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Case Report

Mobile cover depigmentation

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ABSTRACT

Chemical-induced vitiligo is a condition where there is a persistent loss of pigment in the skin. For treatment, it is critical to distinguish it from vitiligo vulgaris. We describe a 43-year-old woman who had two depigmented patches, one covering her left cheek and the other her left breast.

Keywords: Depigmentation, Monobenzyl ether of hydroquinone, Thin-layer chromatography, Vitiligo

INTRODUCTION

Vitiligo caused by chemicals is an acquired loss of cutaneous pigment brought on by persistent exposure to certain chemicals. The results of a literature search revealed that many substances, particularly phenols and catechols, have been implicated in the development of chemically induced vitiligo.[1] This list includes hydroquinone monobenzyl ether of hydroquinone (MBEH), monoethyl ether of hydroquinone, monomethyl ether of hydroquinone, paratertiary butyl phenol, paratertiary amyl phenol, and alkyl phenol.

Ammoniated mercury, arsenic, benzoyl peroxide, brilliant lake red-R, chloroquine, cinnamic aldehyde, corticosteroids, eserine, gunanonitrofuracin, thiotepa, Para phenylene diamine (PPD), crocein scarlet moo, and solvent yellow 31 are a few more compounds that can induce contact depigmentation.

CASE REPORT

A 43-year-old housewife complained of two white patches over the past 18 months: one on the left side of the cheek, right in front of the left earlobe, and the other above the left breast [Figures 1 and 2]. The spots started as little but eventually became bigger. The patient said that other than moderate itching in the beginning, she had no further problems. No vitiligo, thyroid issues, or other disorders run in the family.

On inspection, the depigmented patch was visible and restricted to the preauricular area of the left cheek as well as the front surface of the left breast, including the upper half of the breast. On the left side of the neck, there were also two discrete depigmented macules.

The patient's isolated depigmenting region is present. We speculated that the cell cover may have caused the white spots since the woman had a history of storing her phone in her left breast, under her shirt and was speaking into her left ear. Therefore, we began to consider contact depigmentation and sought to identify the known substances that may be responsible for causing it.

Since MBEH is the most popular depigmenting agent and a cornerstone of depigmentation treatment, we opted to use thin-layer chromatography (TLC) for the examination of mobile coverings.

RESULTS

The TLC from the cover extract did not reveal many compounds. On the TLC, the MBEH aired two segments [Figure 3]. We deduced that MBEH was present in the extract when we discovered that one of the spots from the mobile cover extract covered a comparable distance to that covered by the MBEH. We did not do a fresh TLC or examine the extract from the cover with pure MBEH. TLC revealed that the components of mobile covers and the control both covered the same distance or had a comparable relative front, which clearly suggests the existence of MBEH in the patient's mobile cover.

DISCUSSION

Depigmentation caused by chemicals may have an initial response resembling allergic contact dermatitis; however,

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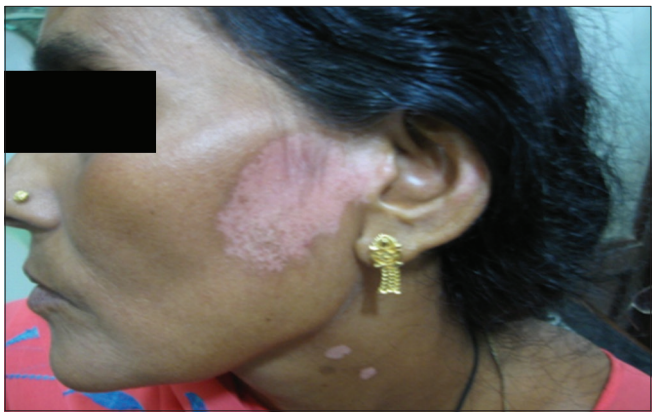

Figure 1: Depigmentation of left side of cheek.

