Anaphylaxis to camel milk in an atopic child

Article *in* Allergy · December 2010

DOI: 10.1111/j.1398-9995.2010.02421.x · Source: PubMed

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etiological, irrespective of modes of inheritance (5). Patients with type 1 HIES have abnormalities in multiple systems of the body, whereas patients with type 2 HIES have abnormalities confined to the immune system. As for our patient, she has both the characteristic of the two types.

The patient was treated with 500 mg ceftizoxime twice a day for pneumonia and lamivudine 100 mg/day for antiviral therapy. Topic therapy included cryotherapy, curettage and mouthwash with sodium bicarbonate. After 1 week, her oral leukoplakia symptoms disappeared, and fungal examination was negative. Chest X-ray review was normal. The number of molluscums reduced after 1 month. The patient is in follow-up now.

In summary, our report shows that HIES not only is an immunodeficiency but also is a multisystem disorder that necessitates thorough medical evaluation for diagnosis and management of previously unrecognized associated diseases.

This work was supported by the National Science Foundation of China (30700726, 30671887).

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Accepted for publication 31 March 2010
Allergy 2010: 65:1622–1623

DOI: 10.1111/j.1398-9995.2010.02401.x

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Anaphylaxis to camel milk in an atopic child

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Keywords: Camel milk; anaphylaxis; milk allergy; protein allergy; shock.

Camel milk has distinct features and compositions, which presumably make it hypoallergenic (1–3). It has a white opaque color, a faintly sweetish odor and a sweet but sharp taste (2). It is thinner than cow or buffalo milk. Its protein epitopes differ from other sources of milk, and they denature at lower temperatures (4). It lacks β-lactoglobulin, a main allergen in cow milk. Its whey proteins include albumin, α-lactalbumin, immunoglobulins, lactophorin and peptidoglycan recognition protein (3).

Anaphylaxis to Camel Milk.

Many physicians recommend camel milk for children with cow milk protein allergy (5–8). The purpose of the report is to describe severe anaphylaxis to camel milk in a 6-year-old atopic child.

This 6-year-old boy had the sudden onset of cough, wheezing, itching and facial swelling very soon after consuming camel milk. The milk was fresh, directly from a camel in the farm and had no additives. It was warmed (not boiled) and then allowed to cool down before the consumption. On arrival to the emergency department, he was unresponsive and had severe respiratory distress with cyanosis. He needed cardiopulmonary resuscitation and mechanical ventilation. He immediately received epinephrine, hydrocortisone, salbutamol, aminophylline and magnesium-sulfate. His laboratory tests were all unremarkable. He was extubated within 24 h of admission. He went home on the 5th day of admission on a weaning dose of prednisolone and inhaled bronchodilators. He was supplied with a dose of epinephrine auto-injector. His discharge diagnosis was anaphylactic shock because of camel milk protein.

The only food he had tasted on that day (since arrival to the farm) was the camel milk. He was not exercising and he had no concomitant food. This was not the first time he tasted camel milk. His mother lately noted that he was deferring from camel milk because it caused perioral itching. These symptoms were associated with consumption of fresh as well as bottled camel milk.

His past medical history was remarkable for asthma, eczema (Fig. 1) and food allergies. He was allergic to peanut, multiple tree nuts, coconut and sesame. These products produced facial angioedema. He never had allergy to cow milk. He had no documented allergies to medications or insect bites. He had four emergency visits and three hospital admissions (one to an intensive care unit) for asthma exacerbations. He was on fluticasone proprionate/salmeterol xinafoate inhaler until 1 month prior to this life-threatening anaphylaxis. He never had anaphylaxis in the past.

He had five siblings, two with asthma. His father also had asthma. His mother had allergic rhinitis, contact dermatitis and allergies to local anesthetics.

