Occupational Asthma and Rhinitis due to Yellow and Red Henna in a Hairdresser

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Hairdressing is a profession that often requires large amounts of chemicals to be handled under conditions of humidity and temperature that favor reactivity of these substances and cause them to penetrate the body. The most common occupational disease affecting hairdressers is allergic and irritant contact dermatitis. *p*-Phenylenediamine is the main causative agent. In the case of occupational asthma and rhinitis, the most frequent causative agent is persulfates [1].

Henna is a vegetable dye that is used as a natural alternative to chemical dyes in allergic patients, although it is also a frequent cause of allergic contact dermatitis. Two cases of asthma and 1 report of occupational rhinoconjunctivitis due to red henna (*Lawsonia inermis*) have been published [2,3]. However, the presence of allergy to red henna or to 2 different types of henna has never been reported in the literature.

We present the case of a 30-year-old female hairdresser who developed rhinitis and occupational asthma despite no prior relevant medical history. One year ago, new botanical hair dyes containing *Cassia obovata* (yellow henna) and *L inermis* (red henna) were introduced in her workplace. When handling the products, the patient complained of hives in the areas of exposure, as well as rhinorrhea, pruritus (eyes and nose), and dyspnea. On one occasion, she required emergency care due to bronchospasm. Her symptoms improved significantly outside the workplace, although she never used respiratory protection devices. Her physician prescribed inhaled beclomethasone/formoterol every 12 hours, and her symptoms improved.

The patient was referred to our clinic, where we performed skin prick tests with the commercial product used in her workplace (NeoBarros) and pure yellow and red henna. The results were positive for all 3 substances (wheal diameter >3 mm) and negative for 3 healthy patients (nonasthmatic, nonatopic). We also performed skin prick tests with the aeroallergens present in our environment including mites, grass pollen, fungi, dog and cat dander, and tree pollen, although the results were negative. We carried out patch tests with the standard allergens recommended by the Spanish Research Group on Contact Dermatitis and Skin Allergy (GEIDAC) and with substances known to be allergens in hairdressing (hydroxyethyl methacrylate, methyl methacrylate, ammonium persulfate, vitalizing cream, dye, and dye mud). The results were negative.

Seven months after the last exposure, the patient underwent a specific inhalation challenge (SIC) with red and yellow henna and a nonspecific bronchial challenge test with methacholine. The methacholine test was negative at baseline (PC_{20} >16 mg/mL). A SIC was performed over 2 consecutive days (cumulative time, 5 hours) in a challenge chamber under dust concentration monitoring (DustTrak, TSI) with NeoBarros (Secretos del Agua, Spain), which contains *C obovata* (yellow henna), *L inermis* (red henna), *Urtica dioica, Betula pendula, Thymus vulgaris*, kolin extract, *Cinchona succirubra, Camellia inensis, Linum usitatissimum*, montmorillonite, *Pimpinella anisum, Syzygium aromaticum, Coffea*, and xantham gum. The results were negative. No significant changes in FEV_1 were observed over the following 24 hours. FeNO levels remained unchanged after the SIC (<25 ppb). A nonspecific bronchial challenge with methacholine performed 24 hours after the SIC was positive (PC_{20}, 3.5 mg/mL).

Acoustic rhinometry was also performed to assess rhinitis before specific provocation and 30 minutes after. The results of this test were positive, revealing a 26% and 21% fall in minimum transverse area in the right and left nostrils, respectively, together with sneezing and rhinorrhea. The results of the SIC and acoustic rhinometry confirmed the diagnosis of rhinitis and oral allergy due to commercial henna extract.

The henna extract with NeoBarros was analyzed using SDS-PAGE immunoblotting (Figure) under reducing conditions (with 2-mercaptoethanol), as previously described [4]. The assay revealed IgE-binding proteins of approximately 40, 60, 75, and 80 kDa in the extract. These results further confirmed the initial diagnosis of asthma and occupational rhinitis due to a high-molecular-weight agent.