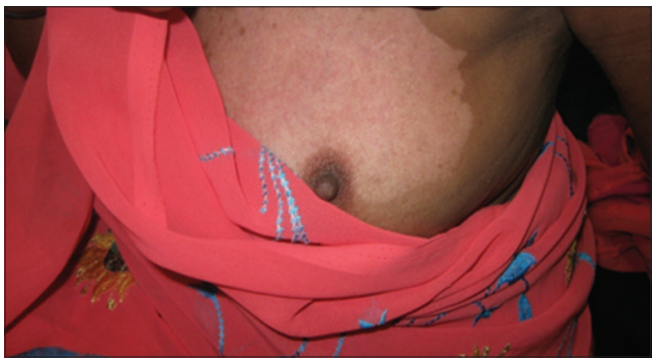

Figure 2: Depigmentation on the left breast.

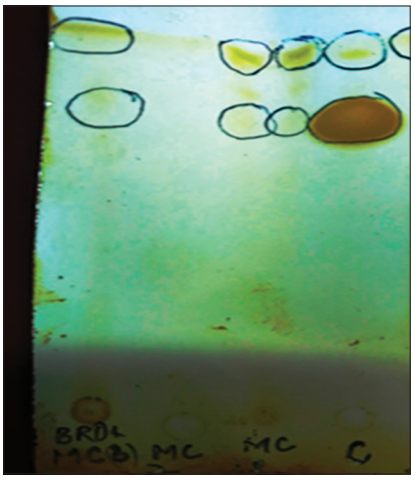

Figure 3: Thin Layer Chromatography (TLC) showing equal distance run by MBEH and Mobile cover component.

many instances happen without any obvious dermatitis1. The data are overwhelming in favor of a more complex pathophysiology that involves either localized direct toxicity to melanocytes or subclinical inflammation that can spread to distant, untouched melanocytes.

The phenol group, which is normally made up of a benzene ring and a hydroxyl side chain, is present in the chemical structure. The most effective depigmenting compounds seem to be phenols with an ether group at the para (or 4-) position and no polar side chains in those positions.[2]

These harmful phenols appear to function as tyrosine analogues on which tyrosinase and other melanogenic enzymes perform crucial alterations that prevent melanogenesis by sharing a chemical structure with the amino acid tyrosine. A few more substances, such as PPD, are not phenols but are "near enough" or are converted into phenols before becoming toxic.[2]

These depigmenting substances penetrate the melanogenesis process and produce toxic byproducts that kill melanocytes from inside. After exposure, the majority of people do not depigment. Chemically induced depigmentation probably involves a hereditary predisposition in the individual.[3]

Kroll *et al*. initially showed that a depigmenting phenol causes melanocyte death by triggering an inflammatory cascade in dendritic cells that trigger the cellular stress response and releases heat shock protein 70 (HSP70) (a proinflammatory heat shock protein).[4] As a result of this cellular stress response, heat shock proteins including HSP70, Unfolded Protein Response (UPR), and a protein called X- box binding protein (XBP1) are produced. These chaperone proteins are a critical mediator in the synthesis of the interleukin IL-6 and IL-8. According to both inflammatory cytokines, phenols that cause vitiligo stress cells and promote the release of pro-inflammatory signals, which, in turn, causes melanocyte death.[4] The enzyme tyrosinase is responsible for the depigmentation caused by MBEH. When MBEH interacts with tyrosinase, it appears to be transformed into a quinone product. This buildup of quinone derivatives specifically causes toxicity in melanocytes and results in melanocyte cell death.[5]

The indistinguishable vitiligo-like skin depigmentation that occurs after exposure to chemical phenols appears to be brought on by the activation of melanocyte-specific autoimmunity. Thus, from a clinical, histological, and pathological standpoint, chemically induced vitiligo and non-chemically produced vitiligo are on the same spectrum. Therefore, instead of being referred to as chemical leukoderma, occupational vitiligo or leukoderma, or contact vitiligo/leukoderma/depigmentation1, the term "chemical-induced vitiligo" should be used instead.

CONCLUSION

MBEH is one of the important substances producing depigmentation. This is the first case when a mobile phone cover is the suspected product giving a depigmentation. TLC can be an important and useful tool in identifying the culprit agent responsible for causing skin problems, in this case depigmentation.

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Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent.

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Conflicts of interest

There are no conflicts of interest.

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