

Leukoderma following occupational allergic contact dermatitis

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We describe 2 cases of occupational allergic contact dermatitis followed by leukoderma. The 1st case was a 49-year-old wood machinist who developed leukoderma in areas of contact dermatitis involving his lips, neck, hands and forearms and was found to be allergic to colophony and pine sawdust on patch testing. The 2nd case involved a 44-year-old man who worked as an epoxy applicator in a ceramics factory. He developed depigmentation in areas of contact dermatitis involving his face, hands, forearms, back, thighs and legs and was found on patch testing to be allergic to epoxy resin. The appearance of contact leukoderma may be indistinguishable from idiopathic vitiligo. However the prognosis for repigmentation may be better in contact leukoderma than in idiopathic vitiligo.

Key words: occupational; allergic contact dermatitis; leukoderma; vitiligo; depigmentation; colophony; epoxy resin; wood machinist; epoxy applicator. © Munksgaard, 1999.

Accepted for publication 9 April 1999

Leukoderma following allergic contact dermatitis is rare. Similarly, leukoderma resulting from chemical contactants that have a toxic effect on melanocytes is uncommon. Clinically and histologically, it may be similar to vitiligo. The 1st case of toxic leukoderma following occupational contact was reported in 1939, in tannery workers exposed to monobenzyl ether of hydroquinone present in rubber gloves (1). Since then, a variety of chemicals causing this type of leukoderma have been described (2). The mechanism of depigmentation in these cases of contact leukoderma is either due to damage to melanocytes or inhibition of melanin synthesis by the offending agents.

We report 2 cases of occupational contact leukoderma which were not due to contact with depigmenting chemicals but followed allergic contact dermatitis due to colophony and epoxy resin, respectively.

Case Report

Case no. 1

A 49-year-old darkly pigmented Samoan man presented with a 4-month history of an itchy vesicular dermatitis involving his forehead, lips, back of the neck, hands and forearms. A week after its onset, he developed depigmentation in all areas corre-

sponding to his dermatitis, except his forehead (Figs. 1, 2). He had worked as a wood machinist for 10 years. His work involved handling timber and he was thus exposed to sawdust. However 4 months before presentation, he began handling MDF particle board which is made of pine. His rash improved after 1 week off work but worsened on return to work. There was no personal or family history of either atopy or vitiligo. His general health was good. Topical corticosteroids improved his dermatitis, but the leukoderma persisted.

Table 1. Patch test results in case no. 1

No	Allergens tested	Results
1	European standard series	+ to colophony
2	plastics and glues series: phenol-formaldehyde resin (PFR) 5%, para-tertiary-butylphenol (PTBP) 2%, para-tertiary-butylcatechol (PTBC) 1%, toluene-sulfonamide-formaldehyde resin (TSFR) 10%, diaminodiphenylmethane (DDM) 0.5%, triethylenetetramine (TET) 0.5%, di-n-butylphthalate (DNBP) 5% and dimethyl-p-toluidine (DMPT) 2%, all in pet.	-
3	Samples of saw dust from workplace (in vaseline)	+

Table 2. Patch test results in case no. 2.

No	Allergens tested	Results
1	European standard series	+ to epoxy resin and benzocaine
2	plastics and glues series: phenol-formaldehyde resin (PFR) 5%, para-tertiary-butylphenol (PTBP) 2%, para-tertiary-butylcatechol (PTBC) 1%, toluene-sulfonamide-formaldehyde resin (TSFR) 10%, diaminodiphenylmethane (DDM) 0.5%, triethylenetetramine (TET) 0.5%, di-n-butylphthalate (DNBP) 5% and dimethyl-p-toluidine (DMPT) 2%, all in pet.	—

He was patch tested and the results are shown in Table 1. Significantly, he showed a + positive reaction to colophony (20% pet.) as well as to samples of pine sawdust (pet.) at D3 and also at D7. There was no depigmentation at the patch test site. A diagnosis of airborne allergic contact dermatitis from colophony present in the pine sawdust was made. He was forced to leave his job in order

