

Immediate Systemic Hypersensitivity Reaction Associated with Topical Application of Australian Tea Tree Oil

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ABSTRACT

Australian tea tree oil has been used as a veterinary antiseptic for many years and, more recently, has been extended into human use. There have been many reports of allergic contact dermatitis and toxicity reactions, but it has never been implicated in immediate systemic hypersensitivity reactions. A 38-year-old man experienced immediate flushing, pruritus, throat constriction, and lightheadedness after topical application of tea tree oil. Our purpose was to determine whether this represented an immunoglobulin E (IgE)-mediated reaction. Skin-prick and intradermal testing was performed, as well as enzyme-linked immunosorbent assays for specific IgG and IgE against tea tree oil. The patient had a positive wheal and flare reaction on intradermal testing with tea tree oil. All five patient controls were negative on skin testing. No specific IgG or IgE was detected. We present the first reported case of an immediate systemic hypersensitivity reaction occurring after topical application of Australian tea tree oil, confirmed by positive wheal and flare reaction on skin testing. (Allergy and Asthma Proc 24:73-75, 2003)

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Melaleuca alternifolia, or the Australian tea tree, grows indigenously in coastal and subcoastal regions of New South Wales and Southern Queensland as well as Portugal and Spain.¹ Tea tree oil is obtained from steam distillation of the plant's leaves and its use has been gaining popularity as a natural topical antiseptic in both animals and humans. The essential oil is also used in several household products such as cleansers and laundry detergents.² Several cases of contact dermatitis and toxicity reactions associated with oral ingestion have been described previously. We present the first reported case of a systemic anaphylactic reaction associated with tea tree oil.

CASE REPORT

A 38-year-old white man with a past medical history of psoriasis placed a drop of commercially available Australian tea tree oil on his finger and then applied this to psoriatic lesions on his legs. He noticed immediate tingling on his finger and immediate intense erythema to his legs at the site of application of the oil. Within minutes, he noted the sensation of throat constriction and observed a change in his phonation, as well as lightheadedness and pruritic, bright red spots diffusely scattered over his skin. He had not taken any medications, including aspirin or nonsteroidal anti-inflammatory drugs. There had been no recent food ingestion and he denied other illness. There was no latex exposure. He had not used tea tree oil previously; hair care products containing tea tree oil had been used. He was taken by ambulance to a nearby emergency room where his following vital signs were taken: temperature, 37.6°C; pulse, 87 beats/minute; respiratory rate, 20/minute; and blood

pressure, 150/73 mmHg. Pulse oximetry was 96% on room air. The patient was noted to have a flushed appearance to his face. The presence or absence of urticaria or angioedema was not noted in the emergency room record. He was treated with intravenous (i.v.) fluids, i.v. methylprednisolone, oral hydroxyzine, and i.v. famotidine. His vital signs remained stable and his pulse oximetry increased to 98% with resolution of the pruritus, shortness of breath, and sensation of throat closing within 2 hours. He was discharged from the emergency room on oral diphenhydramine and a prednisone taper with instruction to avoid any further use of tea tree oil. He did well and had no recurrence of symptoms.

INVESTIGATION

Approximately 1 month after the episode, the patient underwent tea tree oil skin testing with the commercial preparation that was implicated, as well as positive control with histamine and negative control with phenol saline solution. The tea tree oil was diluted with phenol saline solution, and he failed to have any reaction to prick testing at dilutions of 1:10,000, 1:1000, and 1:100, as well as with undiluted tea tree oil. He also failed to react to intradermal dilutions of 1:10,000 and 1:1000 but had a positive response within 20 minutes to the intradermal 1:100 dilution of tea tree oil (7-mm wheal with 20-mm flare). Five control subjects did not react to any dilutions of the preparation.

A tea tree oil extract was prepared by diluting the stock 1:1 (v/v) with phenol saline solution. It was stirred overnight, with subsequent removal of the oily supernatant. A protein assay using a modification of the biuret method³ was performed and the protein concentration was determined to be 0.28 mg/mL. An enzyme-linked immunosorbent assay (ELISA) was performed for both specific immunoglobulin E (IgE) and IgG against tea tree oil but neither antibody type was detected.

DISCUSSION

Australian tea tree oil, derived from the steam distillation of the leaves of the myrtle tree *Melaleuca alternifolia* is marketed as a veterinary antiseptic⁴ and has been extended into human use. It has been shown to inhibit the growth of a variety of bacteria and fungi.⁵ These antiseptic properties are potentiated by the highly lipophilic nature of the oil, which leads to enhanced dermal absorption. The most common clinical signs following skin exposure in animals are ataxia, incoordination, weakness, tremors, behavioral disturbances, and depression.⁴ The majority of these have been reported as occurring 2–8 hours after inappropriate exposures. Similar symptoms have occurred in dogs and cats exposed to terpenes (d-limonene and linalool) and, in fact, tea tree oil contains 50–60% terpenes.⁴ Similar toxic reactions due to ingestion of tea tree oil also have been reported in humans.^{6,7} Central nervous system

symptoms may occur after ingestion of >1 oz of tea tree oil. The minimum lethal human dose is unknown and there is no known antidote.⁷

There also have been several reports of both irritant and contact dermatitis due to tea tree oil. Australian standards require that cineole, a major ingredient of tea tree oil and other essential oils be present at <15%, because higher amounts of cineole can cause skin and mucous membrane irritation.⁴ Most of the cases of allergic contact dermatitis have involved application of the tea tree oil to already broken or irritated skin.^{1,2,8,9} Patch testing has revealed the most common sensitizer to be d-limonene, followed by aromadendrene and α -terpinene.⁸ One report of airborne allergic contact dermatitis was reported in a man with a known turpentine allergy who experienced a pruritic rash on his eyelids and face with subsequent spread to his trunk and extremities 2 days after inhaling the vapors of a tea tree oil solution.² An unusual nonpruritic rash was reported in a patient 1 day after swallowing one-half a teaspoon of tea tree oil. This later proceeded to exfoliation, neutrophilia, and petechiae,¹⁰ inconsistent with an immediate hypersensitivity reaction.

To our knowledge, this is the first reported case of immediate systemic hypersensitivity reaction occurring after topical administration of a small amount of tea tree oil. This appears to be an anaphylactic reaction because skin testing was positive with a dilute solution of the tea tree oil, and five controls did not react at the same dilution, thus making an irritant reaction unlikely. Unfortunately, we were unable to detect specific IgE or IgG against tea tree oil by ELISA. We postulate that this may have been caused by the preparation's oily consistency, resulting in difficulty maintaining protein adherence to the ELISA plate. Other investigators have reported technical difficulty in performing antibiotic susceptibility testing because this property of tea tree oil as well.⁵ It is also possible that this was an anaphylactoid reaction, although the presence of the positive skin test argues against this hypothesis. Finally, it is possible that his lightheadedness and throat constriction symptoms were anxiety related.

In conclusion, the medical community needs to be aware of the potential for a serious reaction to Australian tea tree oil. This concern can be extended to other "natural remedies" made from plant products. As the level of interest in and use of these types of products increases, it is expected that they will be increasingly implicated in serious and even life-threatening allergic reactions.

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