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Original Article

Black hair dye dermatitis is a major cause of concealed facial melanosis in patients with dark complexion

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Abstract

Background Hair dye dermatitis is a common problem that is localized to the head or even generalized, and in many countries, it runs as an outbreak problem. Many patients present with facial melanosis, without an obvious history of dermatitis.

Objective To record patients with facial melanosis that was triggered by black hair dying and to highlight this concealed cause of facial pigmentation.

Methods This is a cross-sectional descriptive study where 102 patients with black hair dye facial melanosis were seen during the period from 2014-2022. All patients with Fitzpatrick skin type III and IV. Full demographic features were clarified. History and examination were carried out, and skin biopsies for histopathological assessment were done in selected cases. All other causes of facial melanosis were excluded. The therapeutic trial used Mometasone Furoate 0.1% cream twice a day for one month.

Results A total of 102 patients with facial melanosis were analyzed; their ages ranged from 30-60 years with a mean of 50 years,70 (68.6%) males and 32 (31.4%) females with a ratio of 2:1. History and examination showed that all patients were applying black hair dye from 0.5-4 years and the duration of facial pigmentation ranged from 1-5 years. Patients mentioned some itching of the face, while others mentioned a history of an erythematous rash of the face in the early course of the disease. On examination, all patients revealed deep brown to dark hyperpigmentation of the face, neck, and hands. Woods light examination gave no contrast, while histopathological assessment showed an increase in the melanin stores both in the epidermis and dermis. Topical therapy with Mometasone Furoate, 0.1% cream, gave a very satisfactory response by lightening the skin.

Conclusion Black hair dye is an important cause of facial melanosis that is often overlooked and confused with other causes of facial melanosis, commonly diffuse lichen planus actinicus, as a result its therapy will be very difficult unless hair dying is prevented. Therapy with mometasone Furoate 0.1% cream showed a satisfactory result.

Key words
Black hair dye; PPD; Facial melanosis; Lichen planus actinicus.

Introduction

The term facial melanosis covers a wide variety of disorders causing hyperpigmentation involving mainly the face and neck. It is relatively common and often presents a challenging diagnostic problem. Those with dark skin, particularly those of Middle Eastern or Asian descent, are more likely to have increased pigmentation due to genetic and racial factors.[1-2]

The common pigmentary disorders that cause facial melanosis are melasma, frictional melanosis, lichen planus actincus (butterfly-like pigmentation), acanthosis nigricans, gazelle eye like facial melanosis, postinflammatory hyperpigmentation, phytodynamic dermatitis, Riehl's melanosis, naevus of Ota and drug-induced hyperpigmentation.[3-7] An outbreak of black hair dye dermatitis and facial melanosis has been recently reported by Sharquie *et al.*[8] The clinical appearance of all pigmentary disorders is remarkably similar, posing a diagnostic challenge for dermatologists.[9] It is believed that hyperpigmentary disorders result from an increase in melanocytes or an increase in their activity, resulting in excess melanin production.[10] Pigment excess can occur on the epidermis or dermis, and it can be localized or diffuse. Melanin production or melanosomal transfer in keratinocytes contributes to the brownish color of epidermal pigments.[11] As a result of the Tyndall effect, dermal pigment is colored blue or gray because melanin is deposited from the basal cells into the dermis (dermal pigment incontinence).[3,12]

The process of dyeing hair involves exposing hair to a solution of propylene glycol and various chemicals, such as ammonia, chloroform, and paraphenylenediamine (PPD).[13] Various adverse effects can occur as a result of hair dyeing products, including allergic contact dermatitis, irritant contact dermatitis, photo contact dermatitis, contact urticaria, contact leukoderma, contact anaphylaxis, lichenoid eruption, actinic dermatitis, facial melanosis, erythema multiforme like eruption.[8,14,15] As reported by Sharquie *et al*. the number of cases of hair dye dermatitis in Iraq is on the rise, forming an outbreak with a male-to-female ratio of 5.2:1 and a mean age of 52.3 years.[8] The main ingredient in permanent hair dye is PPD, which is one of the leading causes of allergic contact dermatitis. Oxidation of PPD produces benzoquinone, P-aminophenol, and N-phenyl-PPD, which are potent sensitizers. Additionally, additives such as pyrogallol and resorcinol can trigger allergic reactions.[16]

