Clinical Communications

Occupational IgE-mediated respiratory allergy to shikakai powder

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Clinical Implications

This report highlights the respiratory sensitizing potential of shikakai powder used to prepare organic hair products and provides evidence supporting an IgE-mediated mechanism underlying the development of rhinitis and asthma.

The development of occupational asthma and rhinitis in hairdressers has been related predominantly to low—molecular weight chemicals including persulfate salts used as hair bleaches[1] and oxidative hair dyes containing para-amino compounds.[2,3] However, there have been anecdotal reports of occupational respiratory allergy caused by plant-derived hair products, including hair dyes based on henna (*Lawsonia inermis*)[4] and indigo (*Indigofera tinctoria*)[5] and hair conditioners containing hydrolyzed wheat protein.[6]

Shikakai (*Acacia concinna*, botanical family of Fabaceae) is traditionally used in India as a hair conditioner and gentle hair shampoo because of its content of vegetable saponins. The pods of shikakai are dried and finely ground into a powder that is mixed with water to produce eco-friendly hair cleansers and conditioners. Shikakai powder is exported from India to North America and Europe and is easily available for the preparation of hair care products, but the number of hairdressers exposed to this plant is currently unknown.

Here, we report clinical and immunological findings in a hairdresser who developed asthma and rhinitis caused by exposure to shikakai powder used to prepare plant-based hair products.

A 47-year-old woman had been working as a hairdresser for 26 years without symptoms when she started to use exclusively vegetable ingredients to produce organic hair products. About 15 months later, she developed rhinoconjunctivitis symptoms and chest tightness when handling plant powders. She noticed that the symptoms occurred electively when she used shikakai-containing powders as hair dyes and conditioners. The patient was evaluated 6 months after the onset of work-related symptoms at a time she was treated only with an oral H1-antihistamine on demand. She had smoked one pack of cigarettes per day for 30 years, but had stopped smoking 4 years before the onset of symptoms. She denied any history of prior allergic disease and did not report skin symptoms, but the skin rarely came in direct contact with the plant powders.

Skin prick test gave negative results with a battery of common inhalant allergens but elicited a positive response to shikakai (10 × 5-mm) powder diluted 1/100 weight/volume (w/v) in phosphate-buffered saline and stirred for 4 hours at room temperature. Skin prick test was negative with extracts of other plants used at work, including *L inermis* (red henna) and *Cassia obovata* (yellow henna).

The patient gave informed consent to perform a specific inhalation challenge (SIC) with the suspected plant powder according to international recommendations.[7] Baseline spirometry showed an FEV_1 of 2.15 L (83% predicted) and an FEV_1/FVC ratio of 0.78. As a control challenge, the patient was exposed to lactose powder for 30 minutes without eliciting significant changes in FEV_1 over the following 6 hours (Figure 1). At the end of this control day, the concentration of histamine causing a 20% decline in FEV_1 measured using the tidal breath method was 1.73 mg/mL, reflecting moderate nonspecific bronchial hyperresponsiveness. Analysis of a sputum sample induced by inhaling increasing concentrations (3%, 4%, and 5%) of nebulized hypertonic saline revealed a 2.5% eosinophil count. On the next day, tipping shikakai powder diluted 1/100 w/v in lactose powder for a cumulative duration of 90 seconds elicited marked symptoms of rhinoconjunctivitis and a fall in FEV_1 of 24% from baseline value with progressive recovery. A delayed asthmatic reaction with a maximal fall in FEV_1 of 16% from the pre-SIC value was recorded 6 hours after the end of exposure. The patient was then administered inhaled salbutamol. A sputum sample collected at that time revealed a marked increase in eosinophil count to 39% compared with the control day assessment. Twenty-four hours after the end of the challenge exposure, the concentration of histamine causing a 20% decline in FEV_1 was decreased to 0.36 mg/mL and the sputum eosinophil count was still increased at 48%. The concentration of FeNO was increased to 125 ppb compared with 47 ppb at the pre-exposure assessment.

We investigated the presence of specific IgE (sIgE) binding against an aqueous extract of shikakai and other plants used at work *in vitro*.[8] A detailed description of immunological studies can be obtained from the authors on request. A high level of sIgE against shikakai (195 kUA/L) was detected using the ImmunoCAP method (ThermoFisher, Uppsala, Sweden). A multiplex immunoassay did not detect sIgE against a comprehensive panel of allergen components and extracts of inhalant and food allergens (Allergy Xplorer [ALEX[2]], MacroArray Diagnostics, Vienna, Austria). IgE immunoblot testing with the patient's serum to shikakai extract (protein content, 0.2 mg/mL) revealed IgE binding to a 22.2-kDa protein band (Figure 2), but mass spectrometry failed to identify homology of the IgE-binding shikakai protein with known plant allergens.

