A Case of an Elderly Women Presented with Severe Pigmented Contact Dermatitis (Riehl’s melanosis) Effectively Treated with Topical Lignin Peroxidase Cream

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ABSTRACT

Pigmented Contact Dermatitis is an acquired allergic contact dermatitis secondary to certain cosmetics or airborne allergens. Although it is rarely reported in fair skin types and in western countries, it is a significant disease with major psychological burden to patients with dark skinned types. Its exact relationship to Riehl melanosis remains controversial. While its exact aetiology and pathogenesis are still unknown, advances in treatment including oral tranexamic acid, glycyrrhizin compound and laser have been proposed. We report a case of an elderly female histologically confirmed to have Pigmented Contact Dermatitis secondary to hair dyes and hydrocarbon oils responded satisfactory to topical Lignin Peroxidase cream applied over six month duration.

Keywords Elderly Chinese women; Riehl's melanosis; Topical lignin peroxidase cream

INTRODUCTION

Pigmented Contact Dermatitis (PCD); originally described by G. Riehl in World War II, is a contact allergic dermatitis of the face complicated by severe hyperpigmentation [1-4]. It's exact aetiology and pathogenesis is unknown. Contact allergy is believed to be secondary to cosmetics, fragrances, textiles, hair dyes, coal tar and azo dyes, metallic jewellery and possibly others like airborne allergies resulted in an asymptomatic presentation of severe dyschromia [5-13]. The condition impacted a significant physical and psychological burden to the patient and their families. Till now, no well proven effective treatment is known [14]. We report a severe case of PCD in an elderly 70 years old woman presented with clinically and histologically Riehl's melanosis after using topical hair dyes and moisturizers containing hydrocarbon oils effectively managed by topical Lignin Peroxidase (LiP) cream.

CASE REPORT

A 70 year old Chinese lady visited our dermatology clinic on November 9, 2017 presented with a severe hyperpigmentation over the face. She also noticed increased pigmentation over the neck, post-auricular regions, chest and axillae. She denied any pruritus and discomfort. There is no significant past medical and allergy history. She reported to have frequent applications of hair dyes but without any side effects. She did not take any systemic and topical medications including herbal medicines. On examination, she was found to have severe generalized grey-black hyperpigmentation involving the face, neck, post-auricular region and part of the upper chest. Skin reticulated hyperpigmentation over the forehead was noted. Her original white hair was tanned to a light grey colour. On further enquiry, she reported to have repeatedly used an over the counter preparation of natural hair dye and conditioner consisted of the following ingredients: Indigofera tinctoria Leaf Powder (Indigo), Cassia auriculata Leaf Powder (Senna), Lawsonia inermis Leaf Powder (Henna). She denied any irritation after each application. In addition, she had also been applied a moisturizer over the face, neck and body which contained Camellia seed oil (tea polyphenols).

At this juncture, the differential diagnosis are exogenous ochronosis, post-inflammatory hyperpigmentation secondary to inflammatory dermatosis, maturational dyschromia, melasma, acquired nevus of Ota, Addison’s diseases and PCD or Riehl melanosis. The clinical history suggested the latter. A diagnostic skin biopsy was performed after careful explanation and counselling to the patient with her consent. The histology reported a piece of skin with scattered isolated and small clusters of melanophages and small lymphocytes in the papillary dermis. The overlying epidermis appears unremarkable. Scantly pigmented melanocytes are seen at the dermo-epidermal junction, no melanocytic proliferation is seen. There is mild superficial perivascular lymphocytic infiltrate. Solar elastosis is seen. The features are compatible with post inflammatory hyperpigmentation due to previous contact dermatitis to topical application (Figure 1).
Figure 1: Histological slides from post-inflammatory hyperpigmentation featuring melanophages and small lymphocytes in superficial perivascular location in the papillary dermis. Also the epidermis show mild atrophy with mild vacuolar interface dermatitis compatible with pigmented contact dermatitis.

The histology and clinical history ruled out ochronosis, melasma, post-inflamatory hyperpigmentation secondary to inflammatory dermatosis but pigmented contact dermatitis or allergic melanosis. Subsequent, investigation included complete blood picture, liver function test, thyroid function test, renal function test, occult systemic and gynaecological malignancy screens, serum cortisol and ACTH level were performed and reported negative. A standard patch testing was performed but failed to provide further clues of the culprits of the contact dyschromia. A provisional diagnosis of PCD or Riehl melanosis was made.

In view of the severe disfigurement associated with the condition, management was started alongside with the investigations. The patient was carefully counselled, explained and reassured about the nature and course of the hyperpigmentation. Salient points were emphasized particularly on total abstinence of cosmetic products, inappropriate sunscreens, moisturizers, hair dyes and fragrances on the skin. The patient was instructed on the importance of sun protection with hats, umbrella, appropriate hypoallergenic sunscreen of a sun protective factor of 50++ and excessive sun avoidance. Hypoallergenic moisturizers were recommended. For the treatment, as no well proven effective medications are documented in the literature and tyrosinase inhibitors and laser therapy are relative contra-indicated and risky, topical cystamine cream was tried but refused by the patient due to stinging sensation and odour. The patient refused to take oral medication including tranexamic acid. Topical LiP cream was prescribed, accepted and well tolerated by the patient applied twice daily over the affected areas of the skin. A gradual improvement of the hyperpigmentation, skin texture and roughness of the patients were observed over a period of six months treatment duration (Figure 2).

