

Killing mites with heat

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Key words: house-dust mite; heat; thermal death point.

● HOUSE-dust mites may be effectively killed by high temperatures (1–3). We sought to investigate the effects of various forms of heat deliverance to household items at varying temperature upon the survival of house-dust mites.

Cultures of *Dermatophagoides pteronyssinus* were used in four separate sets of experiments. In experiment 1, mites were plated in Petri dishes, and the effects of dry heat (incubator heat) and wet heat (hot water) at various temperature settings (40, 50, 60, and 70°C) at 10, 20, 30, and 40 min were examined. In experiment 2, the effect of exposure to dry heat upon the behavior of mites placed in polyester membrane was examined. In experiment 3, we examined the effect of dry heat applied by ironing upon live mites placed on a cotton sheet and on a comforter of 2.5 cm in thickness. Temperatures at various fiber layers of the comforter were measured. Mites were examined at various layers of comforter fiber layer. Experiment 4 was set to examine the effect of sunlight and of vacuum-cleaning upon live mites seeded on to a straw mat. The survival of mites was determined by examining their shape and mobility. All experiments were performed in triplicates.

The combination of a temperature of 50°C or over with a duration of 20 min or more was sufficient to cause 100% death of mites by dry heat. For wet heat, a temperature of 60°C or over for 20 min or more was required to kill all mites. Mites were found to migrate into deeper layers of

polyester fiber in response to dry heat. Dry heat applied via ironing (40°C for 5 s) killed all mites on a cotton sheet and on the top two fiber layers of a comforter. Mites could survive this process in deeper layers of the comforter (40% and 93% in the third and fourth fiber layers). Direct sunlight for 6 h, vigorous shaking, and vacuum-cleaning of the straw mat were not effective to kill or remove mites from the mat since mites could hide in the cracks or cling very tightly to mat fibers.

In our experiments, dry heat seemed to be more effective than wet heat. With a hot wash of over 60°C, a duration of more than 20 min is necessary to kill all mites. Ironing is effective for killing mites on thin sheets, but not on thick clothes or comforters. A straw mat supports the survival of mites well and should not be used by mite-sensitive individuals.

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Exanthema to metamizole

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Key words: biopsy; fixed exanthema; metamizole; nonpigmented.

● FIXED exanthema is a fairly common dermatologic condition with repeated episodes of cutaneous inflammation appearing characteristically at the same sites (1). The responsible agent is a drug in 85% of reported cases. That constitutes 10% of all adverse drug reactions.

The drug most frequently involved is trimethoprim-sulfamethoxazole. Nonsteroidal anti-inflammatory agents are known to cause adverse drug reactions. Fixed exanthemas

caused by metamizole have already been reported

First case of nonpigmented fixed exanthema.

(2–4).

There is a nonpigmented form of fixed exanthema. It is a distinctive entity, as proposed by Abranowitz & Noun in 1933 (5). It has no residual pigmentation or any other trace. The epidermis is not disturbed, and thus no pigmentation occurs. In this case, the dermis is the primary site of the drug sensitivity response. Cases are reported to have been caused by sensitivity to ephedrine and pseudoephedrine (6–8).

We report a case of nonpigmented fixed exanthema caused by Nolotil® (metamizole). In our case, histologic features revealed new data.

A 58-year-old woman with arterial hypertension controlled with a 5-mg dose of amlodipine (Astudal®) once a day was referred to our clinic for a cutaneous disorder. In the last two years, she had showed oval, well-defined, nonitching erythematous lesions with slightly desquamative surface. These lesions

recurred in the same areas of the skin: one on the external face of the right ankle, four on the right flank, and one over the right upper lip. They disappeared within 15 days without postinflammatory hyperpigmentation.

She remembered taking one Nolotil capsule the night before the last episode.

We performed the skin prick test and the intradermal test with Nolotil at 1:1000 and 1:100 concentrations with negative results. Epicutaneous patch tests were performed on unaffected and previously affected skin. The drug was prepared at 10% concentration in petrolatum. These tests also proved negative.

After written informed consent had been obtained, the patient underwent an oral challenge test with a single dose of 250 mg of Nolotil. She developed the same lesions in the same sites after 8 h. Cutaneous biopsy of one lesion from the right flank was performed. It showed local changes of epidermal acanthosis and parakeratosis, edema in the papillary dermis, and an inflammatory subpapillary infiltrate with lymphocytes and eosinophils.

To our knowledge, this is the first reported case of nonpigmented fixed cutaneous exanthema due to Nolotil (metamizole). Despite the epidermal changes, there was no residual pigmentation. It is possible that initial episodes of fixed exanthemas occur without pigmentation, and they might be imputed to other causes. Thus, we may assume that fixed exanthemas are underdiagnosed, and it is in later episodes when residual pigmentation appears that we achieve the correct diagnosis.

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Anaphylactic reaction to formaldehyde

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Key words: anaphylaxis; dental treatment; formaldehyde; urticaria.

● It is known that (para-)formaldehyde and paraformaldehyde, which are commonly used as denture base material, can cause severe anaphylactic reactions in patients with dental treatment (1–3). A total of 14 cases has been reported, but the presented diagnostic criteria are contradictory (1–6). Specific IgE antibodies against formaldehyde or formaldehyde-human

serum albumin (F-HSA) conjugates were detected in eight patients. Four of the patients had a positive patch

IgE sensitization to root canal dental paste.

test, whereas in only two patients was a positive scratch test reported. Prick tests in all of the patients were negative (Table 1) (7). We describe a patient with anaphylactic symptoms after the second application of the root canal paste Toxavit, which contains paraformaldehyde.

A 31-year-old man with no personal or family history of allergies was referred to our outpatient clinic with a massive swelling of his face after a second endodontic treatment. He had had his first contact with Toxavit about 6 weeks before. This formaldehyde-containing tooth-filling material, which is used for devitalization of the tooth pulp, had to remain in the tooth for 6 weeks. Approximately 5 h after the second treatment with Toxavit, our patient developed an anaphylactic reaction (urticaria) with associated cardiovascular symptoms, but without unconsciousness. He received immediate medical help and recovered within 1 day. Our patient was a nonsmoker and did not take any medications at all, not even local anesthetics.

Allergy testing showed a highly positive RAST (Pharmacia and Upjohn, Sweden) to formaldehyde (CAP-FEIA class 3, total: 3.6 kU/l), whereas total IgE was within the normal range (29.7 kU/l). Prick testing with formaldehyde in aqueous solution up to 1% revealed no reaction after 30 min or after 24 h, but an erythematous papule appeared on day 2. Patch testing with Finn Chambers on Scanpore also revealed an erythematous reaction to aqueous formaldehyde (1.0%) but was otherwise uneventful. Although the exact mechanism of sensitization to formaldehyde in our patient is unknown, our results seem to support the notion of contact sensitization leading to a type-I as well as a delayed-type hypersensitivity.