The use of hair dyes has been implicated in the development of facial-pigmented contact dermatitis, lichen planus pigmentosus, acquired dermal macular hyperpigmentation, and depigmentation in some cases.[8,17,18] There are, however, very few studies examining facial melanosis in hair dye users.[19]

The development of contact dermatitis following using hair dyes due either to non-immunological mechanisms and is associated with the irritating effect on the skin of various ingredients of hair dyes or more commonly follow allergic contact dermatitis (ACD) formation which is more complex and is usually caused by delayed-type hypersensitivity, which develops when the skin comes into direct contact with chemical allergens. A prerequisite for the development of ACD is prior sensitization, which is called the induction phase. After this initial contact of the antigen with the skin, Langerhans dendritic cells that are located in the supra layer of epidermis migrate to the lymph node near the site of contact with the antigen. CD4+T lymphocytes react to the formed peptide fragment of the antigen and then proliferate and differentiate into Th1-cells.[20] After repeated contact with the allergen, already sensitized Th1 cells are sent to the contact site, activating the resident and then migrating macrophages. This contributes to the development of inflammation with a

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predominance of cell infiltration over vascular changes, thus, starting the phase of clinical manifestations. In the development of ACD, the role of other groups of cytokines is much more important, such as lymphokines produced by Th1 cells, pro-inflammatory cytokines secreted by macrophages, and chemokines produced by these cells and stromal elements.[21] The ACD develops more easily in individuals with a hereditary predisposition to allergic diseases or in people with foci of chronic infection that create an appropriate background.

The objective of the present work is to record all cases presenting with facial melanosis that had history of using black hair dyes and after excluding other causes of facial melanosis.

Methods

One hundred and two (102) patients complaining of facial melanosis gathered during the period from 2014-2022 years were involved in this cross-sectional descriptive, clinical-histopathological study. All patients with Fitzpatrick's skin type III and IV. The declaration of Helsinki was followed during the study. Informed Consent forms were obtained from all patients after discussing the nature of the study. The close-up photo was taken at the same place with a fixed distance and illumination. In addition, all included patients accepted the idea to share their photos in this present work. Full epidemiological and demographic features were recorded. A paraphenylenediamine (PPD) dye has been found to have been the scientific name of all dyes used by patients. A thorough full history to establish the right clinical diagnosis with a well-established examination was done. Name, age, gender, residence, occupation, personal or family history of atopy (nasobronchial allergy, asthma, childhood eczema), duration of the lesions, chief complain, associated symptoms such as redness, sores, itching, burning sensation, and discomfort, recurrence, aggravating and relieving factors, the site, type, geographical distribution of the lesion, types of hair dye being used and its duration, job, family history, past medical, drug history. Incisional biopsies were done for histopathological assessment and were carried out as a confirmatory test. The other causes of facial melanosis like melasma, diffuse lichen planus actinicus, frictional melanosis and others were excluded both on the clinical and histopathological basis. The therapeutic trial used Mometasone Furoate 0.1% cream twice a day for one month.

Results

One hundred and two (102) patients complaining of facial melanosis were considered in the present work, their ages ranged from 30-60 years with a mean 0f 50 years, 70 (68.6%) males and 32 (31.4%) females with a male-to-female ratio of 2:1.

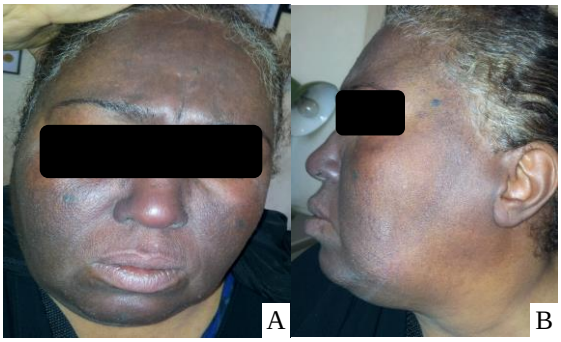

Figure 1 A 45-year-old female showing black hair dye facial melanosis of face and neck.

