

Short Communications

Contact urticaria due to aescin

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Key words: contact urticaria; adverse drug reaction; aescin; aescin polysulfate; medicaments. © Munksgaard, 1997.

Case Report

A 51-year-old-man, with a history of contact sensitization to colophony, mercapto mix and thiuram mix, was referred for investigation of an adverse reaction after local application of Feparil®gel (Madaus Cerafarm Laboratories). This medicament had been recommended for treatment of an inflammatory condition of the right knee; he was not on any other medical treatment. Approximately 30 min after applying Feparil®gel to his knee, he developed generalized urticaria and dyspnea. These symptoms wore off 2–3 h after intramuscular chlorpheniramine (5 mg) and hydrocortisone (200 mg). As far as he knew, he had never used Feparil®gel before.

Prick tests with all the constituents of Feparil®gel (kindly supplied by the manufacturer) were performed, comparing reactions to histamine dihydrochloride (10 mg/ml) as positive control and saline solution as negative control. As shown in Table 1, only aescin and aescin polysulfate were positive. 5 healthy and 5 atopic volunteers were used as a control group and tested following the same procedure, but none of them showed positive reactions. Prick tests with common aeroallergens in our area (pollens, mites, molds, and danders) and specific IgE to the same allergens (CAP System, Pharmacia) were negative.

Table 1. Prick tests with the constituents of Feparil®-gel, and positive (histamine dihydrochloride) and negative (saline solution) controls; wheals were measured as transverse diameters (mm)

Substance	Dilution	Wheal
lavender essence	as is	–
carbopol	as is	–
triethanolamine	as is	–
Estol	as is	–
Tween-80	as is	–
neroli essence	as is	–
diethylamine salicylate	as is	–
aescin polysulfate	1/1000	10×10
aescin	1/1000	–
	1/100	–
	1/10	10×8
Controls		
histamine dihydrochloride	10 mg/ml	9×7
saline solution	as is	–

Discussion

Contact urticaria from aescin must be rare: to our knowledge, it has not previously been reported. Aescin is chemically included among the saponins, and is isolated from the dried seeds mainly of the horse chesnut tree (*Aesculus hippocastanum*) (1). Medicaments containing aescin are used topically, essentially for venous diseases (chronic vein insufficiency) (2).

The fact that our patient reacted on presumably the 1st exposure might suggest a non-immunologic mechanism for the reaction. Nevertheless, negative results elicited in all controls (atopic and non-atopic) dispute this. Prick or intradermal tests are not recommended by some authors in routine screening for contact urticaria, and such tests are considered a research procedure (3). Controls are mandatory for proper interpretation of specificity (3–5), and we selected 10 volunteers as a control group. For confirmation of an immunologic mechanism, however, the presence of specific IgE must be shown (4, 5). Aescin is a glycoside and probably acts as a hapten. Thus, the presence of specific IgE against aescin may be difficult to demonstrate. Furthermore, since aescin (as is) elicited a positive reaction at a higher dilution (1/1000) than aescin polysulfate (1/10), chemical struture might cause changes in aescin allergenicity. All these data lead us to diagnose this case as contact urticaria from aescin, due to an uncertain (although probably immunologic) mechanism.

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Delayed hypersensitivity to silicon causing gingival hyperplasia

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Key words: gingival hyperplasia; allergic contact gingivitis; silicon; dental patients; curing cement; porcelain/metal crowns; methyldibromoglutaronitrile. © Munksgaard, 1997.

Case Report

A 35-year-old woman was evaluated in 1996 for red swollen gums overlying teeth #8 and #9. This condition had been present for 11 years. At the onset in 1985, she was assessed by her dentist and found to have crowns on the teeth adjacent to the hyperplastic areas. The crowns consisted of porcelain fused to metal. After various unsuccessful medical therapies, she was referred to a periodontist in 1987, who performed a surgical procedure to correct what was thought to be a "biological width deformity" caused by the previous crowns. Although her symptoms improved with the placement of temporary crowns, they gradually recurred when the permanent crowns were replaced. These replacement crowns were similar to her first, i.e., porcelain fused to metal. She denied the use of phenytoin, carbamazepine, cyclosporin or calcium channel blockers, all of which can cause gingival hyperplasia (1). She was a computer operator at a retail clothing store and had no significant chemical exposure in the workplace or at home. There was no history of metal intolerance. She had a history of an allergic reaction to penicillin.

She was patch tested to the NACDG standard series and to the University of Kansas Medical Center's dental and supplemental allergens. These chemicals included all of the known allergenic ingredients listed on the MSDS for the dental materials that had been used. Included among these was silicon tetrachloride 2% pet. (pH 5.1). In a prior report (2), this ingredient had been used to screen for silicon allergy. In our experience of testing over 100 patients with this allergen, no prior allergic reaction and only a very few minor irritant reactions had been seen.

At 3, but not 2, days after application of the patches, the patient had a ++ reaction to silicon tetrachloride and a + reaction to methyldibromoglutaronitrile. There was no evidence of an allergic reaction to any of the other chemicals tested. After detailed review of her MSDS, it was noted that the curing cement used in her porcelain crowns contained silicon dioxide. In contrast, the patient had no evident exposure to methyldibromoglutaronitrile. Methyldibromoglutaronitrile/phenoxyethanol is a preservative used in cosmetics and did not appear to be relevant to her gingival hyperplasia.

Discussion

Although the literature contains many references to phenytoin, cyclosporin and/or calcium channel blockers and gingival hyperplasia, silicon-induced gingival hyperplasia has not been reported. In 1 study, patch testing with silicon tetrachloride 2% aq. in 30 patients with contact stomatitis yielded no allergic reactions, although 10 patients developed irritant reactions most likely related to the low pH (0.5) of the solution (2). In a single case report of an allergic reaction to a silicon-based dental material, it was the catalyst that was the offending allergen (3). Nonetheless, silicon is known to induce granulomatous reactions (4). Furthermore, allergic reactions to silicon, although not documented by patch testing, have been blamed for the rejection of ventriculoperitoneal shunts in 3 patients (5). Thus, although silicon is a rare cause of delayed-type hypersensitivity, such reactions seemingly occur.

While it is probable that the dental exposure to silicon was responsible for the patient's gingival hyperplasia, to confirm the allergy would require removing the putative allergen and replacing the crowns with materials free of silicon dioxide. However, silicon may be difficult to avoid in the dental crown materials presently available. The patient was provided with detailed results of her patch testing and was referred to her periodontist for further care. Due to financial matters, she has not yet had the crown replaced and the gingival hyperplasia persists.

References

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