Skin prick tests were performed on day 28, about 3 weeks off steroids and antihistamines. He was not taking any medication. A bottled camel milk (without additives) of the same herd (locally known as 'Azni') produced 8-mm induration and 30-mm erythema (Fig. 1). Cow milk protein reagent (Allergopharma®) and bottled cow milk

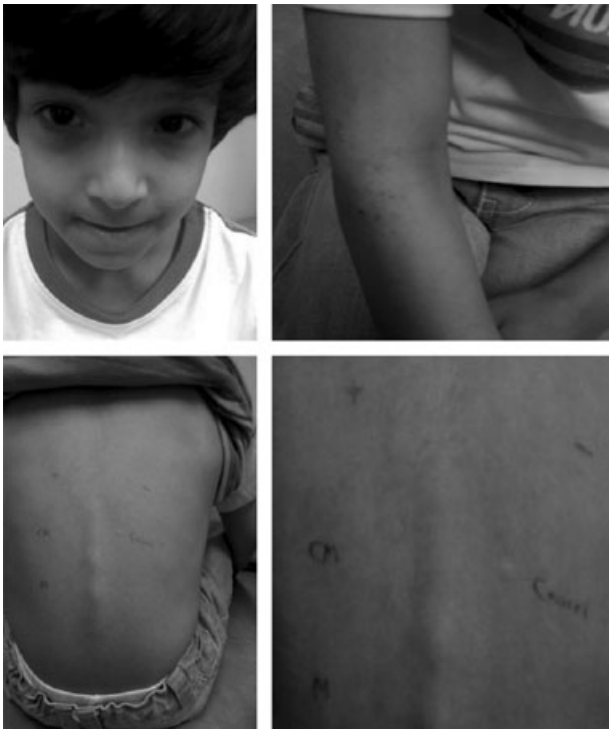

Figure 1 *Upper panels*, allergic shiner on the face of the patient (left) and atopic dermatitis around the elbow joint (right). *Lower panels*, Skin prick tests. Left side (top to bottom), histamine as positive control (positive reaction), cow milk protein reagent (negative reaction) and bottled cow milk (negative reaction). Right side (top to bottom), negative control (negative reaction) and bottled camel milk (positive reaction).

produced no reactions. These results confirm an allergy to camel milk, but not cow milk. Radioallergosorbent tests revealed the following: peanut >100 kUA/l, soy 32.3 kUA/l, wheat 4.41 kUA/l and nut mixture positive. The results for egg, cow milk and fish were all negative.

The camel (*Camelus Dromedarius*) is of significant socioeconomic importance in many arid and semi-arid parts of the world (9). It is considered the most important livestock animal in Northern and Eastern Africa, as well as in deserts of the Arabian Peninsula. It is a multipurpose mammal, used for supplying milk, meat, hides and transport. Its milk is an important component of human diets in these regions. It is used by nomads in the deserts of Egypt, Sudan, Mauritania, Kenya, China and Kazakhstan for feeding their babies (6). Moreover, its consumption among urban population is increasing. For example, camel dairies are present in United Arab Emirates, Saudi Arabia, Mauritania and Kazakhstan, and camel milk and milk products are produced for placing in the markets (9).

Milk protein allergy affects 2–3% of infants worldwide (8). For these young children, replacement diets include extensively hydrolyzed or amino acid-based formulas (7). Presumably, heat and enzymatic treatments of these products alter the protein epitopes and lower the antigenicity. However, these biochemical modifications may produce nutritional losses (7, 10, 11). Soy- or rice-based formulas and camel milk could be considered as possible alternatives (5–7, 12).

The low quantity of β-casein and the lack of β-lactoglobulin have been linked to the hypoallergic feature of camel milk (6). In one study, IgE molecules from sera of children allergic to cow milk were capable of recognizing most parts of milk proteins from sheep, goat and buffalo, and weak cross-reactivity was observed with milk proteins from mares and donkeys, but none with camel milk (12). This finding supports the notion of using camel milk for milk protein allergy. Camel milk also ameliorates food allergy symptoms in children (5).

