

IgE-mediated allergy to henna

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• NOWADAYS there is a rather vigorous back-to-nature trend in Western countries. Consequently, henna as a natural hair dye has become increasingly popular. This shift away from chemical dyes is enforced by the relatively high risk of type IV sensitization to chemical dyes, in both hairdressers and their clients. Henna is derived from the shrub *Lawsonia inermis*, which is native to north Africa and the Middle East.

A 22-year-old female hairdresser had worked for 5 years without health problems. However, after moving to another hair-

Occupational allergy in a hairdresser.

dressing salon, she developed progressively the following symptoms at work: sneezing, conjunctivitis, running nose, dry cough, and severe dyspnea. This last symptom compelled her to take a long sick leave. In the last salon she had to work intensively with henna as a natural hair dye for the first time in her career. She herself thought the henna to be the cause of her complaints. Her only previous atopic complaint had been hay fever; she had no skin problems. Moreover, in her work, she never had skin symptoms such as contact urticaria or contact dermatitis.

Finally, her health complaints, notably the severe dyspnea, caused by this specific sensitization to henna, compelled her to work at another hairdressing salon where henna was not used. After this change of job, she remained free of symptoms.

Commercial henna powder from the salon was used to prepare an aqueous suspension (10% w/v). The skin prick test (SPT) with this extract was highly positive (3+), but

negative in six healthy controls. SPT with routine inhalant allergens including birch, grass, and mugwort pollen gave a positive reaction only for grass pollen (2+). The assay of IgE was 7.4 kU/l for grass pollen, and total IgE was 317 kU/l.

With the same batch of commercial henna powder, an allergosorbent for RAST testing was prepared by coupling 3 mg of extracted henna protein to 100 mg Sepharose-4B. Apart from the patient's serum, RAST testing was performed with some control sera positive for birch pollen, grass pollen, or vegetable IgE-cross-reactive components (such as carbohydrate cross-reactive determinants [CCD] and profilin). Both the CCD-positive and the profilin-positive control showed some IgE binding to our immobilized henna extract (2.8 kU/l), but the patient's serum gave a markedly higher result, namely, 9.9 kU/l, indicating that this serum contains henna-specific IgE. Negative controls showed no binding.

We also performed RAST-inhibition experiments: grass-pollen extract could not inhibit the henna RAST for our patient's serum, indicating that the observed positive henna RAST did not result from grass-pollen/henna cross-reactivity.

Immunoblotting experiments using the same henna extract did not lead to convincing blotting patterns; after a very long radiographic film exposure, some IgE binding to a 25-kDa henna component could be detected.

Patch testing according to ICDRG guidelines with henna was negative, excluding type IV allergy to henna.

Up to now, at least six patients have been reported to have an immediate-type hypersensitivity to henna, all due to occupational exposure to henna (1–5). Our paper, however, reports for the first time that the hypersensitivity is due to induction of specific IgE antibodies to henna. Sensitization to henna has become a severe occupational hazard for hairdressers. Up to

now, the prevalence appears to have been rather low, but underestimation due to the relative unfamiliarity with this allergy cannot be excluded. The main event of sensitization is inhalation of henna powder dispersed in the air. Measures to prevent this airborne sensitization have to be developed in close cooperation with the hairdressers themselves and the manufacturers of henna. A simple approach might be to provide the henna as a ready-to-use paste instead of a powder.

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