

Short Communication

Occupational heat contact urticaria

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Heat-induced local urticaria (HLU) is a rare physical urticaria, recognizable by the formation of wheals in the areas where heat is applied. About 40 cases have been published (1), including the familiar delayed variant (2).

Case Report

A woman aged 40 years, with atopic rhinitis, presented with a 1-year history of pruriginous wheals, occurring only in areas in contact with hot objects, during her work as *churros*-maker. *Churros* is a popular Spanish sweetmeat, made from a stiff batter squeezed out into sticks or rings and deep-fried, sprinkled with sugar and eaten for breakfast or as a snack, often accompanied by coffee or hot chocolate. She also reported lip oedema on eating hot food. There were no systemic symptoms.

We challenge tested with a glass tube at 36, 40 and 45°C for 4 min on her forearm, where pruritic wheals formed in the contact area, appearing sooner and larger at the higher temperatures. Tests for mechanical, exercise, sun-induced, and cholinergic urticaria were negative. The total serum IgE was 115 Ku/l (normal <100). A biopsy was taken of healthy skin and of the heat-induced wheal 10 min after the test. Under the light microscope, in the wheal biopsy, slight dermal oedema with vasodilatation and minimum perivascular lymphocyte infiltration was seen. Immunofluorescence was negative for IgG, IgM, IgA, IgE and λ and κ chains. On electron microscopical examination, degranulation of mast cells was not seen.

Testing for histamine release from basophils showed the absence of significant release both before and after heating the blood to 45°C for 5 min. The left hand and forearm were immersed in water at 45°C for 5 min, and erythema, oedema and itching occurred in the immersed area (Fig. 1). In blood from the cubital vein, obtained both before and 10 min after the immersion test, no significant changes were found in immunoglobulins (IgG, IgM, IgA, IgD and total IgE), complement (C1 esterase inhibitor, C1q, C2, C3, C4, C5, C9 and B factor), histamine, prostaglandin E₂, vasoactive intestinal peptide, circulating immune complexes or α antitrypsin values.

The patient was treated with oral antihistamines with no improvement. Local 0.1% capsaicin was not tolerated. She was advised to avoid contact with hot objects.

Discussion

Among the few cases of HLU reported, 75% were women, of whom 60% were atopic or had high serum IgE levels, and 40% also showed other physical urticarias (3). The pathogenesis of HLU is not yet clear. Allergic mechanisms were soon ruled out (4). Some authors have attributed an important rôle to histamine

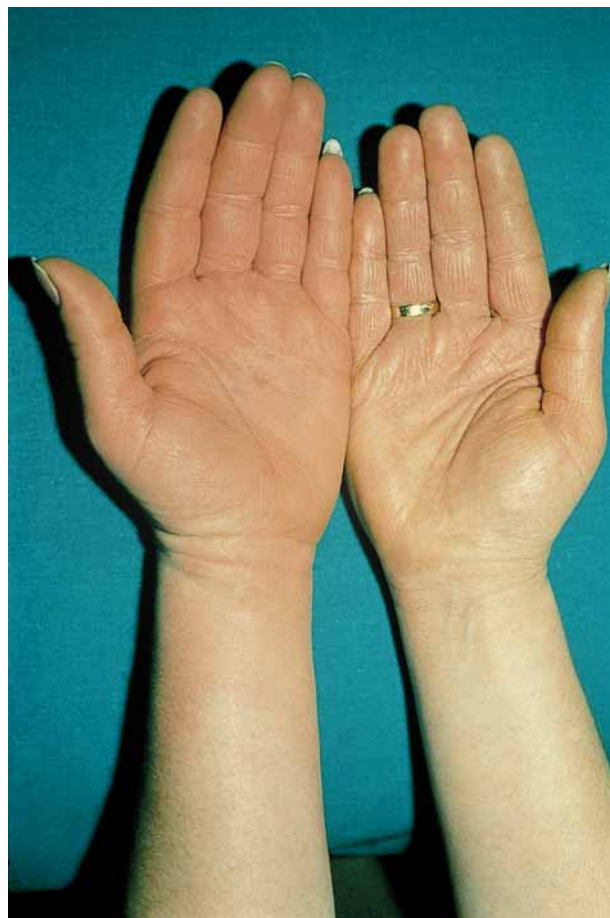


Fig. 1. Erythema and oedema in the immersed area.

(1, 3–10) and other mediators released from mast cells or basophils (2, 11), such as prostaglandin D₂ (3). Others have refuted the above mechanism, suggesting an essential rôle for complement activation (12, 13), although in some cases, complement consumption has not been proven (5–8, 14, 15). Involvement of the kininogen/kinine system has been proposed (16), and others suggest the involvement of sensory nerve endings once the wheals subside with the application of local anesthetics (17) or capsaicin (18). Classification of HLU into 2 major groups has been proposed: type I, mediated by histamine and mast cell degranulation products; type II, mediated by other factors (5). Some of the cases included in type I improve with antihistamines and/or desensitization (7, 19), but type II cases have a poorer therapeutic response. In our case, we could not prove any significant change in the mediators possibly involved, and degranulation of the mast cells was not seen. Heat urticaria should be considered as a potentially occupational skin disease.

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