Selective allergy/anaphylaxis to cow, sheep or goat milk without adverse reactions to other milk proteins has been reported (13–16). This case is the first report of selective anaphylaxis to camel milk protein, a diary product that is advertized as hypoallergenic (a true 'hypoallergenic food' may not exist). Any protein could elicit sensitization leading to severe allergic reaction. Therefore, there is no universal hypoallergenic mammalian milk (17). Thus, camel milk should not be considered constantly safe. Furthermore, allergenicity should be considered on clinical grounds rather than solely on *in-vitro* testing. This approach is more likely to detect patients at risk of developing serious reactions.

Strict avoidance of camel milk products was emphasized. The child is doing well with the avoidance of camel milk. Adrenaline auto-injectors were prescribed, and emergency action plan was taught to the child and his parents.

Patient and parents were confirmed for proper dosage and usage of adrenaline auto-injector with a placebo device. The emergency plan included immediate use of adrenaline intramuscularly when the initial signs and symptoms occur, regardless of their severity.

Epinephrine (adrenaline) is the first-aid medication in all anaphylaxis guidelines (18). Fatalities in anaphylaxis usually result from delayed or inadequate administration of epinephrine.

Adjunctive medications include H_1-antihistamine (e.g. diphenhydramine), H_2-antihistamine (ranitidine), β_2-adrenergic agonists (sulbutamol/albuterol) and corticosteroids. Avoidance of the trigger is the cornerstone of preventing anaphylaxis secondary to a specific food (19).

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Accepted for publication 7 May 2010
Allergy 2010: 65:1623–1625

DOI: 10.1111/j.1398-9995.2010.02421.x

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Correlation between atopy and tuberculin/*Candida* skin test reactivity in a bacillus Calmette-Gue′rin–vaccinated cohort

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Keywords: atopy; *Candida* skin test; tuberculin skin test.

The prevalence of atopy including asthma, atopic dermatitis, and allergic rhinitis has increased in recent years all over the world, mainly in developed countries. Bacillus Calmette-Gu′erin (BCG) is among the group of vaccines capable of eliciting strong Th1 immune response and therefore decreasing risk of Th2-associated atopy. Shirakawa et al. (1) reported an inverse association between tuberculin response and atopic disorders. Some studies found conflicting results to support the inverse correlation between the tuberculin skin test (TST) and atopy. There were some diseases such as Kawasaki disease with enhanced Th2 expression but like an infectious disease clinically (2). The balance between Th1/Th2 responses seems to be more complex so as the relationship between the delayed-type hypersensitivity (DTH) and atopy.

Tuberculin skin test reactivity negatively correlates with atopic dermatitis/allergic rhinitis but not asthma.

We conducted to investigate the relationship between the DTH (including tuberculin and *Candida albicans* antigen) response and atopy at the age of 6 in a nonselected birth cohort infants who were all BCG vaccinated at birth (3). A total of 530 children were followed including physical examination, allergen test, and questionnaire at 6 years of age. Purified protein of tuberculin was administered by the Mantoux method using 0.1 ml (two tuberculin units) of purified protein derivative RT23 (Statens Serum Institut, Copenhagen, Denmark) and 0.1 ml *Candida albicans* (Green Laboratories, Lenoir, NC, USA) at 6 years of age by injecting intradermally on the volar surface of the forearm. The diameter of the indurated area was measured at 72 h. Atopy were defined as clinical diagnosis of asthma, allergic rhinitis, or atopic dermatitis, together with detectable serum-specific IgE levels (>0.7 KU/l in one or more allergens). The specific IgE directly against the common allergens in Taiwan, including *Dermatophagoides pteronyssinus*, cockroach, dog dander, seafood, or pollen mixture, was measured by using the PharmaciaCap system, as previously described (4). Questionnaires were asked on previous and recent (<6 months) symptoms of asthma, allergic rhinitis, or atopic dermatitis. Ever atopy was defined as atopy noted before but acquired remission at 6 years of age. Persistent atopy was referred to ever atopy before and still with atopy when studied. During the follow-up period, no patient had active tuberculosis disease or *Candida albicans* infection detected.