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Case Report Open Access

Chemical Leukoderma to Hair Dye in a Singaporean Indian – A Rare, Adverse Cosmetic Outcome in Relation to a Ubiquitous Cosmetic Product

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Summary

A 54 year old Indian lady developed progressive hypopigmented lesions of her scalp after using an over-the-counter hair dye. On examination, multiple hypopigmented areas with irregular edges are observed over the scalp. Some of these hypopigmented areas are surrounded by a narrow rim of hyperpigmentation.

The diagnosis of Chemical Leukoderma to hair dye was made. Subsequent patch-testing results were all negative. Furthermore, there was no development of hypopigmentation over the patch-tested sites.

Chemical Leukoderma is a rare phenomenon in our local Singapore population. The simultaneous occurrence of both hypopigmentation and hyperpigmentation in our patient, which represents development of Chemical Leukoderma (melanocytic inhibitory reaction) and post-inflammatory hyperpigmentation (melanocytic stimulatory reaction) respectively, is presumptively even rarer. Plausible underlying pathophysiological mechanisms are explored.

Various treatment modalities have been described with varying success. However, definitive management requires prompt discontinuation of the offending product.

History

Our patient is a 54 year old Indian lady who presented with progressive, irregular hypopigmented areas over her scalp. She has been using the same hair dye product called Bigen hair dye for the past 4 years uneventfully until about 2 to 3 months prior to presentation. Bigen hair dye is a chemical (non-plant based) hair dye which lists P-Paraphenylenediamine and O-Paraphenylenediamine (PPD), as well as P-Aminophenol (a phenol compound), among its ingredients. Our patient has been religiously using the same coloured hair dye (black). To our knowledge, there were no changes in the compositions of the dye. There were neither new hair products being used, nor new oral medications consumed during this time.

Our patient reported initial flaking of the scalp but there was no erythema or blistering noted. This subsequently gave way to patchy hypopigmented areas over her scalp.

There was no involvement of other skin areas. Furthermore, there was neither any preceding personal medical history nor family history of vitiligo.

Physical Examination

On examination, multiple hypopigmented patches with irregular edges were noted over the scalp (Figure 1). Some of these hypopigmented areas especially those located at the frontal scalp region (Figures 1A and 1B) were surrounded by a narrow rim of hyperpigmentation. Leukotrichia was also noted over a sizeable area of hypopigmentation at the right frontal scalp region (Figure 1). Overall, the locations of these lesions closely conformed to the circumference of the scalp hairline.

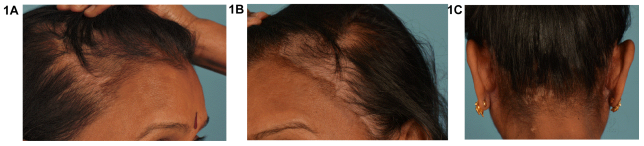

Figure 1: Multiple hypopigmented patches with irregular edges over the scalp.

Initial Diagnosis and Workup

A clinical diagnosis of Chemical Leukoderma to hair dye was made. Our patient was advised to discontinue using her current hair dye and to avoid using new hair dyes in the interim while a patch test was arranged for.

Patch testing was performed using the 1) Standard Battery; 2) para-Phenyenediamine (PPD) Mix; 3) Hairdressing Series; 4) Suspected offending hair dye. Subsequent patch testing results were all negative.

Our patient was reviewed again 1 month later. Crucially, there was no new development of hypopigmented areas over the skin sites where the patch testing was conducted. Excimer laser therapy is currently being arranged for.

Post-inflammatory hypopigmentation was also a possible differential diagnosis. However, there was a lack of overt symptoms of inflammation such as erythema, warmth or itch. Furthermore, the morphology of the depigmented areas on the scalp closely resembled confetti macules, which represents the classical description of chemical leukoderma. A biopsy would have been useful in differentiating between the two but our patient was not keen on it in this instance.

Discussion

Chemical Leukoderma is defined as an acquired hypopigmentation caused by repeated exposure to specific chemical compounds [1,2].

