6, 9]. It was also hypothesised that alteration of natural wheat proteins by hydrolysis produced novel epitopes to which humans had not previously been exposed through oral ingestion [2].

The case of this family is instructive as it serves as a human model of epicutaneous sensitization to food antigens involving the same trigger among genetically similar individuals exposed to the same environment. Within this model it is possible to examine a number of the cofactors highlighted by previous case reports. Although association of goat's milk allergy with the use of the soap in this family does not equate to causation, there are a number of clinical factors that support this as the aetiology. Firstly, the important negative control, the monozygotic twin of the index case, was not exposed to the goat's milk soap and did not develop clinical allergy. Secondly, the sequence of events that was common to both allergic cases, consisting of soap exposure, followed by contact symptoms, followed by clinical allergy, suggests a pathogenic process. This progression was also illustrated by Inomata et al. [7] in a cohort of 15 patients who experienced contact urticaria, which preceded the development of food allergy following occupational epicutaneous sensitization. Finally, both patients had tolerated oral ingestion of goat's milk in the months prior to the development of cutaneous symptoms. Compared with the occupational sensitization series, the time from exposure to onset of cutaneous symptoms in our cases was relatively short (2 and 4 months for cases 1 and 2 respectively, cf. >12 months for 15/15 patients in Inomata's series) [7]. A possible explanation for the more rapid onset of sensitizing symptoms in our cohort was the mode of antigen delivery in a soap, which contains surfactants capable of disrupting the stratum corneum and allowing for the penetration of larger antigens [2]. Although the onset of cutaneous symptoms was not documented in all cases of soap-induced HWP allergy, a proportion of these also had relatively rapid sensitization (<1 month) [6].

The role of underlying atopic dermatitis in the facilitation of epicutaneous sensitization is neatly illustrated in our cases. Both patients who developed allergy on ingestion of goat's milk had moderate underlying eczema. Their mother, who was unaffected by atopic dermatitis, has not developed evidence of allergy on ingestion of goat's milk in the 18 months since her sons' diagnoses. This observation, supported by other reported cases of epicutaneous sensitization [3, 7, 8], suggests that eczema is an important risk factor for this route of sensitization. The aetiology of her reported pruritus and erythema (notably without urticaria) in the absence of goat's milk sensitization is unclear but a possible explanation was the development of dry skin resulting from daily soap application.

Soaps appear to be an effective vehicle for sensitization to food antigens and the risks are potentiated in those with underlying atopic dermatitis. Although such products may be

marketed for this indication, we would advocate for the avoidance of food-based skin care products in this patient group.

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