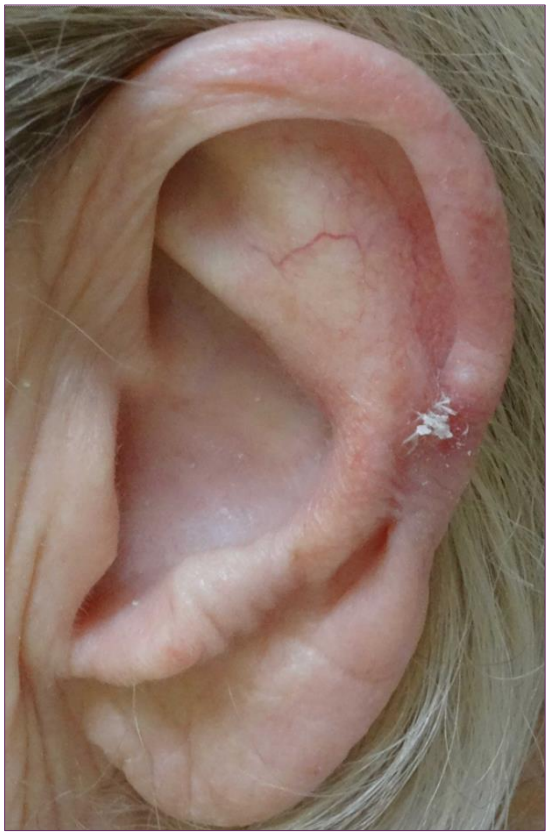
SKIN

SKIMages

Auricular Distortion Following Cryotherapy

Jason T. Bard, BS[1], Heather A. Kornmehl, MD[1], Joseph M. Wentzell, MD, FAAD, FACMS[1]

[1] Eastern Virginia Medical School, Norfolk, VA



Cryotherapy is widely utilized in dermatologic practice for the treatment of more than 50 conditions.[1] Potential complications of cryotherapy include scarring, dyspigmentation, infection, and hair loss. In terms of efficacy, one of the largest prospective studies on cryotherapy for the treatment of actinic keratosis found an overall complete response rate of 67.2%. About half of patients reported burning or stinging pain during treatment; no serious reactions were reported.[2] The ears, particularly in shorter haired individuals, are a common site of actinic damage and are therefore commonly treated with cryotherapy techniques.[3,4]

We report a case of external ear architecture distortion following cryotherapy. A 52-year-old otherwise healthy female with no underlying disease presented for treatment of a recurrent actinic keratosis of the helical rim six months after a session of cryotherapy (two freeze-thaw cycles) by an outside practitioner. By report, her ear was anatomically normal prior to treatment. Physical examination demonstrated the recurrent actinic keratosis and fusion of her helix and antihelix with obliteration of the scaphoid fossa (Figure 1). Unfortunately, this is not an isolated case. We have seen this complication before through referrals from outside sources, however, to our knowledge, there are no published cases in the literature of helix-antihelix fusion as a complication of cryotherapy.

Scarring is a known potential complication of cryotherapy.[1] However, it is not clear that collagen scarring is the mechanism of this helix-antihelix fusion. Rather, we postulate the mechanism is adhesions secondary to re-epithelialization of the suprabasilar stratum malpighii layers. This process bridges the contours of the epidermis without necessarily invoking a process of scarring. This adhesion between helix and antihelix can occur with or without epidermal damage to the intervening scaphoid fossa. This is analogous to other better-known epidermal injury adhesions such as labial fusion, nasal synechiae, finger fusion, or adhesion of skin folds due to epidermal damage in pemphigus. In fact, the cumulative effect of strong keratinocyte cell-cell adhesion during the healing process is the likely mechanism for the fusion process, without stimulating collagen damage and repair.[5] This epidermal adhesion process can create contour distortion in the absence of dermal damage. Correction of fusion between the helix and antihelix of the ear requires surgical separation of the helix and antihelix.

As such, practitioners should account for the risk of epidermal fusion of the external ear's architecture prior to performing cryotherapy on both the antihelix and helix. A potential solution is to refrain from treating both areas simultaneously, allowing each anatomic site to heal before the other is treated, while avoiding damage to the intervening scaphoid fossa. Although, we recognize multiple appointments may result in increased healthcare costs. To prevent unintended overspray between the helix and the antihelix, a structural shield can be applied to protect adjacent skin. Lastly, it may be helpful to limit the extent of the freezing process. This reasoning can be applied to other destructive treatments resulting in re-epithelialization of the epidermis, such as 5-fluorouracil application.

Conflict of Interest Disclosures: None

Funding: None

Corresponding Author:
Jason Bard
721 Fairfax Avenue, Suite 200
Norfolk, VA 23507
Email: BardJT@EVMS.edu

References:

1. Afsar FS, Erkan CD, Karaca S. Clinical practice trends in cryosurgery: a retrospective study of cutaneous lesions. *Postepy Dermatol Alergol*. 2015;32(2):88-93. doi: 10.5114/pdia.2015.48048.

2. Thai KE, Fergin P, Freeman M, et al. A prospective study of the use of cryosurgery for the treatment of actinic keratoses. *Int J Dermatol*. 2004; 43(9):687-92

3. Ibrahim SF, Brown MD. Actinic keratoses: a comprehensive update. *J Clin Aesthet Dermatol*. 2009;2(7):43-48.

4. Ferrándiz C, Plazas MJ, Sabaté M, Palomino R. Prevalence of actinic keratosis among

dermatology outpatients in Spain. *Actas Dermosifiliogr*. 2016;107(8):674-80. doi: 10.1016/j.ad.2016.05.016.

5. Evans ND, Oreffo RO, Healy E, Thurner PJ, Man YH. Epithelial mechanobiology, skin wound healing, and the stem cell niche. *J Mech Behav Biomed Mater*. 2013;28:397-409. doi: 10.1016/j.jmbbm.2013.04.023.