

BRIEF COMMUNICATION

Localized heat and cold urticaria

Rare phenomena occurring in the same individual

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The phenomenon of localized heat urticaria is described and illustrated by the report of a case. The patient also exhibited localized cold urticaria. No definite immunologic mechanisms were defined for the association of these 2 unusual conditions.

The development of localized urticaria after exposure to heat is a very unusual clinical abnormality. It has been stated that too few cases have been described to allow for any generalizations.^{1, 2} Localized heat urticaria differs from cholinergic urticaria in being a localized response to heat; sweating and exercise do not cause hives in these patients, nor does local application of heat cause urticaria in classical cholinergic urticaria.

Evidence for heat-sensitive circulating antibody has been sought by means of passive transfer techniques in at least 3 patients by Baer and Harber² and by Delorme.³ In all 3 cases transfer was not demonstrated. Of particular interest was the observation by Delorme³ that the local urticarial reaction to heat could be blocked by prior infiltration of the site with local anesthetics.

Recently we studied a patient with this unusual manifestation who, in addition, developed localized urticaria after exposure to cold. These two manifestations have never been previously reported to occur together. This report concerns her case history and studies performed.

CASE REPORT

A 29-year-old white woman consulted us in May, 1971, with a 10 month history of urticaria that seemed to be associated only with heat exposure. She first experienced this phenomenon following exposure to the sun. The exposed areas would redden, swell, and itch for approximately 30 minutes after exposure. The interval from exposure to onset of the appearance of these angioedematous lesions varied from 15 to as long as 45 minutes. Direct contact with warmed objects resulted in the appearance of a classic localized hive, the shape of which resembled the contact area. She also noted that if she washed dishes in hot water, massive

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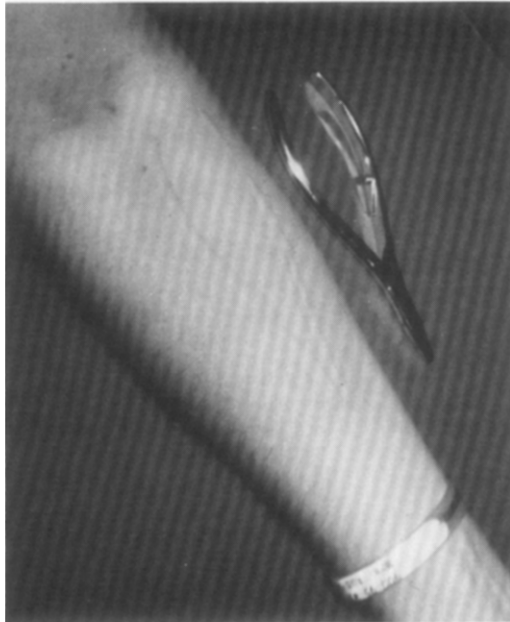


FIG. 1. This wheal and erythema reaction to heated metal nail clippers appeared after 5 minutes of local application to the forearm.

edema of the hands would develop within 5 to 15 minutes. In January, 1971, she developed itching in her throat and mild difficulty in breathing immediately following ingestion of hot soup. The ingestion of any moderately hot liquid would precipitate the same symptoms. Physical exertion, sweating, and emotional stress did not induce urticaria. On one occasion, a 3 or 4 minute exposure to very cold temperatures, i.e., freezer compartment of the refrigerator, resulted in edema of her fingers. This cold-induced reaction appeared within 30 minutes and lasted approximately one hour. She is nonatopic, and there is no family history of warm or cold urticaria or allergic conditions.

Physical examination was completely normal. Dermographism was not present. When a heated nail clipper was applied to the forearm, a wheal the shape of the nail clipper developed within 5 minutes (Fig. 1). There were no pseudopods. When an ice cube was applied to the forearm for 5 minutes, a localized wheal developed within 10 minutes.

Laboratory examinations for pyroglobulins, cryoglobulins, rheumatoid factor, lupus erythematosus factor, and VDRL were all negative. Skin tests for common inhalant and food allergens were negative. Serum immunoglobulin levels (IgG, IgA, IgM, IgD), total complement activity, and B₁A globulin (C3) activity all fell within normal limits. A complete blood count, urinalysis, electrocardiogram, blood urea nitrogen, creatinine, calcium, phosphorus, lactic dehydrogenase, alkaline phosphatase, serum glutamic oxalacetic acid, and chest roentgenogram were all normal.

A conventional P-K test (passive transfer) was performed in multiple sites on 2 recipients. Challenged at 3, 8, and 24 hours with both a warmed (45° C.) small glass of water and an ice cube, no reaction was observed in any instance.

A skin site infiltrated with 2 ml. of 1 per cent Xylocaine gave the usual urticarial reaction following a 10 minute challenge with a heated flask of water (45° C.). Prior infiltration with 0.75 ml. of Benadryl (50 mg. per milliliter) inhibited the response by approximately 30 per cent.

The minimal erythema dose of ultraviolet light from a conventional hot quartz source was normal.

COMMENTS

Localized heat urticaria is a rare disease characterized by the development of urticaria after exposure to heat. It differs from solar urticaria in that it is temperature-dependent with no relationship to specific light wave lengths. A challenge with a mixed spectrum of ultraviolet light did not induce an urticarial response in our patient. In addition to challenge with heat, this patient exhibited a classic localized urticarial reaction following an ice cube challenge test, although clinically the patient has had only minimal symptoms after exposure to severely cold temperatures. This patient did not exhibit a delayed urticarial response to heat, which has recently been described.⁴

Attempts at passive transfer of both the heat and cold urticarial reactions with the patient's serum were unsuccessful. In all previously reported cases of heat urticaria, similar results were found. In addition, her serum failed to transfer the cold urticarial response. Recently the passive transfer of cold urticaria has been accomplished with both IgE⁵ and IgM fractions.⁶

A number of hypotheses have been put forth as explanations of heat urticaria. Allergic mechanisms have been postulated. It has been suggested that mediator-releasing cells (mast cells) may be hyperreactive to heat, or that the vessel walls may hyperreact to the mediators. Delorme implicated a neurogenic mechanism in showing that the response could be blocked by Xylocaine. Our patient, however, responded perfectly well in the presence of Xylocaine anesthesia. None of the ideas currently under consideration can satisfactorily explain the urticarial response to heat that these patients exhibit. As this patient showed localized edema formation to both heat and cold challenge, we suggest that a temperature-dependent, hyperreactive, mediator-releasing cell best explains the mechanism in our case.

Management of patients with localized heat urticaria is directed at avoidance of contact with heat. Antihistaminic therapy, although usually not beneficial, should also be tried. Based upon the recent reported by Wanderer and Ellis⁷ that treatment with cyproheptadine in cold urticaria resulted in a marked amelioration of symptoms, we treated our patient with Periactin, 4 mg. 3 times a day. This therapy was unsuccessful, as was therapy with hydroxyzine, 50 mg. 4 times a day.

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