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Two Cases of Nickel Dermatitis Showing Vitiligo-like Depigmentations

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The authors reviewed two patients showing "vitiligo-like depigmentations" where the skin had been in close contact with a metal spectacle frame made of nickel alloy. In spite of the hypersensitivity to nickel in both patients, they showed clinical and histologic findings indicate that the formation of "vitiligo-like depigmentation" does not result from posinflammatory hypopigmentation but from chemical hypomelanosis. We could not explain the underlying mechanisms; however, the speculation that the "vitiligo-like depigmentation" may come from the direct effect of the nickel itself, prompted us to report these cases.

Key Words: Nickel dermatitis, vitiligo-like depigmentation, nickel sensitivity

Nickel is one of the most common causes of allergic metal dermatitis. Nickel dermatitis may present as a well-defined patch of eczema or as a fissured granuloma of the ear, In the past 20 years the clinical features of nickel sensitivity have changed (Fisher, 1975). Presently, fewer patients present with obvious nickel dermatitis.

We report two cases of nickel dermatitis with unusual clinical features. The clinical and histologic resemblance with vitiligo prompted us to designate those leukodermas under the name of "vitiligo-like depigmentations".

REPORT OF CASES

The first case was a 40-year-old male who visited our hospital for the evaluation of symmetrical macules of unknown duration on both temples. Examination revealed a single pea-sized, hypopigmented, non-pruritic, well circumscribed macule on one side of the temple. Exactly the same sized macule was also noted on the contralateral side of the temple, where the skin had been in close contact with the metal spectacle frame for several years. He denied previous itching sensation or erythema. The patient was otherwise in good health. Results of complete blood cell count, erythrocyte sedimentation rate, urinalysis and liver function tests were within normal range. On patch test with the Hollister-Stier standard tray, the patient showed two positive reactions to nickel sulfate and one positive reaction to neomycin sulfate.

We were interested in the peculiar manifestation of the nickel sensitivity. However, we simply thought that the leukodermas were caused by a nonspecific inflammatory reaction of nickel dermatitis.

The second case was a 56-year-old male who first noted the appearance of tiny, non-pruritic macules on both temporal areas about one year before. He has been wearing spectacles for correcting myopia for more than twenty years. During more recent years he also has been using spectacles for correcting hyperopia. Physical examination disclosed pea-sized, well-marginated, hypopigmented macular patches located on exactly the same sites as the first case (Fig. 1). He also denied previous abnormal sensation or erythema.

Examination of the metal spectalce frames used

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Fig. 1. Single, pea-sized, well-marginated hypopigmented macule on left temple.

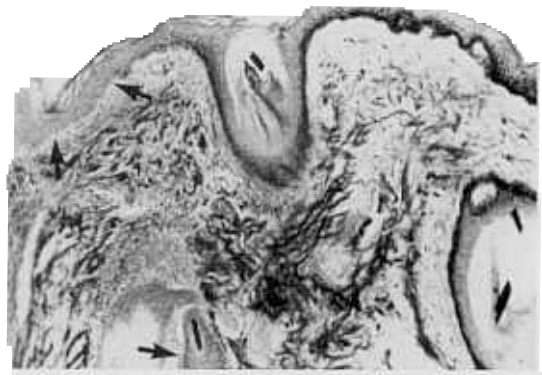

Fig. 3. Abrupt and complete absence of melanin in basal cell layer and hair follicle (arrows) of the skin lesion (Fontana-Masson stain, ×100).

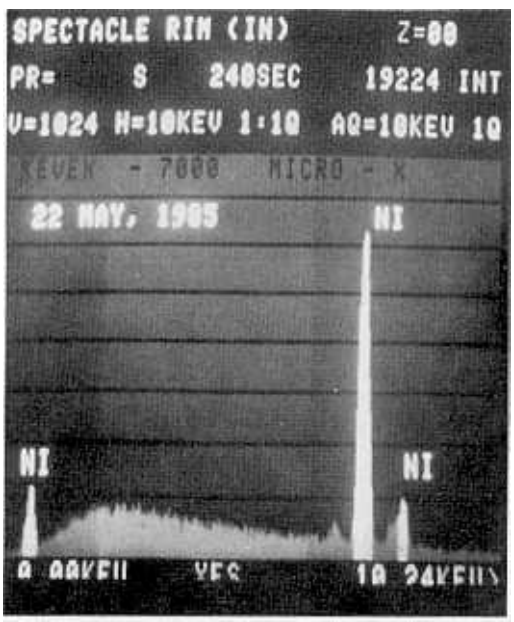

Fig. 2. Prominent peaks of nickel at the inner surface of the spectacle limd by x-ray spectrometry.

