hemangioma (hobnail hemangioma).^{2,8,9} Immunohistochemistry with antibodies to HHV-8 plays an essential role in establishing the definitive diagnosis.^{6,9,10}

The histogenesis of KS has been the subject of considerable debate; discussion centers on whether this disease is blood-borne or originates in the lymphatic endothelial cells. With regard to lymphangioma-like KS specifically, histologic findings suggest that it originates from lymphatic endothelial cells, as initially suggested by Gange and Jones.² This interpretation is consistent with recent immunohistochemical findings showing intense expression of several markers that are specific for the lymphatic endothelium; this was the pattern we saw in the neoplastic cells from our patient. It has been postulated that chronic lymphedema or a history of radiotherapy in the affected area could increase the risk of developing lymphangioma-like KS lesions.^{1,5} Our patient had never received radiotherapy and did not present with chronic lymphedema. In addition, the lesion in which we detected the lymphangioma-like KS was located on the forearm, not on a lower limb as described in most cases of this type of KS.

This new case of lymphangioma-like KS involved a history of histologically classic KS with successive recurrences consisting of lesions with the same clinical appearance, but with histopathologic findings suggestive of lymphangioma. A detailed histologic study in combination with immunohistochemistry, such as staining for HHV-8 latent nuclear antigen, is essential for correctly diagnosing lymphangioma-like KS.

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Queratoacantoma digital distal: importancia del diagnóstico diferencial con el carcinoma escamoso subungueal

To the Editor:

Subungual keratoacanthoma is a rare, destructive variant of keratoacanthoma that seldom regresses spontaneously. It may involve the distal tissue under the nail or the proximal nail fold and sometimes also affects the underlying bone. Histopathology is similar to that of other solitary keratoacanthomas but the subungual form shows more pronounced dyskeratosis with little or no nuclear atypia. We report the case of a 39-year-old Caucasian woman with no relevant past medical history who presented with a very painful hyperkeratotic nodular lesion under the distal portion of the nail of the fourth finger of her left hand; the lesion had grown rapidly during the previous month (Fig. 1). A radiograph of the left hand revealed an osteolytic lesion in the phalanx underlying the nodule. On the basis of these findings we carried out a complete excision of the lesion, adopting a conservative approach with regard to the underlying bone.

Histopathology using hematoxylin-eosin staining revealed epidermal hyperkeratosis, parakeratotic foci, and a



Figure 1 Nodular lesion in the distal subungual region of the fourth finger of the left hand.

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Figure 2 A, Crater filled with amorphous keratin (hematoxylin-eosin, original magnification \times 40). B, Epidermal hyperkeratosis, foci of parakeratosis (hematoxylin-eosin, original magnification \times 100). C, Dyskeratotic cells with little nuclear atypia (hematoxylin-eosin, original magnification \times 400). D, Positive staining for Ki-67 in the stratum basale.

central crater filled with amorphous keratin. Other findings were dyskeratotic cells, a small number of intraepithelial neutrophils and eosinophils, little nuclear atypia, patchy infiltrates of lymphocytes and plasma cells, and little or no fibrosis at the bases of the lesions (Fig. 2a-c). Staining for Ki-67 only revealed positivity in the stratum basale (Fig. 2d). All these findings were consistent with subungual keratoacanthoma.

The differential diagnosis of a painful nodular lesion on the distal phalanx includes dermoid cyst, subungual fibroma, glomus tumor, giant cell tumor of the tendon sheath, digital mucoid cyst, common wart, subungual exostosis, amelanotic melanoma, subungual squamous cell carcinoma (SCC), and subungual keratoacanthoma.¹

Subungual keratoacanthoma is a rare, aggressive variant of this tumor which has a higher prevalence in men and a tendency to appear on the first 3 fingers of the hand, particularly the thumb. It consists of a painful, localized endo-exophytic nodular lesion that has a characteristic central keratin-filled crater. Subungual keratoacanthomas differ from typical forms of this tumor in their appearance on hairless skin and the absence of characteristic epithelial cords.² They tend to be associated less with inflammation and more with deep invasion and a greater number of eosinophilic dyskeratotic cells. The diagnosis should be based on the correlation of clinical, radiologic, and histologic findings.