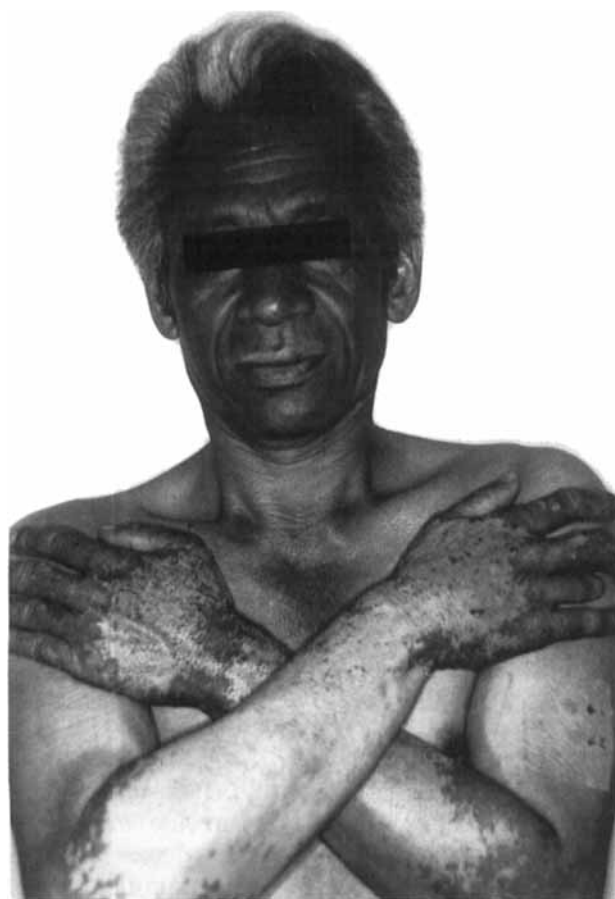


Fig. 1. Depigmentation in areas of allergic contact dermatitis (case no. 1).



Fig. 2. Depigmentation over the arms, close-up of Fig. 1 (case no. 1).

to avoid further contact with pine sawdust. Partial spontaneous repigmentation was noted at follow-up only 4 months later (Figs. 3, 4).

Case no. 2

A 44-year-old darkly pigmented South African man presented with a 12-month history of severe dermatitis involving his face, hands, forearms, back, thighs and legs. 3 months after its onset he developed leukoderma in areas corresponding to his dermatitis (Figs. 5, 6). He had worked as an epoxy applicator in a ceramics factory for 18 months and at the onset of his rash he had begun handling a new resin called AK epoxy. He started wearing rubber gloves at work only after the onset of his dermatitis. He had earlier been a painter for 7 years without developing any skin problems, despite the fact that he had occasionally used epoxy-containing paints. There was no personal or family history of atopy or personal history of vitiligo. There was no evidence of present or past thyroid disease, diabetes mellitus or other autoimmune disorders. His eye examination was normal.



Fig. 3. Partial repigmentation on follow-up after 4 months (case no. 1).

He was patch tested and the results are shown in Table 2. He had a + reaction to epoxy resin (1% pet.) and to benzocaine (5% pet.). He was working with epoxy resins. The benzocaine positive was of unknown relevance. There was no loss of pigment at the site of patch testing. His dermatitis improved after avoiding contact with epoxy resins. He was treated with PUVA therapy which resulted in significant skin repigmentation. At follow up after 10 years, he was reported to have repigmented except for the back of his hands.

