Hydroquinone-Induced Hyperpigmentation: A Case of Exogenous Ochronosis

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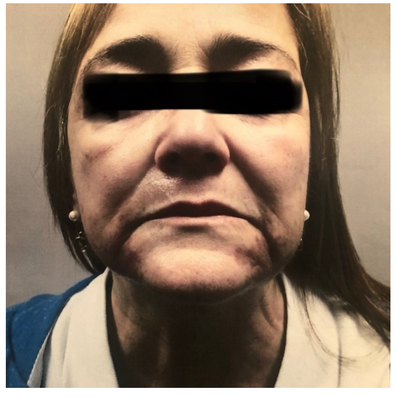
Introduction

- Achieving flawless skin as part of the desire to be perceived as “beautiful” is a common sentiment shared by many cross-culturally.[1]
- The most common agent to attain this effect is hydroquinone (HQ), a topical bleaching agent used to treat hyperpigmentation. HQ concentrations vary from 2% (OTC) to 4%-15% (Rx).
- Exogenous Ochronosis (EO), a rare but serious complication of long-term, high concentration HQ use, is a localized and paradoxical cutaneous disorder characterized by diffuse, symmetrical, asymptomatic hyperpigmentation over sun-exposed skin first described in 1975 in a group of South African patients.[2,3]
- We present the case of a 61 year old female of Venezuelan decent, with olive skin tone, Fitzpatrick skin type IV, diagnosed with EO. Included in her 10+ year skin care regimen was HQ 4% which a plastic surgeon suggested to help achieve a more even complexion.

Case Description

A.



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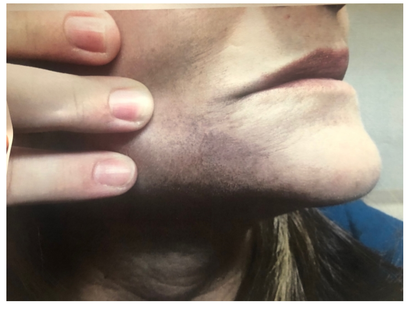

C.

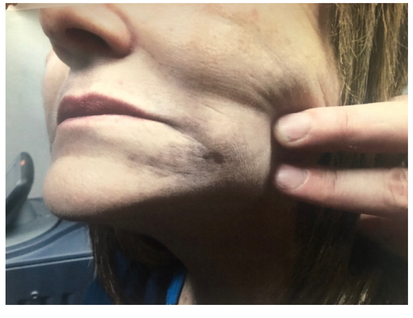

Figure 1. (A,B,C) July 2018: First consultation with physician.

- **2005:** Several small sun marks that she attributed to high amounts of exposure in her youth. When exposed, always tans, never burns.
  → Plastic surgeon gave a product line which included retinol, HQ 4%, benzo-peroxide, among other ingredients.
- **2006:** Resolution of marks.
  → To prevent any re-occurring marks, continues once daily for the next 11 years without sunscreen on applied areas.
- **2006-2016:** Patient states “brighter” complexion, appeared more youthful, and felt more confident about her appearance.
- **2017:** Noticing larger hyperpigmented patches that looked different than in first 2005 occurrence.
  → Consulted GP, diagnosed with mild melasma, told to continue using HQ but to increase frequency to twice daily.
- **2018:** Chin biopsy confirms EO.

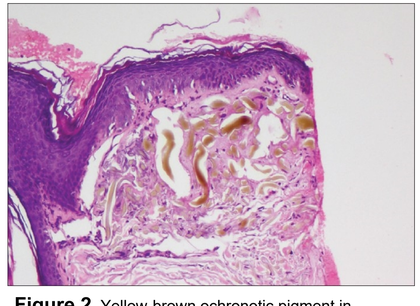

Figure 2. Yellow-brown ochronotic pigment in collagen bundles in the dermis (H&E, ×100). Picture: Jain et. al (2013).

Discussion

- EO is more common in the darker Fitzpatrick skin types IV, V, VI. However, more cases involving fair-skinned people, and with HQ 2% use for shorter time periods are being reported.[6]
- Once thought to be a rarity in the United States, dermatologists are finding that EO more frequently presents on a spectrum than with the extremes described in many dermatological texts and can easily be misdiagnosed—resulting in more HQ use.[2]
- HQ inhibits enzymatic conversions of tyrosine to DOPA (dihydroxyphenylalanine) which decreases the number of melanocytes and melanin transfer leading to lighter skin.[4] HQ requires sunscreen protection and must be monitored for frequency and duration.
- Exact mechanism of EO is unclear. EO is histologically defined by yellow-brown, curvilinear, “banana-shaped” ochre dermal deposits (Fig. 2). Severe form on physical exam will present as blue-black skin.[2,3]
- Treatment for EO is difficult. Chemical peels with glycolic acid, dermabrasion, and the Q-switch Nd Yag 1064 laser have been shown to improve EO-induced hyperpigmentation. Caution: Tx options can inadvertently cause irritation that results in furthering the unwanted hyperpigmentation!![5,6,7]

Conclusion

- HQ’s paradoxical side effect of EO is an important adverse reaction and is the result of an unintended but vicious cycle that should not be neglected by clinicians and consumers.
- With a billion dollar cosmetic industry capitalizing on our beauty-obsessed culture, it is imperative that adequate patient education on HQ-containing products, prescription and over-the-counter, be addressed both clinically early on with a board-certified dermatologist and with more awareness as a society.

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