New-Onset Lichenoid Dermatitis Following Excision of Squamous Cell Carcinoma: Coincidence or Association?

Dear Editor,

Squamous cell carcinoma (SCC) is the second most prevalent skin cancer worldwide. SCC's most substantial risk factors include ultraviolet exposure, fair skin, and immunosuppression. Treatment of SCC is principally surgical, but adjuvant chemoradiotherapy is also used.[1] Lichenoid dermatitis, the most common of which is lichen planus, is among the prevalent clinical and histological examples.[2] To the best of our knowledge, this is the first presentation of lichenoid dermatitis developing after the excision of SCC.

A 67-year-old man with a history of well-differentiated SCC (first diagnosed 2 years earlier) several times excised from the nose and ear but without lymph node, local, or distant metastasis. The patient presented with hyperkeratotic lesions at the last excision sites that developed during the previous 6 months. Dermatologic examination showed erythematous brown hyperkeratotic plaques on the tip and dorsum of the nose, preauricular area, and external ear canal [Figure 1A]. In addition, there were hypertrophic and atrophic scars at the SCC excision sites [Figure 1A]. Ulceration and telangiectasia were not observed. Multiple biopsies obtained from all the lesions were reported as lichenoid dermatitis, with orthokeratosis, hypergranulosis, and irregular acanthosis of the epidermis, but no tumor cells [Figure 1B]. The lesion's spontaneous regression was noted within 2 weeks of the biopsies [Figure 1C]. After 7 months of follow-up, the patient showed no signs of relapse or any likely lesions elsewhere.

The pathophysiology of lichenoid dermatitis is still obscure. On the basis of the relationship between lichenoid dermatitis and paraneoplastic pemphigus, various hypotheses have been proposed about the relationship between lichenoid dermatitis and malignant neoplasms. Lichenoid eruptions may be seen before or after signs or symptoms of the underlying malignancy. A chronic lichenoid reaction pattern may predispose some patients with cancer to develop humoral autoimmunity against basement membrane components. Moreover, cancer might indicate a cell-mediated immune response and give rise to lichenoid dermatitis. Accordingly, autoreactive T cells would respond opposite the ordinarily inactive basement membrane constituents.[3]

In the literature, a case of biopsy-proven lichenoid dermatitis was reported in a patient following basal cell carcinoma (BCC) excision, which was suggested to develop due to the immune response to BCC removal in the excision area. However, considering the relationship between lichenoid dermatitis and BCC detection, the triggering stimulus (BCC) may no longer be detected in a biopsy specimen. Instead, it may be isolated, coexisting with BCC, or be mistaken for BCC, and it may even mask occult BCC.[4] In another case, it was reported that lichenoid dermatitis developed while receiving vismodegib therapy for BCC. It has been claimed that molecular and immune mechanisms cause the formation of lichenoid dermatitis.[5] However, the relationship between lichenoid dermatitis and SCC is unknown.

In our patient, it was observed that lichenoid dermatitis developed in the scar areas. Although it is not clear why lichenoid dermatitis develops after scarring, it is thought that the Koebner phenomenon, which expresses the development of new lesions in post-traumatic areas, is one of the possible provoking factors.[6] In the literature, giant cell lichenoid dermatitis has been reported in herpes zoster scars in a bone marrow recipient. It has been suggested that the varicella-zoster virus may have initiated the hypersensitivity reaction in this patient. It has also been suggested that the patient's immune status may have affected the morphology.[7]

As a result, lichenoid dermatitis might be a paraneoplastic manifestation and can occur before, during, or after malignancy; therefore, additional research is necessary to discern the association between lichenoid dermatitis and SCC; more clearly, patients should be closely monitored.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Nil.

Conflicts of interest

There are no conflicts of interest.

