Neutrophilic and Eosinophilic Dermatitis Caused by Contact Allergic Reaction to Paraphenylenediamine in Hair Dye

Vincent Lönngren, MScMed; Ewa Young, MD; Mecius Simanaitis, MD, PhD; Cecilia Svedman, MD, PhD

Background: Paraphenylenediamine (PPD) in hair dyes can cause systemic as well as cutaneous allergic reactions such as neutrophilic and eosinophilic dermatitis. The symptoms are often severe. The acute lesion is normally histologically indistinguishable from any eczematous reaction with marked spongiosis.

Observations: We report a case of allergic contact dermatitis caused by the use of hair dye containing PPD that developed in a patient who had been using the same hair dye for many years. Her symptoms included scalp dermatitis and widespread skin lesions as well as lymphadenopathy and quite possibly dyspnea resembling

asthma. What is most remarkable about this case is the histopathologic finding of neutrophilic cellulitis and a marked neutrophilic infiltrate with variable spongiosis. This unique finding was confirmed by histologic analysis of a patch test lesion specimen.

Conclusion: It is always important to consider contact allergic dermatitis as a cause of dermatitis because of the variable presentation of the disease, including unique histologic findings that do not fit the conventional picture, as in the present case.

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HE POTENT SENSITIZER PARAphenylenediamine (PPD) is a substance that is often found in permanent hair dyes.1 Contact allergy to hair dyes is common among hairdressers and consumers.^{2,3} As in any contact allergic dermatitis, the acute-stage lesions histologically resemble any eczematous reaction with marked spongiosis. The use of PPD was banned in Sweden until 1992, but the ban was lifted owing to legislative measures within the European Union. Paraphenylenediamine is well known to contribute to darker hair colors in permanent hair dyes but can also be found in lighter shades. When PPD is used in hair dyes, it is mixed with oxidizing agents, such as hydrogen peroxide, and new, partly unknown substances are formed. Contact allergic reactions to these derivatives and oxidation products of PPD are thought to play a role in PPD-related contact allergy.^{4,5} Another risk of developing lifelong allergy to PPD, for children as well as adults, is through temporary tattoos: "black henna tattoos." For such tattoos, henna is mixed with PPD. Serious adverse reactions to PPD, including severe facial and scalp dermatitis and asthma,

have been reported as early as 1924.⁶ We report a case that illustrates some of the severe reactions that may arise from exposure to PPD in hair dyes and discuss some of its unusual histologic and clinical characteristics.

REPORT OF A CASE

A 58-year-old woman presented with a 2-week history of painful and itchy lesions on her chest, back, and neck (**Figure 1** and **Figure 2**). Her son recently had a case of impetigo, and her family physician had suspected that she had the same condition. A course of isoxazolyl penicillin had been completed, without any effect on the lesions. The patient was otherwise healthy except for chronic obstructive pulmonary disease and had no remarkable immunologic or dermatologic history. She was afebrile, with only mild leukocytosis.

The lesions, which were nummular erythematous plaques with excoriations and crusts, were located on the neck, the front and back of the upper chest area, and the scalp. A few lesions were found on the arms, and 1 lesion was found on the right ankle. A punch biopsy was performed, and prednisolone therapy (40 mg/d) was ini-

Author Affiliations:
Department of Dermatology,
Skåne University Hospital,
Malmö (Mr Lönngren and
Dr Simanaitis), and Department
of Occupational and
Environmental Dermatology,
Lund University, Lund
(Drs Young and Svedman),
Sweden.



Figure 1. Back of the upper chest area and neck covered by nummular erythematous plaques with excoriations and crusts.



Figure 2. The patient's neck and ear covered with lesions.

