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Erythema Multiforme Following Application of Hair Dye

[Sankha Koley](#), [Jyotirindranath Sarkar](#), [Sanjiv Choudhary](#),¹ [Suparna Dhara](#), and [Manoj Choudhury](#)²

From the Department of Dermatology, Bankura Sammilani Medical College, West Bengal, India

¹Department of Dermatology, J.N.M.C. Sawangi, Wardha, Maharashtra, India

²Department of Pathology, Bankura Sammilani Medical College, West Bengal, India

Address for correspondence: Dr. Sankha Koley, Subhankar Sarani, Bankura – 722 101, West Bengal, India. E-mail: skoley@gmail.com

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Abstract

Erythema multiforme (EM) is an acute mucocutaneous hypersensitivity reaction with varying degrees of blistering and ulceration. Common causes of EM are herpes simplex virus infection, mycoplasma infection, drug hypersensitivity, vaccination and drug–virus interaction. EM induced by contact dermatitis is rare. Paraphenylenediamine, a common ingredient in many hair dyes, is well known to produce allergic contact dermatitis. We report a 35-year-old lady presenting with EM following severe contact dermatitis to hair dye. So far as we know, this is the first report from India describing EM following contact dermatitis.

Keywords: *Contact dermatitis, erythema multiforme, hair dye*

Introduction

Erythema multiforme (EM) is an acute self-limiting mucocutaneous condition of uncertain etiopathogenesis. The most common precipitating factors are herpes simplex virus infection and drugs. Allergic contact dermatitis, a delayed type of hypersensitivity reaction, results from cutaneous contact with a specific allergen to which the patient has developed specific sensitivity. EM induced by contact dermatitis is rare.^[1] We report a case of EM following allergic contact dermatitis to hair dye.

Case Report

A 35-year-old female patient presented with severe erosive dermatitis of head and neck [Figure 1]. Three weeks earlier she had applied a black hair dye. Severe itching, erythema, and edema of the whole head and neck was followed by vesiculation, erosions, and crusting. After 10 days of apparently no treatment, the acute dermatitis subsided partially. But she noted some new lesions on hands and back. She revealed history of application of the same dye for the first time, 2 months earlier with insignificant consequence. The ears and adjoining face were involved severely with relative sparing of the scalp [Figure 2]. There were papules, plaques, and target lesions on hands, back, and thighs [Figure 3]. Two 4-mm punch biopsies were performed from the left flank and the right thigh. Histopathology revealed patchy interface dermatitis with inflammatory cells in subepidermal region along with subepidermal clefts and mild dermal edema [Figure 4]. The clinical and histological features were suggestive of EM. She was not taking any medication and did not receive any vaccination recently. She did not give any prior history of herpes simplex. The only cause of EM was extensive contact dermatitis to a strong allergen. Paraphenylenediamine (PPD), a constituent of the applied dye, was the suspected causative agent. It was confirmed by patch-testing. Because of the severe contact dermatitis experienced by the patient, we diluted the usually recommended test

concentration of 1% PPD in petrolatum. 0.1% PPD in petrolatum was placed on left upper arm and readings were performed 2 and 3 days later. The patient showed (+++)-positive patch-test reaction. More patch tests to determine possible cross-reactivity between PPD, azo dyes, and other para-amino compounds would have been interesting. But the patient and her relatives did not agree as they were very much concerned about the strong test reaction to PPD, which produced temporary hyperpigmentation.

Discussion

EM has been classified as minor, major, Stevens-Johnson syndrome (SJS) and toxic epidermal necrolysis (TEN), where EM minor is the mildest form and TEN is the severest variant.[2] The lesions in EM are typical targets or raised atypical targets, while in the SJS–TEN spectrum the lesions are flat atypical targets or spots. The typical target is a round lesion consisting of three concentric components: a blister or dusky central purpura, a peripheral pale edema, and an erythematous halo. The atypical raised target consists of two rings. Spots are dusky or erythematous macules resembling target lesions. The typical targets and raised atypical targets are considered as two morphologic presentations of the same pathomechanism.[3]

The initial sensitization typically takes 10–14 days from initial exposure to a strong contact allergen. Once an individual is sensitized to a chemical, allergic contact dermatitis may develop within hours to several days of exposure. She did not receive any kind of tattoo before. Our patient was probably sensitized to the hair dye when she applied it for the first time, 2 months back.

