Vanishing Seborrhoeic Keratosis

Seborrhoeic keratosis (SK) is the most common benign tumor of middle and older ages (1), and can appear in any part of the body except mucous membranes. Its clinical diagnosis is almost always easy and usually offers no problems. Unlike common SK, the inflammatory variety of the disease is often difficult to diagnose and may be clinically confused with basal cell carcinoma, squamous cell carcinoma, and even malignant melanoma (2,3), with eczematous changes in and around SK with an unknown cause. On the other hand, only 8% of cases of inflammatory SK occur on the extremities (2).We report on a patient with an eczematous SK located on one leg, with complete regression a few months after inflammation.

An 89-year-old man with no history of relevant diseases was admitted for a red, slightly elevated, desquamative inflammatory plaque covered with haemorrhagic crusts located on the frontal and inner aspect of his right leg. The lesion measured about 3 centimeters in diameter and had appeared 6 months earlier (Figure 1). Dermoscopy was not performed. A biopsy of the lesion was done, with the presumptive clinical diagnosis of basal cell carcinoma, squamous cell carcinoma, or amelanotic melanoma. The skin biopsy specimen showed an epidermis with acanthosis, hyperplasia of basaloid keratinocytes, and horn pseudocysts with parakeratotic and ortho- hyperkeratosis, along with lymphoid exocytosis, spongiosis, and necrosis of keratinocytes.

There was an inflammatory infiltrate in the dermis, without athypic characteristics (Figures 2 and 3). During suture removal in the second week, the presence of pus was detected, so the patient was treated with oral flucloxacillin at 500 mg every 8 hours for 7 days, as well as topical chloramphenicol. After initial improvement, the lesion worsened and eczema appeared at day 40; a cream containing 0.05% betamethasone dipropionate 0.1% and gentamicin sulfate was then administered 2 times per day. One month later, the lesion had completely disappeared, showing only a slight postinflammatory hyperpigmentation (Figure 4).

The first spontaneous regressions of SK were reported in two patients with multiple lesions. The first case occurred after an exfoliative erythroderma with the inflammation of preexisting SK and also the appearance of new ones, which was associated with a benign renal cyst (4): The second patient, who presented with pruritus of the trunk and extremities that had lasted for 1 month, showed multiple inflammatory SK along with a concomitant nasal adenocarcinoma with an evolution of two years, which produced nasal obstruction (5). On both occasions there was some regression of SK after its inflammatory phase, along with the removal of tumors. The inflammation that preceded the disappearance of SK consisted in a mononuclear inflammatory process of the dermis, with exocytosis in the epidermis and epidermal degenerative changes. This pattern is common to other

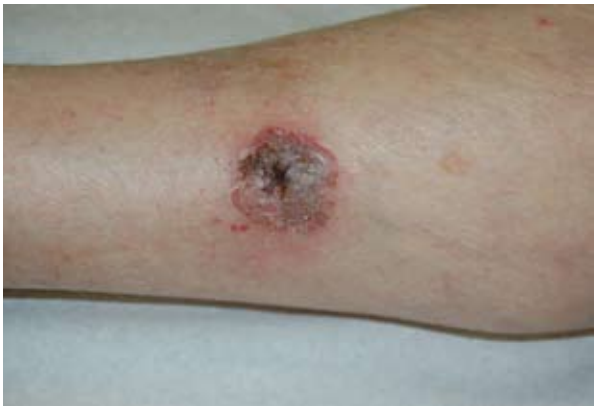

Figure 1. A desquamative red inflammatory plaque covered with haemorrhagic crusts located on the inner aspect of the leg.

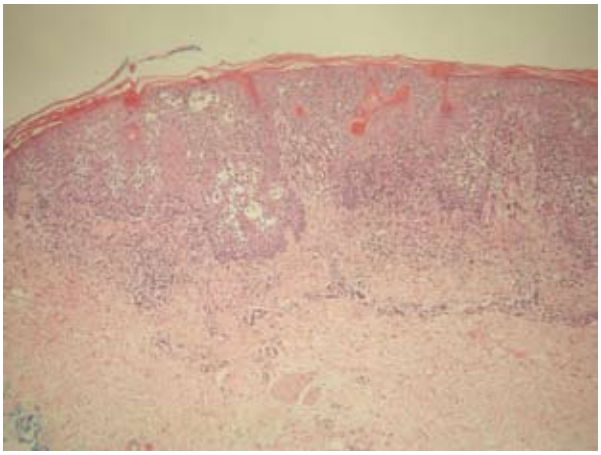

Figure 2. Epidermis with acanthosis, hyperplasia of basaloid keratinocytes, hyperkeratosis, and horn pseudocysts (H&E; x 40).

