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SHORT COMMUNICATIONS

PROBABLE MECHANISM OF BINDI-INDUCED DEPIGMENTATION

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Several cases of bindi-induced depigmentation have been presented in the past but the mechanisms underlying para-tertiary-butyl-phenol induced-depigmentation remain unclear. A case is presented here in order to emphasize the current support on immune mediated mechanisms leading to damage to the melanocytes.

Key Words : Contact depigmentation, Chemical depigmentation, Para-tertiary-butyl-phenol

Introduction

Contact depigmentation of the forehead is a common feature seen especially among the rural folks in India, due to the habit of using bindi. A white patch on the forehead is a cause for concern especially in a dark-skinned individual; this cosmetic disfigurement leads to psychological turmoil.[1] Due to sometimes finding of additional patches of depigmentation elsewhere,[2] further aggravates the agony due to fear of vitiligo.

Until recently, not much was known concerning the mechanisms leading to depigmentation due to adhesive containing para-tertiary-butyl-phenol (PTBP). Here I present a case and discuss the probable causes underlying PTBP (adhesive) induced depigmentation.

Case report

A 45-year-old Indian women from a village near Delhi, presented with depigmentation of forehead of 3 years duration in March 1987. Earlier she had noticed a mild erythema which resolved with a hypopigmented patch later turning white. There was no family history of vitiligo or other autoimmune diseases. She was otherwise normal with normal haemogram, liver function tests, and thyroid function. No analysis for circulating antibodies to melanocytes was performed. Patch test with bindi adhesive was positive showing mild irritation. However no depigmentation was observed later in the area tested. Patient had had earlier treatment with local 8-methoxy psoralen plus sunlight and fluocinolone acetonide ointment without any apparent result. The patient did not respond to discontinuation of the use of bindi. Thin Thiersch grafting performed a year later, gave excellent cosmetic result with matching with the surrounding skin colour within a year.

Discussion

Depigmentation of the forehead with PTBP in bindi adhesive is common and has been reported by several authors.[3-6] Para-tertiary-butyl-phenol induced depigmentation was noticed in industrial workers exposed to this chemical.[2-7] Vitiligo-like depigmentation was seen in

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the area of contact. Recently, extensive studies after analyzing bindi adhesive in 100 consecutive cases revealed that 80% of these cases were related to para-tertiary-butyl-phenol.[5] Test showing depigmentation was observed in only 3 of 15 patients tested with bindi adhesive material. This seems reasonable as only few cases have been found to be positive (depigmentation) to PTBP when 1220 suspected patients were patch tested.[8] Usually very few cases of bindi depigmentation are seen compared to thousands of women using bindi regularly in India. These authors suggested an increased susceptibility and/or prolonged exposure.[5] In another recent article, they hypothesized a toxic mechanism in a report of 2 cases of bindi depigmentation.[4]

More recently,[3] histological analysis were performed from contact depigmented skin of 12 patients due to the use of bindi out of which 8 showed a positive reaction. Histology of all the cases showed predominantly dermal vasculitis with perivascular mononuclear infiltrate and melanin incontinence. These authors proposed an immunological reaction by the chemical resulting in contact depigmentation.

Table I. List of phenols, catechols and related compounds[9]

Alkyl phenol	Catechol
P-tertiary butylphenol	4-Isopropylcatechol
P-tertiary amylphenol	Methyl catechol
	P-tertiary butylphenol
Hydroquinone	
Monobenzyl ether of hydroquinone	
Monoethylether of hydroquinone	
Monomethylether of hydroquinone	
Butylated hydroxytoluene	
Dihydroxyphenylmethane	

The list of phenols, catechols and related compounds is growing (Table I). It seems that the optimal depigmentation from phenols and catechols occurs when the 4 position of an aromatic ring is hydroxylated and position 1 has a nonpolar side chain.[9] The detailed mechanisms of chemical induced depigmentation are not yet known. Several theories have been proposed[9] such as : 1. melanocyte destruction via the immune system in contact-sensitized skin (postinflammatory hypopigmentation); 2. melanocyte destruction via free radical formation induced by the depigmenting agent; 3. inhibition of the synthesis of tyrosinase; 4. interference with the production of melanosomes to keratinocytes.

Histology of depigmentation of completely white patch of stable vitiligo, and white patch of long standing para-tertiary-butyl-phenol adhesive is identical with normal appearing epidermis devoid of melanocytes.[9,10] In active vitiligo vulgaris frequently a mononuclear infiltrate is observed at the periphery of the advancing lesions,[11,12] somewhat similar to that has already been reported in contact depigmentation.[3] Antimelanocyte antibodies have been demonstrated in 80-85% cases of vitiligo.[13] Antibenzene ring antibodies have recently been found in the sera of vitiligo patient suggesting that exposure to chemicals with benzene ring may play a role in the induction of immunological injury in these patient.[14,15] This may explain the dissemination seen with PTBP that may occur far from the primary site in a few cases probably with a similar mechanism as observed in vitiligo. All these findings lead us to suggest that both cell mediated and humoral immune

mechanisms may be responsible for the cause of local and disseminated patch if any in PTBP-induced depigmentation. However, the precise mechanism would be clear after demonstration of anti-melanocyte antibodies and immuno-phenotyping of the cellular infiltrates in PTBP-induced depigmentation.

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