CORRESPONDENCE

A case of facial hyperkeratosis induced by a cosmeceutical containing alpha-hydroxy acid and sunlight overexposure successfully treated using oral acitretin

 Chemical peels cause destruction of the epidermis and subsequent repair. Postpeel care is important.

 An 18-year-old man presented with progressive erythematous keratotic papules with associated greasy debris bilaterally on the cheeks, postauricular, and submandibular areas, and to a lesser extent the bridge of the nose and forehead, that had been present for the past 4 months (Figure 1). He was otherwise in good health and denied any similar symptoms or hereditary keratotic dermatosis among his relatives.

 The patient washed his face twice daily with a mild cleanser and gently removed the hyperkeratotic debris using a gauze scrub; however, it would recur within a few days. Removing the debris revealed skin without bleeding or erosions but with mild itching. The patient had applied an acne cosmeceutical containing 15% alpha-hydroxy acid (AHA) to his face, especially his cheeks, once daily before bedtime for 1–2 months before the gradual onset of facial keratosis. The patient had often played basketball outdoors at midday without applying sunscreen. Treatment with oral doxycycline, topical fusidic acid, adapalene, and urea had not improved his condition.

 Laboratory findings were within reference ranges. Bacterial cultures yielded negative results. Pathology examination revealed diffuse thick and loosely laminated and ring form orthokeratotic hyperkeratosis of the stratum corneum and opening of hair canals respectively, focal follicular occlusion, a granular cell layer of reduced thickness, and nonsignificant inflammatory infiltration (Figure 2). Based on the clinicopathologic findings, we made a diagnosis of facial hyperkeratosis as a side effect of improper use of a cosmeceutical containing AHA and sunlight overexposure.

 We had considered several differential diagnoses including dermatitis neglecta and Darier disease. Poskitt et al1 first described

Figure 1 A healthy 18-year-old man with progressive erythematous keratotic papules and associated greasy debris on the cheeks, postauricular and submandibular areas, and, to a lesser extent, the bridge of the nose and forehead over a period of 4 months.
Substantial cellular infiltration can markedly improve the condition. The symptoms should not recur if good hygiene practice is maintained. Histopathologic features include orthokeratotic hyperkeratosis without substantial cellular infiltration.

Dariar disease is a genetic disease of keratinization and presents greasy keratotic papules in seborrheic areas including the face. Histopathologic examination shows suprabasal acantholysis and epidermal dyskeratotic cells.

In our case, we made a diagnosis of facial hyperkeratosis induced by improper use of a cosmeceutical containing AHA and sunlight overexposure could have enabled ultraviolet radiation to stimulate the exposed mitotically active basal epidermis, resulting in rebound hyperkeratosis as a natural protective response to solar radiation.

In this study, we report the first case of facial hyperkeratosis induced by improper use of a cosmeceutical containing AHA and sunlight overexposure that responded to oral acitretin. Potential risk factors include a higher concentration of free acid, larger volume applied to the skin, facial area, longer duration of contact, higher ultraviolet dose received and inherent genetic keratotic diathesis. It is recommended that AHA concentrations below 10% should be used with strict sun protection measures in susceptible individuals.

References


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