

the etiology of the patient's dermatitis was contemplated.¹ Expanding on this, our recommendation to clinicians is "not to neglect the parents."

References

1. Wilkinson DS. Connubial photodermatitis. *Contact Dermatitis* 1975; 1:58.
2. Pratt MD, Belsito DV, DeLeo VA, et al. North American Contact Dermatitis Group patch-test results, 2001-2002 study period. *Dermatitis* 2004;15:176-83.
3. Hogeling M, Pratt M. Allergic contact dermatitis in children: the Ottawa hospital patch-testing clinic experience, 1996 to 2006. *Dermatitis* 2008;19:86-9.
4. Bruckner AL, Weston WL, Morelli JG. Does sensitization to contact allergens begin in infancy? *Pediatrics* 2000;105: e3.

Contact Leukoderma from para-Phenylenediamine

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Précis Contact leukoderma is a common reaction to monobenzyl ether of hydroquinone, alkyl phenols, hydroquinone, and several para-substituted phenols, but it is only rarely due to para-phenylenediamine (PPD).

Discussion A 77-year-old African American man had a 5-month history of depigmentation of the scalp. He regularly colored his hair without problems; 6 months earlier, however, he had experienced a pruritic dermatitis 2 days after applying a hair dye. The dermatitis spread to his trunk and extremities and required topical and systemic corticosteroids for resolution. Normal skin coloration and appearance followed the resolution of the generalized dermatitis. However, 3 weeks after the dermatitis cleared, the patient developed depigmentation of the scalp. Physical examination revealed a complete loss of pigment on 30% of the scalp (Fig 1) and no involvement on the rest of the body.



Figure 1. Scalp depigmentation seen on physical examination.

Patch testing with the North American Contact Dermatitis Group standard series revealed a +++ reaction to PPD at 48 and 96 hours but did not induce depigmentation. The remainder of the patch-test results were negative, with the exception of a +++ reaction to nickel, which was not considered relevant.

Contact leukoderma from PPD in hair dye is uncommon. The first report was presented by Taylor and colleagues in 1993¹; few cases have been reported since then.²⁻⁴ The individuals afflicted by PPD-induced contact leukoderma from hair dye in those cases had relatively dark skin: five were African American, one was a black Yemenite, one was Hispanic, and one was Indian.¹⁻⁴ Seven of these patients were male.

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DOI 10.2310/6620.2008.08038

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Vitiligo, Koebner-induced vitiligo, postinflammatory hypopigmentation, and cutaneous lupus erythematosus are possible explanations for the scalp depigmentation in the subject in this case. However, the absence of vitiligo on other skin locations, the temporal relationship to the hair dye application, and the positive contact allergy to PPD support a diagnosis of contact leukoderma. Furthermore, the depigmentation was confined to the sites of hair dye application. Some cases of contact leukoderma from PPD in hair dye have been preceded by inflammation including pruritus,^{3,4} edema, and exudation.³

Although the pathomechanism of contact leukoderma is unknown, new evidence implicates the destruction of melanocytic deoxyribonucleic acid (DNA). Bajaj and colleagues noted that calf thymus DNA was altered significantly when infused with PPD.⁵

Contact leukoderma can resolve spontaneously with time.² One case of contact leukoderma was successfully treated with narrowband ultraviolet B therapy administered three times weekly for 1 year.⁴

Zebra

PPD was chosen as Contact Allergen of the Year by *Dermatitis* in 2006.⁶ We highlight a rare case of contact leukoderma, which makes this case report a “zebra.”

References

1. Taylor J, Maibach H, Fisher A, et al. Contact leukoderma associated with the use of hair colours. *Cutis* 1993;52:273–80.
2. Brancaccio R, Cohen D. Contact leukoderma secondary to para-phenylenediamine. *Contact Dermatitis* 1995;32:313.
3. Bajaj A, Gupta S, Chatterji A, et al. Hair dye depigmentation. *Contact Dermatitis* 1996;35:56–7.
4. Trattner A, David M. Hair-dye-induced contact vitiligo treated by phototherapy. *Contact Dermatitis* 2007;56:115–6.
5. Bajaj A, Dubey S, Misra K. A simple spectroscopic technique proposed for scanning the depigmenting potential of azo dyes. *Contact Dermatitis* 2004;51:97–8.
6. DeLeo V. p-Phenylenediamine. *Dermatitis* 2006;2:53–5.