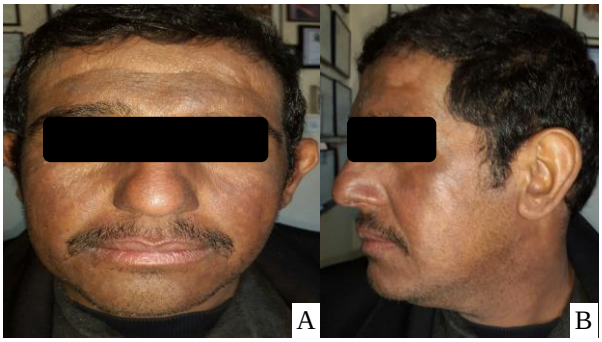

Figure 2 A 38-year-old patient showing diffuse black hair dye dark facial melanosis.

Figure 3 A 45-year-old male only with marked black hair dye facial melanosis.

History and examination showed that all patients were applying black hair dye from 0.5-4 years and the duration of facial pigmentation ranged from 1-5 years. Patients mentioned some itching of the face while others mentioned a history of erythematous rash of the face in the early course of the disease. On examination, all patients revealed deep brown to dark hyperpigmentation of the face, neck, and hands in a diffuse uniform pattern, although some patients had trunkal involvement. Woods light examination gave no contrast while histopathological assessment showed an increase in the melanin stores both in the epidermis and dermis in addition to mild dermal inflammatory infiltrate. Topical therapy with Mometasone Furoate 0.1% cream gave a very satisfactory response by lightening the skin **(Figures 1-4; Tables 1-3).**

Table 1 Sociodemographic features of the study (n=102).

Characteristics	*n (%)*
Age in years	
- Min.	30
- Max.	60
Gender	
- Male	70 (68.6%)
- Female	32 (31.4%)

Table 2 Duration of hair exposure and duration of disease symptoms (n=102).

Characteristics	*Duration in years*
Duration of black hair dye application	
- Min.	0.5
- Max.	4
Duration of Facial Pigmentation	
- Min.	1
- Max.	5

Discussion

A lack of attention has been given to the problem of facial melanosis caused by hair dyes on the face and neck. In a Korean study, Han *et al.* reported hyperpigmentation in 3.8% of hair dye allergies.[22] In an Indian study, Patel *et al.* reported pigmentation in 10% of hair dye users in India.[23] As a result of two previous studies by Sharquie *et al*., facial melanosis has been reported to exist in (5.3%) of the patients in the first study[8] and (43.6%) in the second. The lateral forehead (88.3%), the helix of the ear (88.3%), the central forehead (80.8%), and the zygomatic area (6.7%) were the most frequently affected areas in a study performed by Meghana *et al.*[19] According to the present study, the distribution of facial melanosis was found to be prevalent in the face, neck, and hands in diffuse uniform pattern. The color of the skin is considered a risk factor for the intensity of pigmentation on the skin. As a general rule, darker skin types are more likely to suffer from post-inflammatory hyperpigmentation as revealed by the present work, including distinct morphological types such as pigmented contact dermatitis, and lichen planus pigmentosus.[24] In addition, this may also play a role in developing the facial melanosis that is caused by hair dyes. As compared to dyes that originate from Europe, paraphenylenediamine is more common and is present in higher concentrations in dyes that originate from non-European countries.[19] It is recommended that paraphenylenediamine should not exceed 2% in the end product after mixing in oxidative conditions, and toluene-2,5-diamine should not exceed 4%.[25] There may also be a link between higher concentrations of paraphenylenediamine in poorly regulated dyes and the higher proportion of pigmentary changes seen in these dyes. Based on the results of the current study, we determined that the mean age of individuals with facial melanosis was 50 years with a male-to-female ratio of 2:1 with Fitzpatrick's skin type III and IV. We found

Table 3 Clinical description of patient with black hair facial melanosis (n=102).