for correcting hyperopia disclosed the the coatings of the inner surface of the spectacle limbs were worn out. On patch test, the patient showed two positive reactions to nickel sulfate and a questionable reaction to ammoniated mercury. Howerer, negative reaction was noted to spectacle limb scrapings. The dimethylglyoxime spot test performed on the metal spectacle frame was positive only on the inner suface of the limb where the silver coatings were worn out. X-ray spectrometer analysis of the spectacle frome and tissue obtained from the lesion site was done. Prominent peaks of nickel were noted at the surface of the spectacle frame (Fig. 2). The outer surface where the coatings were intact showed peaks of nickel as well as a peak of gold. However, the peaks of nickel were not noted in the tissue. Except for the reduction or absence of melanin in basal cell layer, no other abnormalities were found in the biopsy specimen taken from the skin lesion. Fontana-Masson stain for melanin also disclosed a complete and abrupt absence of melanin in the basal cell layer of the lesional skin and outer root sheath of hair follicles (Fig. 3). No melanocytes were found with DOPA reaction.

The patient was advised to use plastic spectacle frame instead of using the metal spectacle frames. About four weeks after discontinuing use of the metal spectacle frames, the color of the lesion sites was slightly restored without specific treatment.

DISCUSSION

Nickel is widely known as a common source of contact dermatitis. Nickel sensitivity amounts to nearly ten percent of allergic contact dermatitis patients (Cronin, 1980). Silver-colored metal spectacle frames are generally made of an alloy containing 30 percent nickel, 1 to 3 percent manganese and 65 to 68 percent copper (Fisher, 1975). Nickel dermatitis seems to frequently develop on sites where direct contact with nickel substance has been made.

Casual use of the terms "vitiligo" and "leukoderma" has caused considerable confusion in the scientific literature over the last century up to this day.

However, vitiligo is more precisely defined as a circumscribed, acquired, idiopathic, progressive hypomelanosis of the skin and hair which is often familial and is characterized microscopically by total absence of melanocytes. The definition stressing an absence of etiology carefully excludes chemically-induced depigmentation secondary to various other dermatoses and other entities from which the precipitating factor seems clear (Ortonne *et al.* 1983).

Although the clinical appearance and histologic features of the leukodermas found in our patients resemble those of vitiligo, we prefer the term "vitiligo-like depigmentation" to describe this entity. Patch tests were positive for nickel in both patients, and nickel was detected from the spectacle frames by dimethylglyoxime spot test and x-ray spectrometer. In addition, the skin lesions developed only where the coatings were worn out. All these findings make it apparent that the leukoderma is related to nickel. Vitiligo is generally without an identifiable precipitating factor. Leutodermas of oui cases result from an identifiable precipitating factor. Therefore, it seems justifiable to separate these entities.

The probable explanation for the appearance of these "vitiligo-like depigmentations" may be that the eczema due to nickel hypersensitivity resulted in the hypopigmentation. Hypomelanosis can result from a wide variety of inflammatory dermatoses including eczematous dermatitis. The primary defect may be a pathologic change in the malpighian cells or an increased keratinocytes turnover. A disturbance in transfer of melanosomes from melanocytes to keratinocytes is probably responsible for the pigmentary dilution (Ortonne, 1983). However, although not proven by electron microscopy, melanin was completely absent in the biopsy specimen of the second case on Fontana-Masson stain, and DOPA reaction was negative. The margins of the lesions were sharp, and both patients denied past eczematous skin lesions or symptoms. These findings negate the possibility of a nonspecific posinflammatory hypopigmentation.

We suspected that the "vitiligo-like depigmentations" may be regarded as a chemical hypo-melanosis. A large number of chemical compounds can induce depigmentation in humans and in experimental animals. Cutaneous chemical depigmentation, which often resembles vitiligo in clinical appearance, may result from direct contact or from systemic exposure of various phenol derivatives, sulfhydryl compounds, and others (Fisher, 1976). Malten *et al.* (1971), in a comprehensive review of "occupational vitiligo", pointed out that depigmentation may occur without preceding irritation, dermatitis or burns. Fizpatrick (1973) indicated the leukoderma from contactants can readily be confused with the "idiopathic" variety, and that histologic examination is of no help since melanocytes are similarly degenerating in both conditions. Idiopathic vitiligo is usually permanent, while vitiligo due to contactants may sometimes clear if the causative contactant is removecd. There were no reports that nickel can act as a melanocyte degenerating agent. However, considering that mercury may inhibit melanin formation by competing with copper in tyrosinase, and therby interfere with the action of this enzyme (Lorincz, 1985), nickel also may act as a competitive inhibitor of copper. Frenk and Kocsis (1974) reported that contact vitiligo could be distinguished from the idiopathic variety by the presence of "unusual clear cells" in the electron microscope. Despite a lack of electron microscopic evidence, the absence of previous symptoms, clinical resemblance to vitiligo, negative DOPA reaction, complete absence of melanin, and slight improvement after avoidance of using the metal spectacle frames all lead us to conclude that the leukoderma may be induced by the yet unknown effect of nickel. It may be proved by animal studies with continued application of nickel compounds.

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