Subungual keratoacanthoma should be considered in the differential diagnosis of subungual tumors, especially SCCs (Table 1). Radiologically, a subungual keratoacanthoma is almost indistinguishable from a subungual SCC. However, a keratoacanthoma causes a phalangeal lesion with a well-defined border as it expands but does not infiltrate the bone; in addition, keratoacanthomas usually occur in the fifth decade of life, whereas SCCs usually occur in the

	Keratoacanthoma	Squamous Cell Carcinoma
Clinical characteristics	Fifth decade	Seventh decade
	Rapid growth	Slow growth
	Spontaneous regression	No regression
Radiologic findings	Osteolytic lesion with well-defined borders	Poorly-defined borders
Histopathologic findings	Symmetric exophytic lesion	Proliferation of epidermal cells with
	Epidermal hyperkeratosis	marked cellular and nuclear atypia
	Central crater filled with keratin	Abnormal mitotic figures
	Dyskeratotic eosinophilic cells	Invasion of the dermis and underlying
	Little nuclear atypia	tissues
	Patchy dermal infiltration of lymphocytes	
	and plasma cells	
Immunohistochemical characteristics	No increase in Ki-67/p53 expression	Elevated Ki-67/p53 expression

 Table 1
 Keys to Differential Diagnosis Between Subungual Keratoacanthoma and Squamous Cell Carcinoma.

seventh decade of life. SCCs grow slowly, whereas keratoacanthomas grow rapidly for a few weeks or months before they stabilize and regress spontaneously. If a subungual keratoacanthoma does not regress spontaneously, it can become locally destructive, making differential diagnosis difficult, and cases of malignant transformation to SCC have been reported. The differential diagnosis is currently subject to debate. Some authors believe that these keratoacanthomas are SCCs of low-grade malignancy that can be locally invasive and spread to the underlying bone; however, keratoacanthomas often involute spontaneously.^{3–5} Recent studies of differences in the expression of certain markers between these 2 tumors (higher expression of Ki-67 and p53 proteins in SCC) that help to establish the diagnosis have concluded that they are separate lesions that behave differently.^{6,7}

Therefore, although subungual keratoacanthoma is a rare clinical entity, it is a destructive variant and its differential diagnosis with respect to subungual SCC is essential. Both can present as a painful nodular lesion associated with inflammation and can affect the distal subungual tissue and underlying bone.⁷⁻¹⁰ Although the 2 conditions can be almost indistinguishable, their prognosis and treatment are different. Subungual keratoacanthoma is treated conservatively, whereas Mohs micrographic surgery is indicated for SCCs that are noninvasive (without bone involvement) and amputation is performed when an SCC has invaded the bone (in cases of rapid growth or delayed diagnosis).^{11,12}

In conclusion, we have reported an unusual case of subungual keratoacanthoma on the fourth finger of a 39-year-old woman, highlighting the importance of considering this diagnosis when SCC is suspected, in the interest of avoiding mutilating treatments.

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Alopecia lipedematosa concomitante con psoriasis del cuero cabelludo

To the Editor:

Lipedematous scalp is a rare condition of unknown etiology characterized by a thickening of the subcutaneous tissue on

the scalp. It typically affects the occipital region, and may be accompanied by pain and itching in the affected area. When the condition is associated with hair loss, it is called lipedematous alopecia.¹ We report a case of lipedematous alopecia and briefly review the literature.

The patient was a 49-year-old Spanish woman with a history of mild to moderate psoriasis involving both the scalp and the nails since the age of 20 years. She had had surgery for breast cancer (T3 N1 M0) 3 years before this consultation and had been receiving treatment with tamoxifen for about 6 months. She came to our clinic because of severe pain and thickening of the scalp, which had started some 3 months earlier, and more recent hair loss in the occipital region. Three weeks earlier, the patient had visited the emergency department of our hospital complaining of severe scalp pain. A computed tomography scan revealed asymmetry in soft tissue volumes, with significant thickening of the

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