Dermatology Practical & Conceptual

Bilateral Eyelash Poliosis with Raccoon Erythema and Hypopigmentation-Contact Follicular Leukoderma

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Introduction

Contact chemical leukoderma is an acquired hypopigmentation-to-depigmentation disorder due to repeated exposure to chemical agents which are toxic to melanocytes in genetically susceptible individuals [1].

Case Presentation

A 36-year-old male patient presented with whitish discoloration of eyelashes and periorbital pruritus for 10 months. The patient had a history of ankylosing spondylitis with uveitis and had used topical medications, including olopatadine and tobramycin for two months, followed by azelastine and cyclosporine for three months, along with topical steroids for eight months. Most of these topical agents contained preservatives such as benzalkonium chloride and cetrimide. The patient had no history of auditory or neurological symptoms. Examination revealed eyelash poliosis sparing eyebrows. Bilateral periocular areas showed diffuse hypopigmented macule with perilesional erythema, telangiectasia, and fine scaling (Figure 1). A possible diagnosis of follicular vitiligo, and Vogt-Koyanagi-Harada (VKH) syndrome was considered for uveitis and poliosis. Fundoscopy did not reveal any optic disc pallor, retinal depigmentation, or scarring. As late-onset poliosis and fundoscopy did not show features of sunset glow, the possibility of VKH syndrome was discarded (Figure 2A and 2B). Later we performed a patch test with the suspected eyedrops and preservative cetrimide, which was negative on readings on days 2, 4, and 7. Periocular features

of erythema, scaling, and pruritus with temporal association with topical therapy led us to speculate contact leukoderma or steroid-induced hypopigmentation despite a negative patch test.

Diagnosing contact leukoderma requires three of four criteria: acquired vitiligo-like depigmented macules, patterned lesions at exposure sites, confetti lesions, or prior chemical exposure [1,2]. Inflammation (itching, irritation) before leukoderma onset supported this diagnosis over vitiligo. Contact leukoderma has been described mostly secondary to phenol, benzol, and catechol derivatives (monobenzylether of hydroquinone in tannery workers, rubber, plastic, leather, and dye industries; benzyl alcohol hair dye and rinses, and azo dyes) which have selective melanocytotoxicity [3,4]. In most cases, there is no linear or temporal correlation between exposure and leukoderma, while there is dose-dependent melanocyte destruction [4]. There are reports of contact leukoderma or hypopigmentation secondary to corticosteroids at the site of application and the local spread of chemical leukoderma along the lymphatics beyond the site of contact [5]. We could not clarify whether the chemical leukoderma was due to corticosteroid or any other component of eyedrops used for uveitis which led to melanocyte apoptosis or inflammation.

VKH syndrome is a rare multisystem disease of unknown etiology and is characterized by bilateral panuveitis, white forelock, and auditory and neurological manifestation [6].

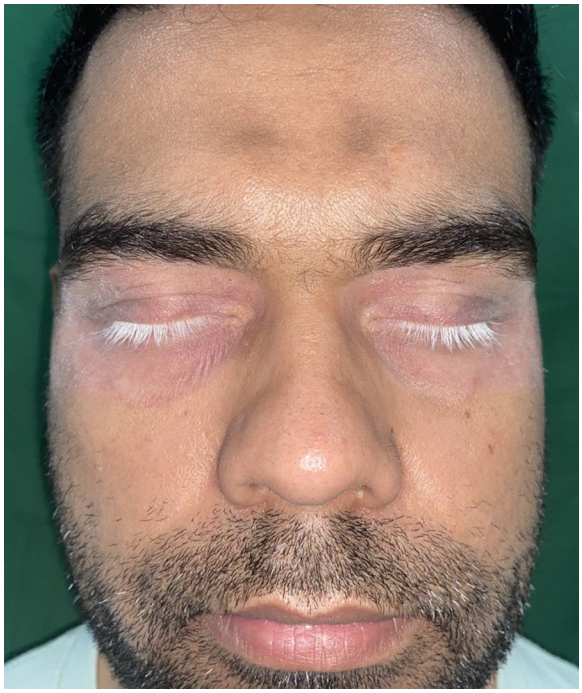

Figure 1. Bilateral eyelash poliosis with periocular erythematous hypopigmented fine scaly macule.