Disease Characteristics:
History: Facial itching; erythematous rash of the face in the early course of the disease.
Examination: Deep brown to dark diffuse hyperpigmentation of face, neck and hands.
Woods light examination gave no contrast.
The histopathological assessment showed an increase in the melanin stores both in the: epidermis and dermis.
Topical therapy with Mometasone Furoate 0.1% cream gave a very satisfactory response by lightening the skin.

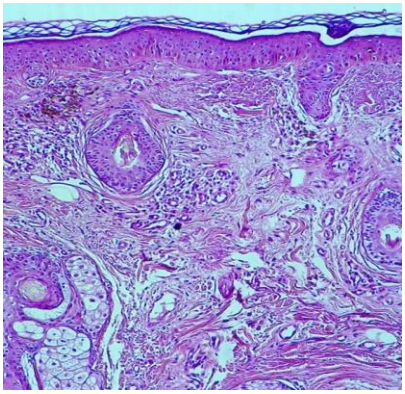

Figure 4a Histopathological picture of black hair dye melanosis showing increased melanin stores in the epidermis and dermis HE stain 20X.

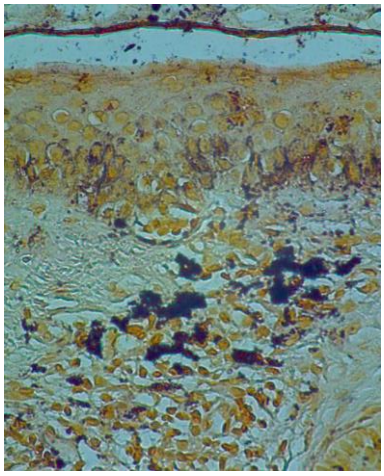

Figure 4b The same patient with Fontana stain showing melanophages loaded with melanin in dermis and increased epidermal melanin stores in a patient with black hair facial melanosis X20.

that itching was the main symptom of facial melanosis in the present study and that erythematous rash of the face appeared in the early phases of the disease. In most cases of the present study, on examination, there were areas of hyperpigmentation ranging from deep brown to dark brown, and Wood's light examination revealed no obvious contrast on the skin. Upon histopathological examination, it was found that both the epidermis as well as the dermis had increased levels of melanin in addition to mild dermal inflammatory infiltrate. As a result of topical treatment with Mometasone Furoate 0.1% cream twice a day for one month, significant improvements in the condition of the skin have been observed. The cause of facial melanosis being black hair dye has not been mentioned before; however, this is the first study that explores this subject in greater detail.

Conclusion

Black hair dye dermatitis is an important cause of facial melanosis in the absence of other features of hair dye dermatitis that is often overlooked and commonly confused with diffuse lichen planus actinicus and other causes of facial melanosis where its therapy will be very difficult unless hair dying is prevented. Therapy with Mometasone Furoate 0.1% cream showed a satisfactory result.

References

1. Khanna N, Rasool S. Facial melanoses: Indian perspective. *Indian J Dermatol Venereol Leprol*. 2011;**77(5)**:552-63;quiz 64.
2. Bandyopadhyay D. Topical treatment of melasma. *Indian J Dermatol*. 2009;**54(4)**:303-9.
3. Prasad V, Mohta A, Mehta RD, Ghiya BC, Soni P, Pareek S. Clinical and dermatoscopic correlation in facial melanosis: A cross-sectional study at a tertiary hospital. *Our Dermatol Online*. 2023;**14(2)**:161-9
4. Dharman BK, Sridhar S. Diffuse facial melanosis-An overview of etiology and