Discussion

Contact leukoderma may occur from the toxic effect of a depigmenting chemical or as a result of a preceding allergic contact dermatitis. The mechanism is either destruction or inhibition of melanocytes by the offending substance. The depigmented areas may either spontaneously repigment or persist indefinitely. The appearance of contact leukoderma may be indistinguishable from idiopathic vitiligo. Gradual coalescence of hypopigmented macules without perifollicular sparing tends to favour toxic contact leukoderma (3). Ocular and per-

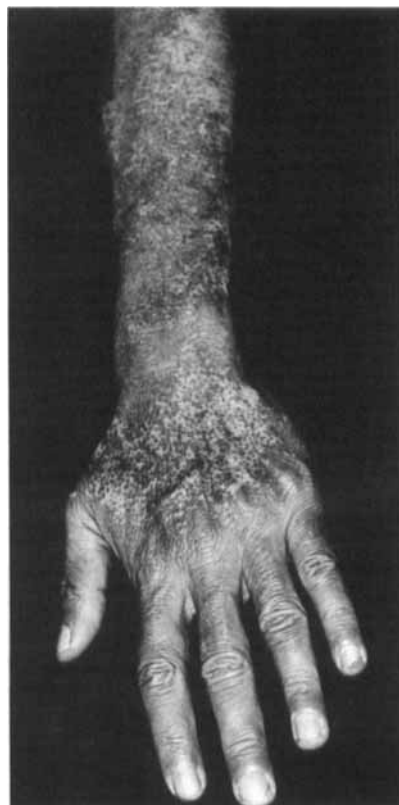


Fig. 4. Partial repigmentation of the arms after 4 months, close-up of Fig. 3 (case no. 1).

iocular depigmentation, if present, favours vitiligo (2). Histology and electron microscopy do not help in differentiating the 2 conditions (4, 5).

Both our cases were darkly pigmented and had no known diathesis of vitiligo. They developed leukoderma in areas corresponding to a preceding allergic contact dermatitis from colophony and epoxy resin, respectively. The completely depigmented skin contrasted sharply with their type V racially pigmented skin. The allergy was confirmed by patch testing. They did not develop depigmentation at patch test sites, although readings were not carried out between 3 and 6 weeks.

Contact leukoderma occurring as a toxic effect, without preceding dermatitis, has been reported secondarily to a number of substances, including hydroquinone and monobenzyl ether of hydroquinone. The latter is a potent depigmenting agent causing pigment loss even on skin distant from areas of contact. Phenols and catechols are also implicated, especially when their benzene ring is substituted in the para position. Para-tertiary-butylphenol (PTBP), found in germicide detergents, neoprene adhesives, glues, plastics, etc., can lead to contact leukoderma. PTBP-containing 'bindi', which is applied as a self-adhesive cosmetic mark on the forehead by Indian women, has been re-

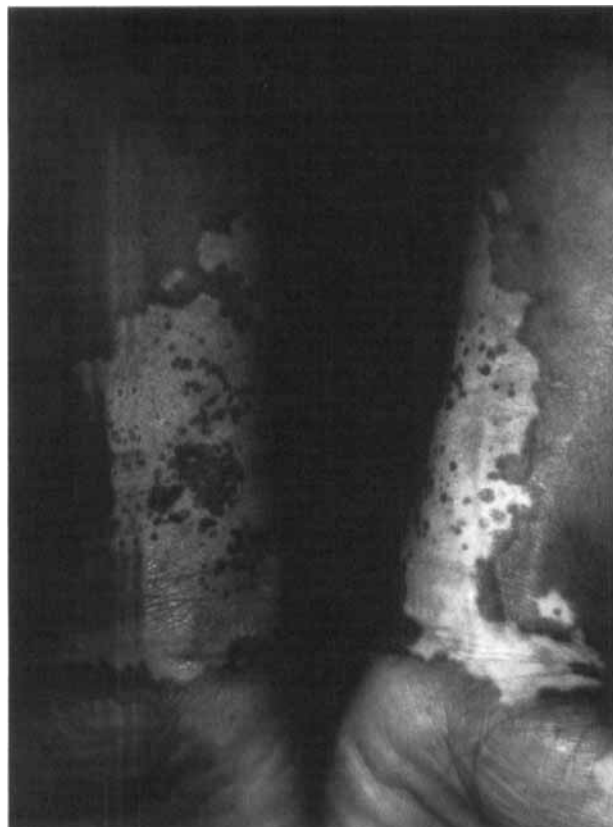


Fig. 5. Depigmentation over wrists at sites of allergic contact dermatitis (case no. 2).



