

Toast-makers' fingers

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Key words: irritant contact dermatitis; caterers; occupational; heat; friction; margarine; toast. © Munksgaard, 1998.

Case Report

A 55-year-old woman was referred with worsening hand eczema of 22 months duration. She had worked in a canteen for 30 years, having the occasional episode of eczema affecting the dorsum of her hands alone. 2 months prior to the onset of the recent eruption, she changed to a different workstation and became responsible for "buttering" approximately 200 slices of toast in the mornings in under 1 h. She was right-handed and took the toast from the toaster with this hand, holding the slice, prior to spreading with margarine, in her left. She developed problems with her palms and fingertips of both hands. There was minimal exposure to onion and none to garlic. She noticed that her hands improved when on holiday and at the weekends. There was no history of atopy. Hormone replacement therapy had been started 5 years earlier. On examination, she had circumscribed fissured scaly patches on the fingers and palms, with accentuation on the pulps of the fingers. There were associated hyperkeratotic patches on the palms. She was patch tested to the European standard series and fresh onion but showed no relevant positive reactions. She was advised to use hydrocolloid dressings on her fingertips for protection and gloves whilst at work, but found these measures impractical.

Discussion

Contact dermatitis on the palmar surfaces of the hands in caterers is well-described (1). This patient was exposed, in the time-pressured environment of a busy can-

teen, to both mechanical and thermal injury. Toast is hard and rough, a type of surface described before as causing palmar dermatitis (2). The lowest temperature at which a thermal burn can occur has been estimated as 44°C (3). The surface temperature of fresh toast from a 2-element toaster measured with a probe (Comark Ltd, KM22 Foodcheck thermometer) at 0 s is 140°C and at 30 s is 77°C (average of 10 readings taken in a room at an ambient temperature of 20°C). Chronic exposure to infrared radiation results in erythema ab igne. Further exposure can cause hyperkeratotic nodules along with skin changes resembling those seen in chronic actinic exposure. After 20 to 30 years, skin cancers can arise, from which caterers are known to be at risk (4). Essential oils used in cooking can act as irritants or allergens (5). It is possible that melted margarine could act in the same way over long exposure.

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Allergic contact dermatitis from kojic acid

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Key words: allergic contact dermatitis; kojic acid; medicaments; depigmenting agents; bleaching agents; hyperpigmentation. © Munksgaard, 1998.

Case Report

A 30-year-old woman had undergone sclerotherapy for varicose veins, following which she had developed hyperpigmentation. She was prescribed a cream containing 10% urea and Kojicol Plus® (hydroquinone 2%, lac-

tic acid 4%, hazel 74%, castor oil 5%, citric acid 1%, cellulose 1%, propylene glycol 10%, and kojic acid 3%). After applying this cream for 4 months, she noted no improvement and so Angiogel® was added (a mixture of melilotus, alpha bisabolol, *Ginkgo biloba* extract and ascorbic acid). A few weeks later, she presented with an

eczematous eruption on and around the hyperpigmented areas.

Patch tests with the GEIDC standard series proved negative. A patch test with the entire Kojicol Plus® product was ++ after 4 days. The patient was later tested with all the individual components of the product, including hydroquinone 1% pet. and with aqueous kojic acid solutions of 5%, 1%, 0.5% and 0.1%. All the patches with kojic acid were positive after 2 and 4 days (++ to 5% and 1%). Patch tests with the other components of the product were negative. 20 controls tested with the same kojic acid solutions were negative. We recommend 1% aq. for patch testing kojic acid.

Comment

Kojic acid (5-hydroxy-2-(hydroxy methyl)-4-pyrone) is a fungal derivative produced by certain species of *Acetobacter*, *Aspergillus* and *Penicillium*. It inhibits the production of free tyrosinase (1, 2). The Ames test shows that it is mutagenic for bacteria, it causes the production of certain toxins, and is toxic by ingestion in male broilers

(3–5). This case confirms that it is also a potential contact sensitizer (6).

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Trimethoprim-induced fixed drug eruption: positive topical provocation on previously involved and uninvolved skin

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Key words: fixed drug eruption; trimethoprim; positive patch test; adverse drug reactions; antibiotics. © Munksgaard, 1998.

Case Report

A 42-year-old Caucasian woman presented with 2 well-demarcated, round to oval shaped, violaceous macules of 3-cm diameter on the interscapular area. This was her 3rd attack in 12 months. Lesions lasted for 8–10 days and healed with residual hyperpigmentation. She had a history of cotrimoxazole intake on the day before the eruption had begun. Histopathological examination confirmed the diagnosis of fixed drug eruption (FDE).

Within 1 week of withdrawal of cotrimoxazole, lesions cleared, leaving residual hyperpigmentation (Fig. 1). 4 weeks later, an oral challenge with $\frac{1}{8}$ of a single cotrimoxazole dose was performed. The previously-involved FDE sites reactivated completely, with marked violaceous erythema and edema starting within 1 h. There was no additional skin involvement. 4 weeks later, tape-stripped occlusive patch testing was performed. Trimethoprim and sulfamethoxazole at concentrations of 10%, 20% and 50% pet., and petrolatum as the negative

control, gave no reaction on previously involved and uninvolved skin of the back. After 2 weeks, open topical testing with the drugs at the same concentrations in dimethyl sulfoxide (DMSO), and pure DMSO as the negative control, was performed. Solutions were applied both to previous FDE lesions and to unaffected skin sites of the back every 12 h up to 4×. As there were only 2 areas of skin affected by the FDE, open tests were performed at 1-week intervals on these sites. A positive reaction was seen on both the previously involved and uninvolved skin sites with trimethoprim 20% and 50%, starting approximately 1–2 h after the 1st application of the test preparations and progressing into itchy edematous plaques within 12 h (Fig. 2). The reaction lasted more than 24 h. Histopathological examination of the previously uninvolved test site revealed sparse dyskeratosis, mild edema and mononuclear perivascular inflammatory infiltration, including a few eosinophils in the papillary dermis. Repeated open tests with trimethoprim at concentrations of 20% and 50% in DMSO in 15 healthy controls remained negative.