

A CASE OF CHEMICAL LEUKODERMA EFFECTIVELY TREATED WITH PUNCH GRAFT SURGERY

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ABSTRACT

Chemical leukoderma is a hypomelanotic disorder due to destruction of melanocytes or inhibition of melanogenesis secondary to application of harmful chemicals household or industrial. It can be easily diagnosed clinically by a positive history of frequent exposure to a depigmenting agent at the site corresponding with the pattern of the object applied. It has a psychosocial significance because of the dyspigmented presentation of the disease which often simulates other conditions associated with social stigma, like leprosy or vitiligo. Here, we report a case of thirty year old female with chemical leukoderma who presented to us with depigmented patches over forehead (bindi leukoderma) and dorsae of feet (footwear dermatitis) caused by application of sticker bindi and wearing of rubber footwear respectively. Complete repigmentation was observed after treatment with topical corticosteroids and melanocyte transfer surgery.

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INTRODUCTION

Exposure of skin to certain chemicals have a destructive effect on melanocytes resulting in hypopigmentation or depigmentation known as chemical leukoderma. It is caused by either destruction of melanocytes or inhibition of melanogenesis (1-5).

The condition is clinically diagnosed by a positive history of repeated exposure to a known or suspected depigmenting agent at the site corresponding with the pattern of the article (6-7).

Patient's skin color, mostly because of the patchy depigmentation has a psychosocial significance as it is regarded a stigma in our society. Most of the distressed patients seen by dermatologist are patients with depigmented skin lesion. They are regarded as unclean, contagious and are often perceived as a case of leprosy (8).

We hereby report a case of thirty year old female with chemical leukoderma that showed an excellent response to punch graft surgery.

CASE REPORT

A thirty year old housewife presented to us with a solitary round depigmented macule on the forehead and linear depigmented macules on dorsae of both feet. She had no past medical history including autoimmune disease. There was no relevant family history. History of application of sticker bindi on forehead and wearing of hawaiian slippers with rubber

straps was present since last three years. On examination the pattern of depigmentation on forehead (Fig. 1a) and feet (Fig. 1b) matched with her bindi and strap of footwear respectively.



Fig 1a: Hypopigmented Macule Over Forehead



Fig 1b: Linear Hypopigmented Macules Over Feet

The condition had affected patient's quality of life adversely as she often complained of restriction in social activities and mood disturbances due to leukoderma. A clinical diagnosis of chemical leukoderma was made. The patient was advised to avoid use of bindi and rubber footwear completely. She was counselled and treated with mometasone furoate 0.1% cream and tacrolimus 0.1% ointment for 2 months with partial response (Fig. 2a,2b).



Fig 2a: Partial Response To Topical Corticosteroids Over Forehead



Fig 2b: Partial Response To Topical Corticosteroids Over Feet



Fig 3a: Complete Response After Punch Graft Surgery Over Forehead



Fig 3b: Complete response after punch graft surgery over feet

Punch graft surgery was performed and the remaining depigmented areas were covered with 2 millimeter punch grafts harvested from patient's upper thigh. A complete repigmentation was observed after 3 months (Fig. 3a,3b).

DISCUSSION

Chemical leukoderma is a disorder of acquired hypopigmentation which is caused by repeated exposure to different chemicals, and clinically simulates idiopathic vitiligo (4). It is reported that *p*-tertiary butyl phenol (PTBP) in the adhesive glue in the bindi, and monobenzyl ether of hydroquinone (MBH) also known as "Agerite alba" which is a rubber antioxidant cause chemical leukoderma (9-12). There are a variety of chemicals causing chemical leukoderma, most of them are aromatic or aliphatic derivatives of phenol or catechol (7). These chemicals inhibit tyrosinase which is essential for the synthesis of normal melanin pigment (13).

Diagnosis is mainly clinical which can be confirmed by a patch test. Management of chemical leukoderma is complete discontinuation of contact allergen and topical corticosteroids which is the first line of treatment (14).

Timely diagnosis of this condition is significant as identification and elimination of contact of the concerned chemical containing apparel/object will result in formation of normal melanin and recovery from the disease.

We report this case to highlight the importance of surgical management of chemical leukoderma in resistant cases. An early recognition and complete treatment is essential owing to the psychosocial comorbidity associated with chemical leukoderma.

CONCLUSION

Chemical leukoderma is an acquired hypopigmentary disorder caused by contact to certain household or occupational objects. It has a psychosocial significance because it mimics vitiligo leading to social stigma. Chemical leukoderma runs a good prognosis with timely diagnosis and management. Diagnosis is mainly clinical, and avoidance of allergen with topical corticosteroids form the mainstay of treatment. Resistant cases may require phototherapy or melanocyte transfer surgery as in our case.

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