dermoscopic findings. *J Skin Sex Transm Dis*. 2020;**2(2)**:86-93.
5. Sharquie KE, Ahmad EM. Lichen planus actinicus: Clinical and histopathological study. A Diploma Dissertation in Dermatology and Venereology, College of Medicine, University of Baghdad. 1991.
6. Sharquie KE, Noaimi AA, Al-Ogaily SM. Acanthosis Nigricans as a Cause of Facial Melanosis (Clinical and Histopathological Study). IOSR Journal of Dental and Medical Science. *IOSR J Dent Med Sci*. 2015;**14**:84-90
7. Sharquie KE, Noaimi AA. Gazelle Eye like Facial Melanosis (Clinico-Histopathological Study). *J Pigment Dis*. 2014;**1**:1-2
8. Sharquie KE, Noaimi AA, Hasan Ra. Outbreak of Hair Dye Dermatitis with Imitating Tendency. *Am J Dermatol Venereol*. 2020;**9(1)**:1-5.
9. Hourblin V, Nouveau S, Roy N, de Lacharrière O. Skin complexion and pigmentary disorders in facial skin of 1204 women in 4 Indian cities. *Indian J Dermatol Venereol Leprol*. 2014;**80(5)**:395-401
10. Halder RM, Grimes PE, McLaurin CI, Kress MA, Kenney JA, Jr. Incidence of common dermatoses in a predominantly black dermatologic practice. *Cutis*. 1983;**32(4)**:390.
11. Alexis AF, Sergay AB, Taylor SC. Common dermatologic disorders in skin of color: a comparative practice survey. *Cutis*. 2007;**80(5)**:387-94.
12. Grimes PE. Management of hyperpigmentation in darker racial ethnic groups. *Semin Cutan Med Surg*. 2009;**28(2)**:77-85.
13. Doddamani SH, Bhavya BM, ShubhashreeM N, Naik RM, Thakur R. Management of hair dye related contact dermatitis: A case report. *Int J Res Ayurveda Pharm*. 2020.
14. Sahoo B, Handa S, Penchallaiah K, Kumar B. Contact anaphylaxis due to hair dye. *Contact Dermatitis*. 2000;**43(4)**:244.
15. Gupta M. Hair-Dye induced erythema multiforme like allergic contact dermatitis. *Our Dermatol Online*. 2017;**8**:177-8.
16. Handa S, De D, Mahajan R. Epidemiological trends in contact dermatitis to hair dye: Comparing para-phenylenediamine positivity after a decade long interval. *Indian J Dermatol Venereol Leprol*. 2011;**77(4)**:511-2. doi: 10.4103/0378-6323.82386.
17. On HR, Hong WJ, Roh MR. Low-pulse energy Q-switched Nd:YAG laser treatment for hair-dye-induced Riehl's melanosis. *J Cosmet Laser Ther*. 2015;**17(3)**:135-8.
18. Woo YR, Kim JS, Lim JH, Choi JY, Kim M, Yu DS, et al. Acquired diffuse slate-grey facial dyspigmentation due to henna: an unrecognized cause of pigment contact dermatitis in Korean patients. *Eur J Dermatol*. 2018;**28(5)**:644-8.
19. Meghana V, Gopinath H, Karthikeyan K, Venugopal V. Face and Neck Pigmentary Alterations in Hair Dye Users: A Cross-sectional Study from South India. *Indian Dermatol Online J*. 2020;**11(5)**:760-5.
20. Rubin IM, Dabelsteen S, Nielsen MM, White IR, Johansen JD, Geisler C, et al. Repeated exposure to hair dye induces regulatory T cells in mice. *Br J Dermatol*. 2010;**163(5)**:992-8.
21. Bonefeld CM, Geisler C, Giménez-Arnau E, Lepoittevin JP, Uter W, Johansen JD. Immunological, chemical and clinical aspects of exposure to mixtures of contact allergens. *Contact Dermatitis*. 2017; **77(3)**:133-42.
22. Han JH, Lee HJ, Bang CH, Lee JH, Park YM, Lee JY. P-Phenylenediamine Hair Dye Allergy and Its Clinical Characteristics. *Ann Dermatol*. 2018;**30(3)**:316-21.
23. Patel D, Narayana S, Krishnaswamy B. Trends in use of hair dye: a cross-sectional study. *Int J Trichology*. 2013;**5(3)**:140-3.
24. Rodrigues M, Ayala-Cortés AS. Post-inflammatory Hyperpigmentation. In: Kumarasinghe P, editor. Pigmentary Skin Disorders. Cham: Springer International Publishing; 2018. p. 197-208.
25. Antelmi A, Bruze M, Zimerson E, Engfeldt M, Young E, Persson L, et al. Evaluation of concordance between labelling and content of 52 hair dye products: overview of the market of oxidative hair dye. *Eur J Dermatol*. 2017;**27(2)**:123-31.