The underlying pathogenic mechanism of EM following contact dermatitis has not been established yet. One hypothesis is acute type IV hypersensitivity. However, a study of immunologic parameters of 23 patients with EM pointed toward a type III hypersensitivity reaction.[4] Interestingly, polymorphic light eruption (PLE), thought to be an abnormal immune response to an endogenous cutaneous antigen induced by UV, has also been reported to produce EM. This contradicts the theory that PLE is a type IV hypersensitivity response to a photoinduced antigen within the skin.[5]

In India, black hair dyes are most preferred. They generally come as powder hair dyes, oxidation liquid type, and emulsion type. According to standards regulating bodies such as Bureau of Indian Standards (BIS), the concentration in powder form should not be more than 30% and not less than 3% after dilution. For the oxidation liquid type, the PPD should not be more than 4% in the undiluted state and not less than 1.2% after dilution.[6] The manufacturers are required to give information on the maximum and minimum PPD content.

But the real picture is something else. It was reflected when Consumer Education and Research Society (CERS), Ahmedabad, performed a laboratory test on 14 permanent hair dyes.[6] Three of them did not conform to the parameters of total active matter (dye content). Most (12 out of 14) brands failed to conform to the packing requirements. Most of them failed to disclose information on maximum PPD content. Except two companies, all the other brands of powdered dyes failed to reveal information on minimum PPD content. Another critical information required is the “Warning: Shall not be used to dye eyebrows and eyelashes.” One brand even failed to give this information in its cartons.

In India, most people at home or in salons do not perform the required patch test before dying their hair every time. They believe that no complication during last application means that they will be fine this time also. Nothing can be further from the truth. Our patient had also used the same dye earlier without any side effect. Doing the patch test prior to dyeing the hair could give an advanced warning of an acute reaction.

Recently, numerous case reports of suicidal poisoning after hair dye ingestion have been published from India, many of which are from Andhra Pradesh. Super-Vasmol, a cheap, freely-available hair dye is emerging as a major cause of suicidal agent in India. It contains potential toxins such as paraphenylenediamine, resorcinol, sodium thylenediaminetetraacetic acid and propylene glycol.[7]

PPD is a frequent component of permanent hair dye products and temporary henna tattoos.[8]

Exposure to it in hair dye products may cause severe facial edema and acute dermatitis. It has been reported to cause contact dermatitis followed by EM in sensitized patient.[9,10] Other different chemicals causing EM are bromofluorene, colophony, fragrances, epichlorhydrine, 1,2-ethanedithiole,[11] and epoxy resin.

This case is reported for the uncommon presentation of erythema multiforme as a consequence of application of hair dye and also to highlight the importance of performing the patch test before each application of the dye. According to best of our knowledge, this is the first report of erythema multiforme following allergic contact dermatitis to hair dye from the Indian subcontinent.

Footnotes

Source of Support: Nil

Conflict of Interest: Nil.