Conclusion

Follicular vitiligo is characterized by leukotrichia with or without interfollicular minimal hypo-to-depigmentation. In non-segmental vitiligo the interfollicular skin is affected with intact hair pigment in the early stages, while the reverse is seen in follicular vitiligo. Differentiating vitiligo from contact leukoderma can be challenging, as initial contact leukoderma may trigger an autoimmune response leading to vitiligo, and pre-existing vitiligo could be mistaken for chemical-induced depigmentation. We hereby report a rare case of contact follicular leukoderma secondary to topical eyedrops in a patient with uveitis mimicking VKH syndrome. Further research is required to know the exact pathomechanism involved and the etiological agent in eye drops which led to unusual presentations.

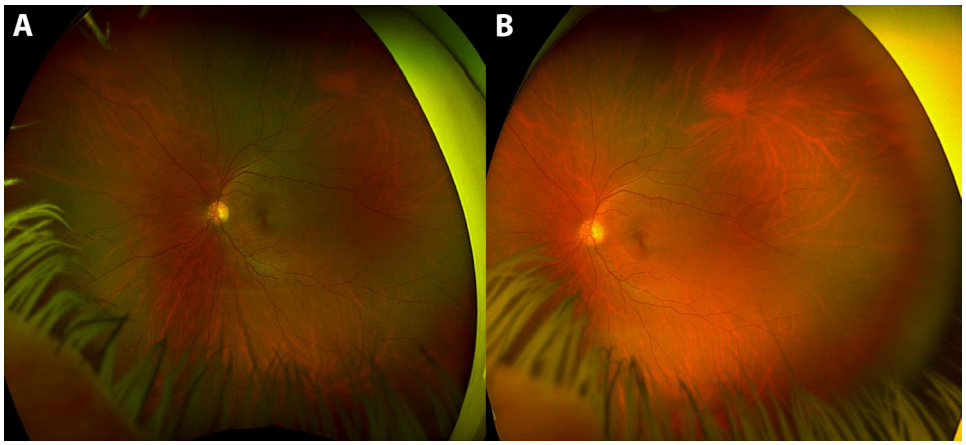

Figure 2. Fundoscopy demonstrates both eye cup disc ratios 0.4:1, healthy neuroretinal rim, sharp foveolar reflex. and arteriovenous ratio 2:3 suggestive of normal fundus (A and B).

References

1. Takeuchi A, Egawa G, Nomura T, Kabashima K. Contact leukoderma induced by rotigotine transdermal patch (Neupro®). *Eur J Dermatol*. 2019;29(2):215-217. DOI:10.1684/ejd.2019.3500
2. Oliver EA, Schwartz L, Warren LH. Occupational leukoderma: preliminary report. *JAMA*. 1939;113(11):927-928. DOI:10.1001/jama.1965.03090220021006.
3. Pandhi RK, Kumar AS. Contact leukoderma due to 'Bindi' and footwear. *Dermatologica*. 1985;170(5):260-262. DOI:10.1159/000249545
4. Neal D, Arnold J, Moss T. Serpiginous hypopigmentation secondary to intra-articular corticosteroid injection. *Dermatol Online J*. 2017;23(4):13030/qt3zr7f5n2.
5. Greco A, Fusconi M, Gallo A, et al. Vogt-Koyanagi-Harada syndrome. *Autoimmun Rev*. 2013;12(11):1033-1038. DOI:10.1016/j.autrev.2013.01.004
6. Mehta N, Abdallah M, Gupta S. Unraveling the paradox of reverse chronology in follicular vitiligo. *Dermatol Rev.* 2022;3(5):284-288. DOI: 10.1002/der2.161