The most frequent cause of sensitization to henna is through the skin and not by inhalation. This route is commonly seen in hairdressers who have tattoos [5]. The mean frequency of skin allergy to paraphenylenediamine caused by black

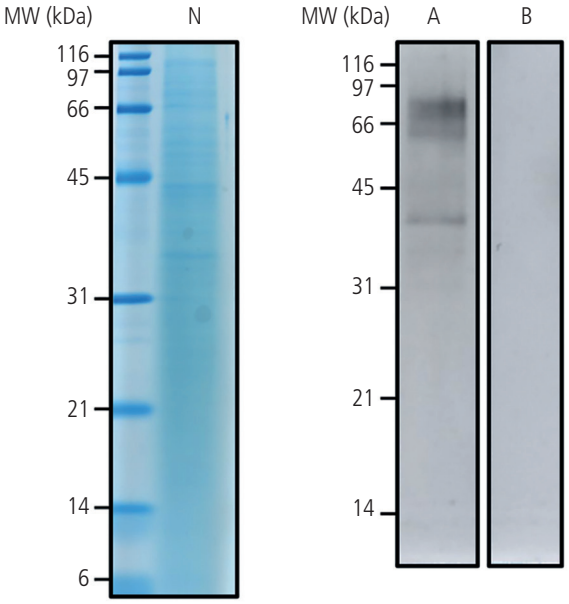

Figure. SDS-PAGE IgE immunoblotting of the henna extract. Lane A, patient's serum; Lane B, control serum (nonatopic individual); Lane N, molecular mass standards.

henna tattooing is estimated at 2.5% per application [6]. In the case presented here, the patient appeared to be sensitized via inhalation, given the presence of respiratory symptoms such as rhinitis and bronchial hyperreactivity when she handled the product. Most low-molecular-weight substances that cause oral allergy act through a mechanism that, although likely to be immunologic, does not involve IgE [7]. The positive skin prick test results in this patient indicate that the underlying mechanism is IgE-mediated. IgE is frequently involved in high-molecular-weight substances such as henna. The present case has characteristics that are typical of the high-molecular-weight–oral allergy phenotype, namely, positive skin prick test results and the presence of rhinitis, conjunctivitis, and urticaria. SIC did not significantly increase the FeNO level, a finding that is also described more frequently in low-molecular-weight phenotypes [8]. This may be attributable to the long-period between the last occupational exposure and the SIC (7 months). The clinical and immunological characteristics that make this case different from others described elsewhere [2,5,6] may be due to the absence of paraphenylenediamine in the dye used by our patient, which contained a mixture of *C obovata* and L *inermis* as its main components.

Diagnosis of oral allergy remains challenging for clinicians and requires a rigorous, protocol-based approach, as well as better standardization and generalization of diagnostic tests. In the case described here, SIC and immunoblotting proved helpful in reaching an accurate diagnosis. In immunoblotting, the bands may correspond to products other than the hennas. The diagnosis of allergy to the 2 hennas was based mainly on the positive skin test results for both (the challenges and the immunoblot were performed with the commercial product, which contained other components).

Henna products with *L inermis* and *C obovata* should be considered potential allergens in hairdressers due to their ability to cause rhinitis and occupational asthma, even in the absence of paraphenylenediamine.

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Conflicts of Interest

JS reports having served as a consultant to Thermo Fisher, Novartis, Sanofi, Leti, FAES FARMA, Mundipharma, and GSK. He has also received lecture fees from Novartis, GSK, Stallergenes, LETI, and FAES FARMA and grant support for research from Thermo Fisher and ALK.

VV, MJR, IE, CPV, and JC declare that they have no conflicts of interest.

References

1. Quirce S, Fernández-Nieto M, del Pozo V, Sastre B, Sastre J. Occupational asthma and rhinitis caused by eugenol in a hairdresser. Allergy. 2008;63(1):137-8
2. Pepys J, Hutchcroft BJ, Breslin AB. Asthma due to inhaled chemical agents-persulphate salts and henna in hairdressers. Clin Allergy. 1976;6(4):399-04
3. Starr JC, Yunginger J, Brahser GW. Immediate type I asthmatic response to henna following occupational exposure in hairdressers. Ann Allergy. 1982;48(2):98-9
4. Pastor C, Cuesta-Herranz J, Cases B, Pérez-Gordo M, Figueredo E, de Las Heras M, et al. Identification of Major Allergens in Watermelon. Int Arch Allergy Immunol. 2009;149(4):291-8
5. Bregnhøj A, Søsted H, Menné T, Johansen JD. Exposures and reactions to allergens among hairdressing apprentices and matched controls. Contact Dermatitis. 2011;64(2):85-9.
6. Mukkanna KS, Stone NM, Ingram JR. Para-phenylenediamine allergy: current perspectives on diagnosis and management. J Asthma Allergy. 2017;10:9-15
7. Sastre J, Vandesplas O, Park H-S. Pathogenesis of occupational asthma. Eur Respir J. 2003;22:364-7.
8. Sastre J, Costa C, del Garcia Potro M, Aguado E, Mahillo I, Fernández-Nieto M. Changes in exhaled nitric oxide after inhalation challenge with occupational agents. J Investig Allergol Clin Immunol. 2013;23(6):421-7.

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