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Figures and Tables

Figure 1

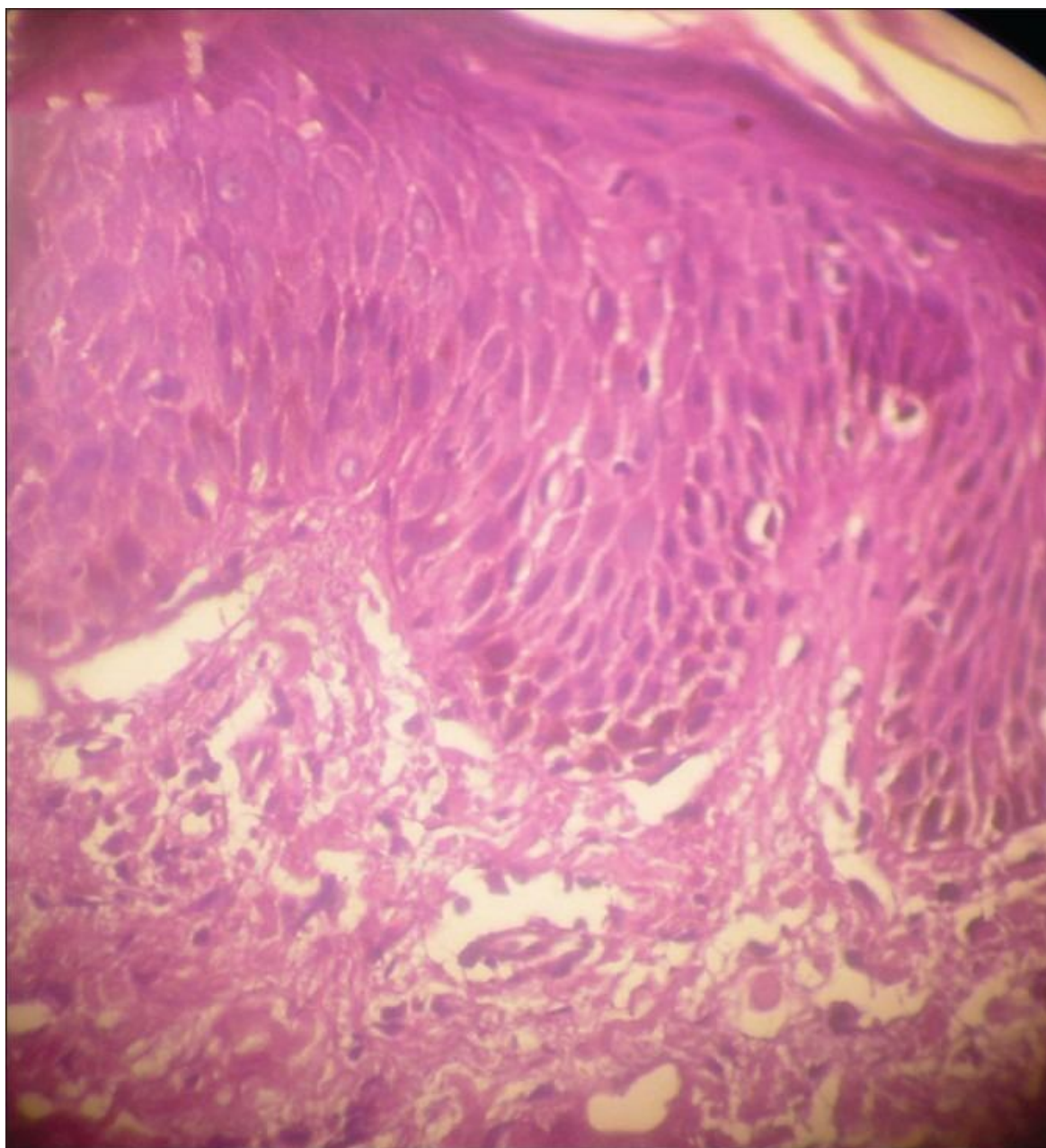
Severe contact dermatitis caused by black hair dye after 3 weeks of application: the face showing erythema, edema, vesicles, erosions, and widespread crusting

Figure 2

Involvement of ears and adjoining face with relative sparing of the scalp

Figure 3

Multiple papules, plaques and target lesions on bilateral hands and back

Figure 4

Photomicrograph showing interface dermatitis along with subepidermal clefts and mild dermal edema (hematoxylin and eosin $\times 40$)

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