PCD is considered by some authorities a form of allergic contact dermatitis with resultant severe hyperpigmentation [5,6,14]. The allergic contact dermatitis is believed to be due to a variety of topical and airborne allergens or a lichenoid immune reaction that may be caused by intrinsic or extrinsic factors. Strictly speaking, the original cases described by Riehl seventy years ago may represent a separate disease entity; at least the nutritional factors seem to play a predominant role in the classical reported cases of Riehl melanosis.

Nonetheless, PCD especially in the recently reported cases in the literature has a cause and effect relationship secondary to allergic sensitization to applied cosmetics, fragrances, textiles, hair dyes, metallic Jewelleries allergens and hydrocarbon oils. Nakayama has suggested that the PCD is possibly a type IV allergy due to repeat small...
amount of contact sensitization to the skin immune system with the results of pigmentary incontinence and engulfing of melanin by melanocytes [5,6,8,14]. Our reported case also illustrated this clinical history. In addition, our patient gender, dark skin type ( Fitzpatrick III) and late middle age are in consistent with the described epidemiological data of the documented cases of PCD [14,15]. Further confirmation of the diagnosis can be aided by dermoscopy, confocal microscopy and sophisticated extended patch and photo patch testing series [15,16].

Currently, no double blinded control trial has been performed for evidence-based therapy of PCD [14]. Avoidance of the cosmetics, use of hypoallergenic skin care products, sun-protection and use of topical tyrosinase inhibitor, azelaic acid, retinoids, lasers have been attempted but may not be effective and acceptable to the patients due to its irritancy and unpredictable results [17,18]. A small study of 10 patients suffered from PCD were treated with oral tranexamic acid combined with glycyrrhizin acid topical preparations for three months followed by further three months of tranexamic acid orally alone were reported to be efficacious [19]. Although the number of recruited subjects were small, but the action of tranexamic acid in inhibiting the transfer of melanin is a plausible mechanism. Another small pilot study used a triple combination therapy with a low-fluence 1064 nm Q-switched Nd: YAG laser, hydroquinone cream and oral tranexamic acid for recalcitrant Riehl's melanosis was reported to complete clear the melanosis in the majority of patients; albeit tyrosinase inhibitor may not be available in some countries [20]. In our current report, topical LiP cream is shown to be an efficacious, well tolerated treatment option with a high patient's acceptability. Topical LiP may provide an alternative treatment modality to this severely disfiguring, still pathogenetically unresolved pigmentary disorders in the female who frequently applied cosmetics. We previously also reported another case of a 40 years old male presented with unilateral asymptomatic Riehl melanosis after applying expired topical sunscreen treated satisfactorily managed by topical LiP cream [21].

LiP was discovered in the extracellular medium of the white-rot fungus, Phanerochaete chrysosporium [22,23]. Since then, at least six other isoforms of the ligninolytic enzymes were characterized from various fungal organisms, namely: Tramates versicolor, Phlebia radiata and Phanerochaete sordida but not bacterium. Each has a different isoelectric point, sugar content, substrate specificity and stability [23,24]. Melanin; expressed as eumelanin and phaeomelanin; produced by the process of melanogenesis are physiologically stored in melanosomes and transferred to the keratinocytes in the epidermis which serves its role of protection against ultraviolet radiation and environmental stress [23]. However, aberrant or uncurbed transfer of melanin to the dermis and epidermis may result in the frequently seen hyperpigmentation and dyschromia due to various pathophysiological mechanisms. Lignin and coal in the polymers form are made up of indole and phenolic subunits have a close structural resemblance to melanin [23]. Hence, naturally occurred ligninolytic enzymes like LiP is a good organic candidate endowed by nature to oxidize and denature abnormal melanin in the skin which biologically resemble lignin. The biochemical pathway and mechanism of LiP is illustrated in Figure 3. Clinically, crude LiP was demonstrated to decolorize synthetic melanin [22-25]. This is further illustrated in another study that LiP act as a melanolytic enzyme capable of degrading human skin melanin [23]. The application of the LiP cream provided a significant faster skin lightening effect than the tyrosinase inhibitor, 2% hydroquinone cream [26]. Draelos demonstrated LiP has a skin lightening effect comparable to hydroquinone with no reported adverse side effects but with superiority in skin texture and roughness [27]. Our case report also demonstrates the overall improvement in discoloration, texture and skin roughness as commonly seen in solar elastotic skin patient come from an intensely pigmented ethnicity. However, as the cost of LiP cream is not inexpensive and the duration of treatment maybe long and extensive; this may impede the patient's compliance to the management. Patient should be fully explained, counselled on the different alternative regimes for the treatment of PCD and followed up regularly and continuously with a team care approach.

![Figure 3: Mechanism of action of lignin peroxidase as cosmetic lightening agent.](image_url)