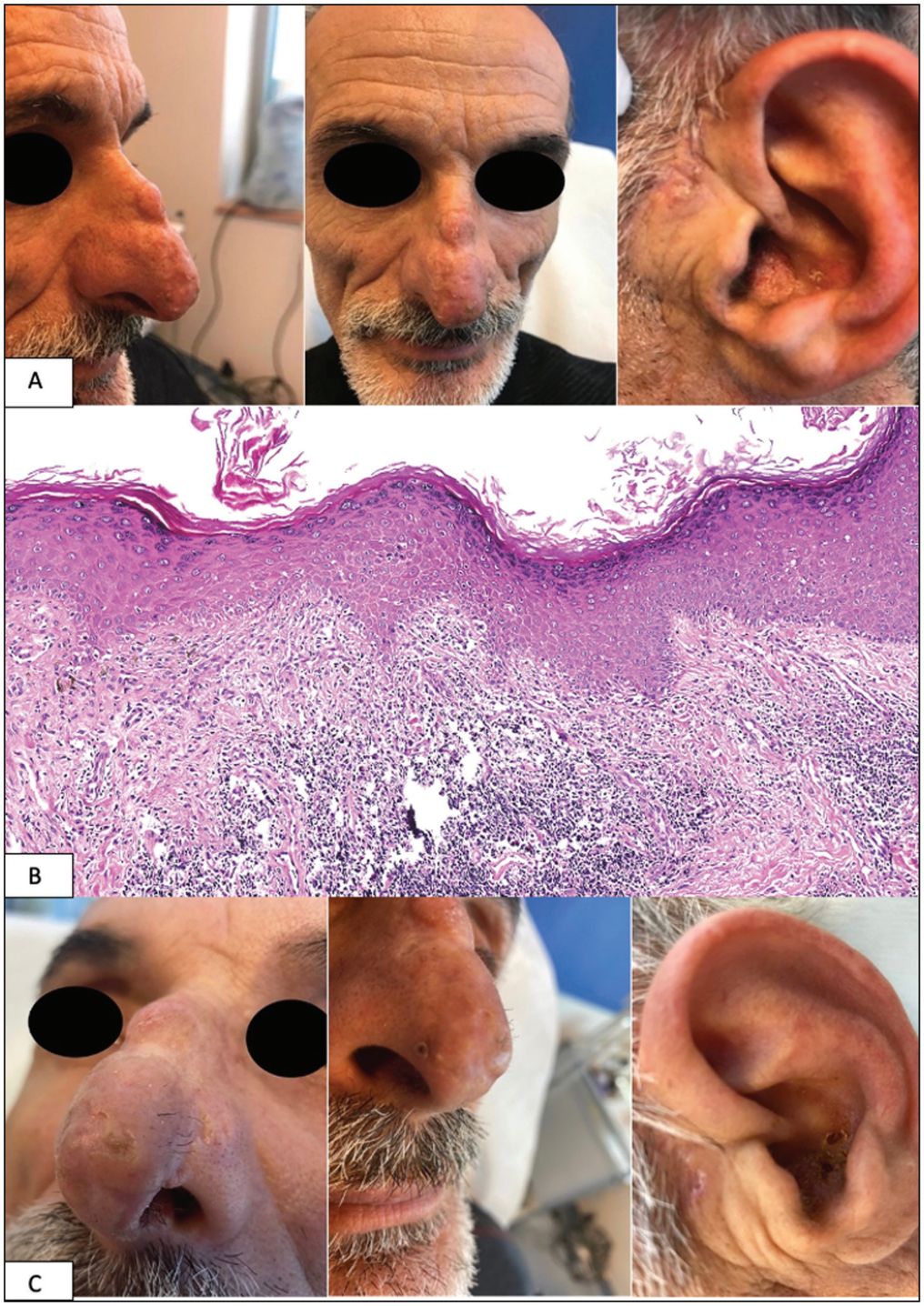

Figure 1: (A) Hyperkeratotic plaques on the nose, preauricular area, and external ear canal. (B) Lichenoid tissue reaction resembling lichen planus. Note orthokeratosis, hypergranulosis, and irregular acanthosis of the epidermis. Note the hydropic degeneration and eosinophilic colloid bodies at the basal cell layer (H&E, X200). (C) Regressed hyperkeratotic plaques on the nose and preauricular area. Note the crust on the external ear canal

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