Since there is no single histopathological feature which can reliably distinguish Chemical Leukoderma from other differential diagnoses such as vitiligo, its diagnosis remains mainly a clinical one [1-4]. Although there are no formal diagnostic criteria at present, consistent supporting features have been identified -preceding history of repeated chemical exposure (history),presence of numerous acquired confetti or pea-sized hypopigmented macules (morphology), and close

correspondence of hypopigmented macules to areas of exposure (distribution) [1,2].

Patch testing can be employed to further support the diagnosis of Chemical Leukoderma [3,5]. On top of a positive skin reaction, subsequently development of leukoderma over the patch-tested sites containing the offending agent will be virtually diagnostic. While a positive patch test would have rendered the diagnosis of chemical leukoderma reassuring, a negative patch test does not exclude this possibility. In other instances, the development of chemical leukoderma occurs belatedly and therefore may not be picked up using conventional time frames employed by standard patch testing. Kwok et al. reported development of chemical leukoderma following patch testing with an acrylate series only 9 months later [6].

In our patient, the leukodermic area of the scalp closely mirrors the pattern of application of the offending hair dye. Confetti macules are noted over the upper posterior neck region (Figure 1C), but are not appreciated over the frontal scalp region (Figures 1A and 1B) presumably because they have coalesced to form confluent leukodermic areas [7].

What is further evident in our patient is that the affected leukodermic scalp is bordered by a thin rim of hyperpigmentation. This juxtaposition of both hyperpigmentation and hypopigmentation suggests that there was a preceding inflammatory dermatitis phase with resultant post-inflammatory hyperpigmentation, before the occurrence of the leukodermic phase. This inflammatory dermatitis phase is a common prequel to the development of Chemical Leukoderma, but is not an obligatory phase.

Localized leukotrichia over a leukodermic area represents another peculiar phenomenon in our patient. The underlying pathogenic mechanism of Chemical Leukoderma has been postulated to represent a direct cytotoxic effect to melanocytes, which manifests either by melanocyte destruction or inhibition of melanogenesis [8]. Therefore, localized leukotrichia may represent melanocyte or melanin disruption in the embedded hair follicles of a leukodermic area. One plausible mechanism which could explain the delayed onset of Chemical Leukoderma is that prior sensitization may sometimes occur. This is analogous to a delayed (Type IV) hypersensitivity reaction.

Chemical Leukoderma is a rare phenomenon whereas the implicated offending agents are widely used in many household items. Therefore, a minority of individuals must have inherent susceptibility to developing melanocytic injury, possibly conferred by their genetic composition [1,2,8].

Who are these individuals?

It does appear that majority of Chemical Leukoderma cases have been reported in Indian patients [1-3,5,7,8], as with our patient. Other than inherent genetic susceptibility, there are a number of plausible explanations. Firstly, there could be a higher pick-up rate simply due to a greater degree of contrast between the leukodermic area and the normal skin. Secondly, there could be a greater chance of exposure to potential offending chemicals due to any combination of religious, cultural or social reasons in this group of patients [7,8]. For example, the use of Bindi5 and Henna [9,10], which are important cultural and religious symbols to Indians, has been implicated in cases of Chemical Leukoderma. For affected Indian patients in India, other pertinent factors may come into play - a lack of regulation in manufacturing industries which results in common household items being produced with higher amounts of potential offending agents, such as p-phenylenediamine (PPD) in hair dyes; hypopigmentation being misconstrued as harbinger of an infectious disease such as leprosy, or even “impurity”, resulting in a higher rate of health-seeking behaviour [8].

The scalp has been reported to be the least commonly involved area of the body.1One suggested explanation is that the scalp is rich with densely packed hair follicles which represent rich reservoirs of melanocytes [8]. Ostensibly, it would take a strong and sustained melanocytic effect to render the scalp leukodermic.

However, the inherent conundrum is that the scalp is by far the most common region for application of hair dyes. With increasing premium placed on youthfulness, the use of hair dye is even more ubiquitous than before. It is not only been frequently implicated in allergic contact dermatitis of scalp, but also has been reported as the most common agent responsible for Chemical Leukoderma [1].

Various treatment options have been proposed for Chemical Leukoderma. Reported efficacious treatment modalities include, but are not limited to, topical steroids, narrow band ultraviolet B radiation (NBUVB) [11], pulsed steroid therapy [12] and excimer laser [13]. Regardless, by far the most important step is to identify and then to completely avoid the offending agent which drives the melanotoxic reaction.

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