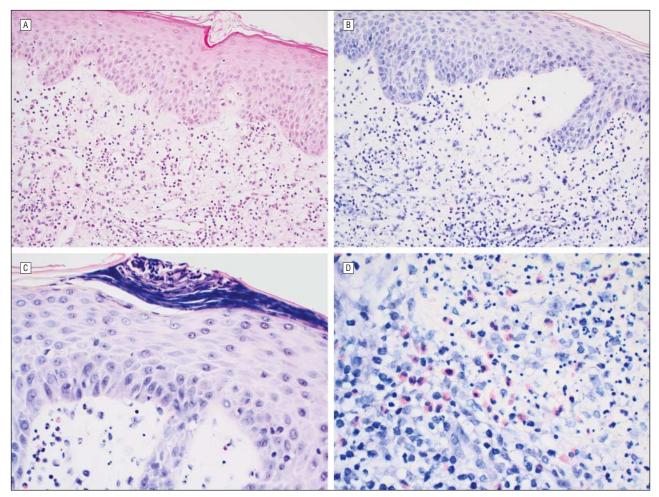


Figure 3. Four photomicrographs of skin from the unusual lesions. A, Diffuse dermal infiltrate of neutrophils with variable epidermal spongiosis (hematoxylin-eosin, original magnification ×200). B, Pronounced edema of the papillary dermis (Giemsa, original magnification ×200). C, Focal parakeratosis (Giemsa, original magnification ×400). D, Admixture of eosinophils (Giemsa, original magnification ×400).

tiated. Histologic analysis revealed the presence of neutrophilic cellulitis and eosinophilic granulocytes. There were remarkable dermal edema, variable spongiosis, and only a few lymphocytes. A diagnosis of Sweet syndrome was suggested, although the high frequency of eosinophilic granulocytes was difficult to explain (Figure 3).

The results of direct immunofluorescence microscopy were negative.

Within a few days, the lesions had spread further, involving the perianal and genital regions. They had become increasingly wet and weeping, and topical treatment with betamethasone-clioquinol and potassium permanganate was initiated. Bacterial culture revealed only moderate growth of *Staphylococcus aureus*. Ten days after the first visit, there was no significant improvement. Neutrophilic cutaneous lupus erythematosus was suspected because of the neutrophilic dermatitis in the upper part of the dermis, although there was no interface dermatitis. Hydroxychloroquine sulfate therapy (400 mg/d) was initiated.

Incidentally, the patient developed an episode of dyspnea, which was treated at the emergency department. The cause of the dyspnea was not entirely clear, but it did not display the characteristics of a typical exacerbation of chronic obstructive pulmonary disease and lasted only a few hours. Two weeks after the first visit, the lesions had finally started to fade, and a few days later, only postinflammatory hyperpigmentation and some desquamation remained. The results of all immunoserologic tests were negative, as were repeated attempts at direct immunofluorescence microscopy. All treatment except topical betamethasone was discontinued.

Surprisingly, a few days later, erythematous nummular lesions reappeared on the patient's neck and forearms. It was noted that she had dyed her hair. She had been using the same hair dye for many years. The distribution of the lesions raised the suspicion of allergic contact dermatitis due to the use of hair colorants despite the atypical histologic findings. Epicutaneous patch testing with the base line series and textile series showed a contact allergic reaction to several allergens, most importantly a 3+ reaction to PPD and a positive reaction to disperse orange 3. The patient was urged to discontinue the use of hair dye. A few months later, the lesions returned in full force after she colored her hair again with the same hair dye. This time, her occipital and cervical lymph nodes were swollen and remained so for at least 2 months. Later, additional epicutaneous allergen testing revealed a reaction to a low concentration of PPD (0.001%), among other substances. Considering the unusual histologic features of the previous specimen, a punch biopsy was performed in the area of the positive reaction to PPD. The sample was found to be histologically identical to the previous ones, quite unlike the conventional histologic appearance of eczema.

COMMENT

It is well known that PPD can cause unusually severe contact allergic reactions in susceptible individuals. In this case, the lesions were not typical of contact allergic dermatitis

or baboon syndrome. The unusual clinical and histologic appearance of the lesions initially misled us away from the diagnosis of contact allergy. There have been some reports of erythema multiforme—like lesions in contact allergic dermatitis to PPD. However, the histologic appearance in our case was not that of erythema multiforme. Instead, it was similar to neutrophilic dermatitis or Sweet syndrome, apart from the pronounced eosinophilia. It did not resemble a conventional eczematous reaction. The diagnosis was finally confirmed by the provocation with PPD as well as by the results of patch testing, including those of histologic examination of the patch test reaction site. It is important to remember that a contact allergic reaction to PPD can present with unusual clinical and histologic features and to ask about hair dye use.

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Correspondence: Vincent Lönngren, MScMed, Department of Dermatology, Skåne University Hospital, Södra Förstadsgatan 101, 205 02 Malmö, Sweden (vincent .lonngren@skane.se).

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