

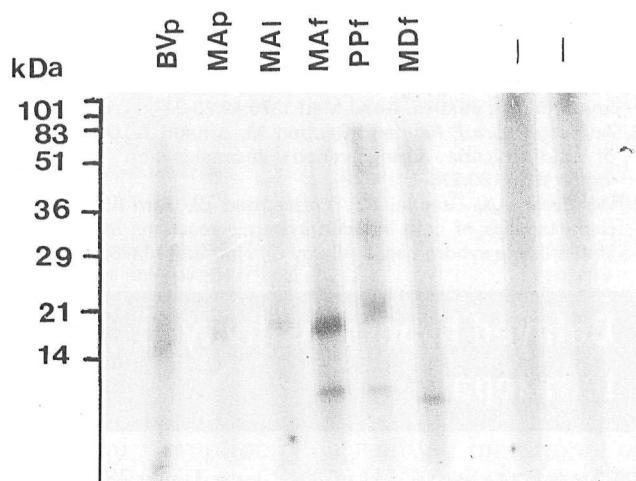


FIGURE 1 SDS-PAGE immunoblotting.

incubated with the patient's serum diluted to 1/3 with 5% BSA in PBS. Then this was incubated with 200 000 cpm of radioactively labeled human anti-IgE, and autoradiography was performed. As negative control (-strips), MDf and PPf extracts incubated with diluent instead of the patient's serum were used (Fig. 1). The more evident results were obtained with the three fruit extracts (MDf, PPf, and MAf). For MAf, two distinct allergenic bands were obtained with pm of about 18 and 10 kDa. The first band (18 kDa) was also detected with the BVp, MAI, and PPf extracts and less clearly with MAP. This is probably in accordance with the Bet v 1 allergen and homolog proteins. There was also a second band of 10 kDa in the MAf, MDf, and PPf extracts which did not seem to correspond to prophylines. In this respect, it has been reported that the most abundant allergen in peach skin has a pm of 10 kDa (5).

As a preliminary conclusion and contrary to what usually happens (6), this case record could be clinically explained as an initial sensitization to fruits (Rosaceae), developing specific IgE against 18- and 10-kDa proteins, which later gave rise to the symptoms after exposure to MA leaf or fruit and to skin sensitization to *Betula* pollen, an effect that usually passes unnoticed in our environment.

Key words: asthma; food allergy; cross-reactivity; *Morus alba*; mulberry.

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Wasp sting-associated cold urticaria

THE development of urticaria in certain subjects after exposure to cold was reported as early as 1866 Bourdon (1). The disorder is characterized by the development of urticaria and/or angioedema and sometimes painful burning plaques after exposure and may be classified as primary and secondary. A great variety of disorders have been described in the literature as associated with this condition, the most common being malignancies, systemic leukocytoclastic vasculitis due to cryoglobulinemia, and various infectious diseases. We describe a patient who developed cold urticaria (CU) during three periods of her life, each time

A patient with repeated episodes of cold urticaria after wasp sting.

after having been stung by a wasp, suggesting a causal relationship between wasp stings and CU.

A 44-year-old woman without a family history of CU presented at our department with complaints of hives appearing upon exposure to cold. She had experienced the same symptoms after cold water or rain exposure 26 years before after having been stung by a wasp. The symptoms resolved spontaneously after a few months until 11 years ago when she was restung by a wasp. The symptoms of CU recurred, worsened during the winter, and persisted for about a year.

Three months before, when she was doing well and had not had hives after cold exposure for about 10 years, she was restung by a wasp, CU recurring at the beginning of November. Exposure to cold

air or washing hands with cold water ($<10^{\circ}\text{C}$) resulted in the rapid induction of urticarial lesions.

After all three wasp stings, she had developed only a moderate local reaction to the sting.

Treatment with ketotifen was begun without significant effect on the symptoms. Physical examination showed no abnormalities. The ice-cube test on the volar forearm was positive. All other laboratory tests performed to rule out an underlying factor, including CBC, ESR, CRP, ANA, rheumatoid factor, infectious mononucleosis spot test, syphilis serology, total complement, cold agglutinin, cryofibrinogen, cryoglobulin, serology for hepatitis A, B, and C, cytomegalovirus, Epstein-Barr virus, and anti- α -1-antitrypsin, were normal or negative. Total IgE was 41.4 UI/ml with specific IgE to wasp venom at 3.07 UI/ml (class 2, CAP System, Pharmacia®, Uppsala, Sweden). The intensity of the CU lesions decreased progressively over 6 months. About a year after the last wasp sting, the patient was totally asymptomatic. At that time, the ice-cube test was performed on the volar forearm with and without injecting 0.1 ml of patient serum from a symptomatic period. Both tests were negative.

The history of this patient suggested a causal relationship between natural wasp sting and the induction of transient CU. In the literature, natural or therapeutic exposure to Hymenoptera venom has only rarely been described as associated with this condition. Houser et al. (2) described a case of CU that worsened after a bee-sting reaction, and Anfosso-Capra et al. (3) described a case of CU appearing during rush desensitization to yellow-jacket venom.

As CU is a relatively rare disorder and is associated with numerous biologic abnormalities or diseases (4), we think that patients with CU should be asked whether they had suffered natural Hymenoptera stings. Our attempt to reproduce CU lesions during remission with the patient's serum dating from a symptomatic period failed, suggesting that no cold-sensitive factor was present in the serum or that it was removed by freezing and/or storage of the serum.

Key words: cold urticaria; wasp venom.

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Delayed hypersensitivity to tetrazepam

TETRAZEPAM ($\text{C}_{16}\text{H}_{17}\text{ClN}_2\text{O}$, mol. wt. 288.78), a benzodiazepine used as a myorelaxant, rarely produces cutaneous side-effects. Neurologic (sedative or paradoxical effects) and digestive disorders are the most frequent side-effects like those classically attributed to the benzodiazepines. There have been a few reports (1-5) of cutaneous reactions to tetrazepam. In these case reports, patch testing with tetrazepam yielded positive results. We report three patients who suffered cutaneous reactions to tetrazepam. We performed oral challenge tests with this drug and other benzodiazepines to study possible cross-reactivity.

■ Patient 1 was a 33-year-old woman who developed a widespread pruritic urticarial eruption and angioedema of the oropharyngeal tract 6 h after ingestion of tetrazepam 50 mg (Myolastan®, Sanofi Winthrop). She needed treatment with systemic corticosteroids and antihistamines.

■ Patient 2 was a 78-year-old woman with arthralgia who presented with a generalized and pruritic micropapular rash on the trunk 8 h after ingestion of one tablet of Myolastan. After withdrawal of tetrazepam and treatment with systemic corticosteroids, the rash disappeared within 8 days.

■ Patient 3 was an 83-year-old man who developed shortness of breath and a pruritic generalized erythematous eruption 6 h after ingestion of one tablet of Myolastan. He was admitted to the emergency room and treated with systemic corticosteroids. These symptoms subsided within 2 days.

■ Skin prick tests (5 mg/ml) and the histamine-release test with tetrazepam were negative in all patients. Patch testing with tetrazepam 1% and 5% petrolatum was positive at 48 and 96 h, respectively. Results in four controls were negative. Oral challenge with tetrazepam was positive in patients 1 and 2 (Table 1). Patient 1 developed a pruritic

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