Rapid Development of Keratoacanthomas After a Body Peel

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Resurfacing techniques have been traditionally limited to the face because of a lack of predictability and standardization for peeling nonfacial skin. There is a need for medical and surgical intervention for treating nonfacial skin that is actinically damaged. Medium-depth chemical peels (Jessner +35% trichloroacetic acid) remove the photodamaged epidermis to stimulate the production of new collagen in the dermis and remove lesions associated with facial actinic damage, including lentigines and actinic keratoses. Widespread actinic damage is common on the arms and chest. A 70% glycolic acid gel plus 40% trichloroacetic acid peel (Cook Body Peel) is a controlled peel that predictably enables peeling of nonfacial skin in a uniform and safe fashion with specific clinical endpoints. An unusual complication of this body peel is reported.

S. COX, MD HAS INDICATED NO SIGNIFICANT INTEREST WITH COMMERCIAL SUPPORTERS.

A 72-YEAR-OLD healthy female with extensive actinic damage on the arms and hands was referred to our office for treatment of widespread actinic keratoses. The patient had a history of previous squamous cell carcinoma on the right arm. She had undergone previous liquid nitrogen and topical 5-flourouracil treatments for the actinic keratoses. She was unable to tolerate the irritation from the topical 5-flourouracil. She had been treated with tretinoin and 12% lactic acid lotion twice a day for several months. The initial evaluation revealed dozens of erythematous, scaly plaques located on the hands and arms. There was diffuse hyperpigmentation and areas of hypopigmented scars. The patient wanted to treat the residual precancerous lesions. We opted to pursue a controlled body peel using the “Cook Total Body Peel.” The skin of the arms and hands was degreased with acetone. A 70% glycolic acid gel was applied with 4 x 4 gauze. This was then followed by the application of 40% trichloroacetic acid applied with large cotton-tipped applicators. The areas were watched for the initiation of fine white speckling on mildly erythematous skin. Once this clinical endpoint was achieved, copious neutralization with 10% sodium bicarbonate was performed. After stinging subsided, aquaphor ointment was applied liberally to the areas.

Two weeks after the peel, the patient presented with several 4- to 6-mm indurated red plaques located only on the left forearm (Figures 1a–c). A biopsy of the largest nodular site demonstrated marked acanthosis of the epidermis with a central area of an inverted growth pattern. There was hyperkeratosis and parakeratosis. Focal squamous atypia was present surrounded by fibroplasia (Figure 2). The histologic pattern correlated with a keratoacanthoma, possibly involuting. A second biopsy of another plaque revealed a similar diagnosis. One month after the peel, several of the smaller plaques were resolving without treatment. The patient followed up with her general dermatologist who placed her on diclofenac sodium 3% wt/wt topical gel with the eventual resolution of any remaining lesions. At 6 months after the peel, no premalignancies or malignancies were evident.

Comment

This unique case demonstrates multiple rapidly developing keratoacanthomas in response to a chemical peel. The etiology of keratoacanthomas remains unclear. Multiple keratoacanthomas have been reported after a severe sunburn, repetitive sun exposure, psoralen, and ultraviolet phototherapy after a thermal burn and after radiation therapy. The development of keratoacanthomas after a chemical burn or by any of the above mentioned insults suggests aberrant keratinocyte behavior and a defective repair response. Impaired cell-mediated immunity has been suggested in patients with multiple keratoacanthomas. The role of ultraviolet light and the development of carcinogenesis and cocarcinogenesis through DNA damage and local injury to the Langerhans cells are well known. Therefore, the development of keratoacanthomas after sunburn or radiation is plausible. The

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development of keratoacanthomas after a chemical peel can be hypothesized as an abnormal tumor immunologic response to chemical injury to the skin in the same way a thermal burn can induce a carcinogenic response. An example of this is the Kangri cancer occurring in Kashmir among natives who use the Kangri jar carrying hot coals for warmth next to their skin with the development of squamous cell carcinoma. Another possibility is that the abnormal cells were present before the chemical peel. There is a recent case report of the rapid development of nonmelanoma skin cancer after CO₂ laser resurfacing. It was suggested that the cancer was not clinically apparent at the time of resurfacing but developed from abnormal follicular cells. If this is the case, then CO₂ resurfacing is not destructive enough to the abnormal cells that exist in the deep follicular epithelium.

Chemical peeling has been used to treat widespread actinic keratoses and damage. The level of injury when peeling the body needs to be more superficial than when peeling facial skin due to the increased risk of scarring on nonfacial skin. It may be unrealistic to think that body peels can reduce premalignancies; however, we do see both clinical and histologic improvement of the skin with the production of a papillary dermal repair zone with new collagen remodeling. Most likely early actinic keratoses can be improved; however, the abnormal cells deep within the follicular epithelium are little affected. It is unknown whether these widespread keratoacanthomas arose as a result of the chemical peel or were present subclinically before peeling. The fact that they occurred within 2 weeks of the peel and resolved without treatment suggests a direct relationship to the peel. It also suggests that keratoacanthomas can be a self-limited process that may involute spontaneously without invasive treatments. However, it is impossible to predict how long it will take. More importantly, grade 1 squamous cell carcinoma cannot always be
excluded even with a biopsy; therefore, with larger discrete keratoacanthomas, surgical treatment is the safest course of action. When widespread, close observation for spontaneous involution may suffice.

References