Fig. 6. Depigmentation over the hands at sites of allergic contact dermatitis (case no. 2).

ported to cause reversible depigmentation (6). Occupational contact leukoderma has been caused by para-tertiary-butylcatechol (PTBC), which is used in the manufacture of rubbers, plastics, petroleum products and paints (7). Both PTBP and PTBC are structurally related to monobenzyl ether of hydroquinone (8). Contact with alkyl phenols can lead to loss of skin pigment in industrial workers (8).

Leukoderma has infrequently been reported after allergic contact dermatitis from substances including a cinnamic-aldehyde-containing toothpaste (9), hair colours and rinses (3), Carbyne[®] used as a herbicide (10) and alstroemeria (11). It has also been described after airborne contact allergic dermatitis from musk ambrette and santolol in burning incense (12). However, there are no previous reports of depigmentation or leukoderma following allergic contact dermatitis from colophony (pine) or epoxy resin. Conversely, generalized contact dermatitis has led to repigmentation of previous vitiligo (13).

Although various agents may cause leukoderma either due to a direct toxic effect or secondary to an allergic contact dermatitis, there may be an overlap in these mechanisms. Thus leukoderma

can occur after contact with PTBP as a toxic effect and has also been reported after allergic contact dermatitis with a PTBP-containing lip liner (14).

In conclusion we report 2 cases of occupational leukoderma after allergic contact dermatitis from colophony and epoxy resin, respectively. In contrast to idiopathic vitiligo, these cases are compensable.

References

1. Oliver E A, Schwartz L, Warren L H. Occupational leukoderma: preliminary report. *JAMA* 1939; 113: 927-928.
2. Fisher A A. Ask the experts: The differential diagnosis of contact leukoderma. *Am J Contact Derm* 1997; 8: 52-55.
3. Taylor J S, Maibach H I, Fisher A A, Bergfield W F. Contact leukoderma associated with use of hair colours. *Cutis* 1993; 52: 273-280.
4. Fisher A A. *Contact dermatitis*, 4th edition. Maryland: Williams and Wilkins, 1995: 765-777.
5. Stevenson C J. Occupational vitiligo: clinical and epidemiological aspects. *Br J Dermatol* 1981; 105: 51-56.
6. Bajaj A R, Gupta S C, Chatterjee A R. Contact depigmentation from free para-tertiary butyl phenol in bindi adhesive. *Contact Dermatitis* 1990; 22: 99-102.
7. Gawkrödger D J, Cork M J, Bleehen S S. Occupational vitiligo and contact sensitivity to para-tertiary butyl catechol. *Contact Dermatitis* 1991; 25: 200-201.
8. Gellini G A, Possick P A, Perone V B. Depigmentation

- from 4-tertiary butyl catechol: an experimental study. *J Invest Dermatol* 1970; 25: 190.
9. Mathias C G T, Maibach H I, Conant M A. Perioral leukoderma simulating vitiligo from use of tooth paste containing cinnamic aldehyde. *Arch Dermatol* 1980; 116: 1172.
10. Brandaccio R, Chamales M H. Contact allergy and depigmentation produced by the herbicide Carbyne[®]. *Contact Dermatitis* 1977; 3: 108.
11. Van Ketel W G, Verspyck Mijnsen G A, Neering H. Contact eczema from alstromeria. *Contact Dermatitis* 1975; 1: 323.
12. Hayakawa R, Matsunaga K, Arima Y. Depigmented contact dermatitis due to incense. *Contact Dermatitis* 1987; 16: 272-274.
13. Edwards Jr E K, Edwards Sr E K. Spontaneous repigmentation of vitiligo following generalised acute allergic contact dermatitis. *Contact Dermatitis* 1995; 33: 123.
14. Angelini E, Marinaro C, Carozzo A M. Allergic contact dermatitis of the lip margins from para-tertiary butyl phenol in a lip liner. *Contact Dermatitis* 1993; 28: 146-148.

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