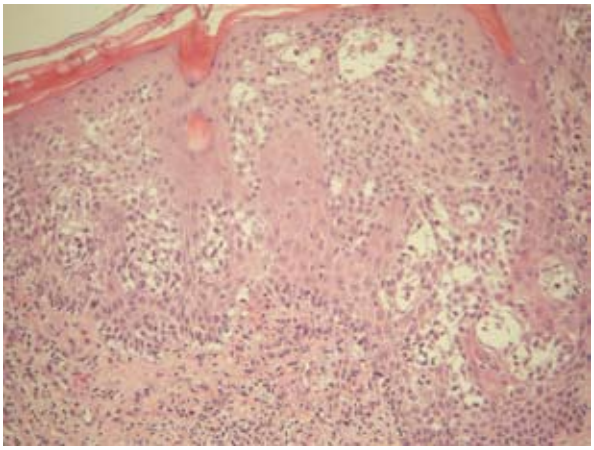

Figure 3. Epidermis shows lymphoid exocytosis, spongiosis and scattered necrotic keratinocytes. Lymphohistiocytic perivascular and interstitial infiltrate in the dermis without atypia (H&E; x 100)

inflammatory skin tumors which show spontaneous regression, including verruca vulgaris, halo nevus, molluscum contagiosum, keratoacanthoma, and even partial or complete regression of basal cell carcinoma and melanoma (4).In recent years, the disappearance of SK through an intermediate stage called lichenoid keratosis has been reported by Zaballos et al. (2,6-8). Dermoscopy was helpful in the differentiation of pigmented lesions in these cases – some of them with clinical diagnoses of malignant melanoma or basal cell carcinoma– by showing areas of SK and lichenoid keratosis in the same tumours (2).

In 1966, lichenoid keratosis was classified as a form of lichen planus because histological features were very similar to that of lichen ruber planus. This was not a diagnosis we could consider in our patient, because there was no band-like mononuclear inflammatory infiltrate in the dermis or blurring of the dermoepidermal junction. In other cases, the disappearance of SK after transformation to lichenoid keratosis has been reported, leading to complete regression (8). The regression of the lesion in our case took about 7 months. In more than half of the reported cases the lesions disappeared completely or almost completely in the first 12 months after initiation of inflammation (8). Another circumstance in which the regression of SK is observed, occurs after treatment or removal of a malignancy in a patient presenting the sign of Leser-Trelat (9). The relevant features of our case were the size and appearance of the SK, which was not clinically suspected at any point, and its spontaneous regression.

The possibility of an inflammatory variant of SK in any location must be kept in mind in order to avoid unnecessary or aggressive treatments.

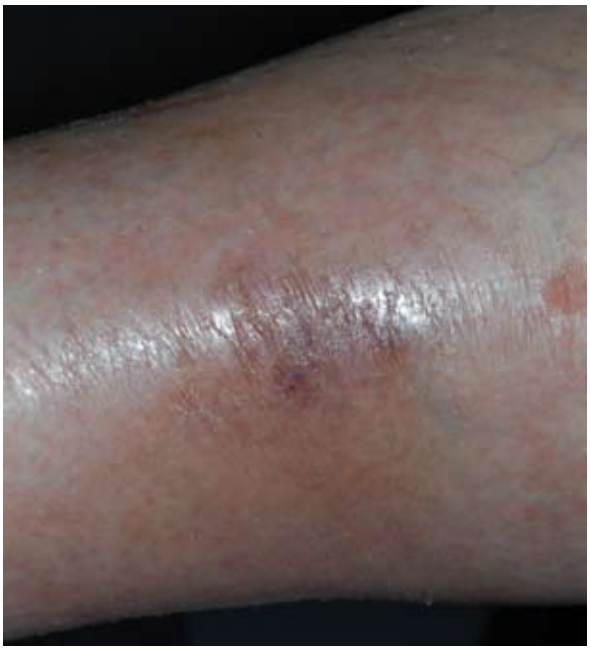

Figure 4. Complete regression of the lesion leaving slight postinflammatory hyperpigmentation.

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