To the best of our knowledge, this is the first report of sIgE-mediated occupational airway allergy to shikakai used to prepare organic hair products. The causal relationship was ascertained through an SIC and immunological studies. The asthmatic reaction induced by challenge exposure to shikakai powder was associated with a significant increase in the level of FeNO concentration and marked eosinophilic airway response supporting an immunologically mediated T_H2 mechanism.[9] ImmunoCap testing provided evidence of IgE-

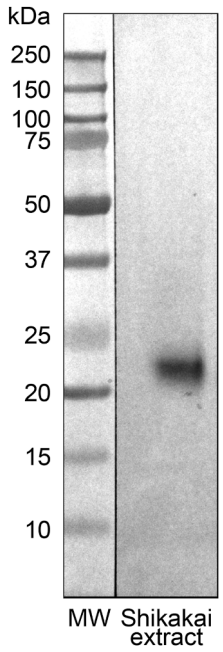

FIGURE 1. Results of specific inhalation challenges illustrating changes in FEV_1 after a control exposure to lactose powder for 30 minutes (*open circles*) and challenge exposure for 90 seconds to shikakai powder (*Acacia concinna*) diluted 1/100 in lactose powder (*closed squares*). *Pre,* pre-exposure; *BD*, inhaled bronchodilator.

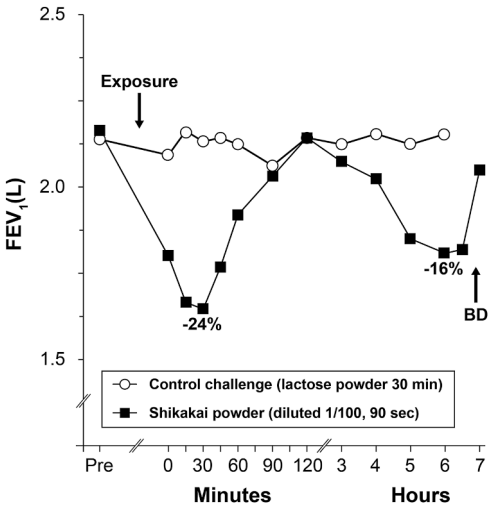

FIGURE 2. Immunoblot analysis of binding of IgE antibodies from the patient's serum to the shikakai extract. Molecular weights of marker proteins are indicated on the *left*. The patient demonstrated strong IgE binding to a protein with a molecular weight (MW) of 22 kDa.

mediated sensitization to shikakai. In addition, immunoblotting assay of a shikakai extract using the patient's serum identified an IgE-binding protein with a molecular mass of 22 kDa. However, mass spectrometric identification of this IgE-binding shikakai protein failed to reveal homology to known plant allergens.

This report further highlights the potential for inducing IgE-mediated respiratory allergy resulting from inhalation exposure to plant-derived products used in powder form. Awareness of this possibility may be clinically relevant owing to the growing demand for natural, chemical-free hair care products.

Acknowledgments

All authors were involved in the conception of the study and contributed to the collection and interpretation of the data. They provided input into the drafting of the manuscript, critical feedback, and final approval of the manuscript. V. Doyen is the guarantor of the final content of the manuscript.

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This work was supported in part by a grant from the Fondation Mont-Godinne and by the DGUV (German Social Accident Insurance) as part of Institute of the Ruhr University Bochum (IPA) Project IPA-014.

Conflicts of interest: I. Mittermann and M. Aumayr are employees of MacroArray Diagnostics GmbH. The rest of the authors declare that they have no relevant conflicts of interest.

Received for publication November 12, 2025; revised December 2, 2025; accepted for publication December 4, 2025.

Available online ■■

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2213-2198

https://doi.org/10.1016/j.jaip.2025.12.005

REFERENCES

1. Doyen V, Thirionet R, Migueres N, Vandenplas O, Sastre J, Valverde-Monge M, et al. Phenotypic characteristics of occupational asthma caused by persulfate salts in hairdressers: a multicenter cohort study. J Allergy Clin Immunol Pract 2025;13:1397-404.e5.
2. Helaskoski E, Suojalehto H, Virtanen H, Airaksinen L, Kuuliala O, Aalto-Korte K, et al. Occupational asthma, rhinitis, and contact urticaria caused by oxidative hair dyes in hairdressers. Ann Allergy Asthma Immunol 2014;112:46-52.
3. Doyen V, Walusiak-Skorupa J, Wiszniewska M, Vandenplas O, Suuronen K, Sastre J, et al. Occupational asthma due to hair dyes containing para-amino compounds. Am J Ind Med 2026;69:42-8.
4. Starr JC, Yunginger J, Brahser GW. Immediate type I asthmatic response to henna following occupational exposure in hairdressers. Ann Allergy 1982;48:98-9.
5. Haltia T, Jungewelter S, Airaksinen L, Suomela S, Lindstrom I, Suojalehto H. Occupational asthma, rhinitis, and contact urticaria from indigo (Indigofera tinctoria) hair dye. J Allergy Clin Immunol Pract 2021;9:3500-2.
6. Airaksinen L, Pallasaho P, Voutilainen R, Pesonen M. Occupational rhinitis, asthma, and contact urticaria caused by hydrolyzed wheat protein in hairdressers. Ann Allergy Asthma Immunol 2013;111:577-9.
7. Vandenplas O, Suojalehto H, Aasen TB, Baur X, Burge PS, de Blay F, et al. Specific inhalation challenge in the diagnosis of occupational asthma: consensus statement. Eur Respir J 2014;43:1573-87.
8. Sander I, Kespohl S, Merget R, Goldscheid N, Degens PO, Bruning T, et al. A new method to bind allergens for the measurement of specific IgE antibodies. Int Arch Allergy Immunol 2005;136:39-44.
9. Doyen V, Migueres N, van Kampen V, Suojalehto H, Mason P, Munoz X, et al. Exhaled nitric oxide and sputum eosinophils are complementary tools for diagnosing occupational asthma. Allergy 2025;80:1015-24.