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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Lipedematous Alopecia in a Patient With Scalp Psoriasis☆

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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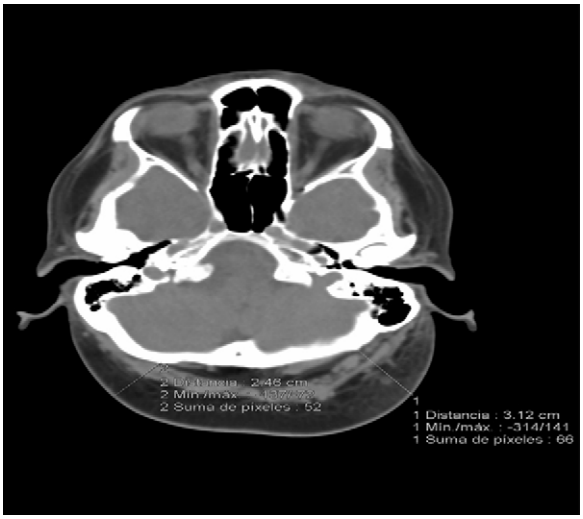

Figure 1 Computed tomography scan showing thickening (3.12 cm) of the subcutaneous cell tissue in the left occipital area of the scalp.

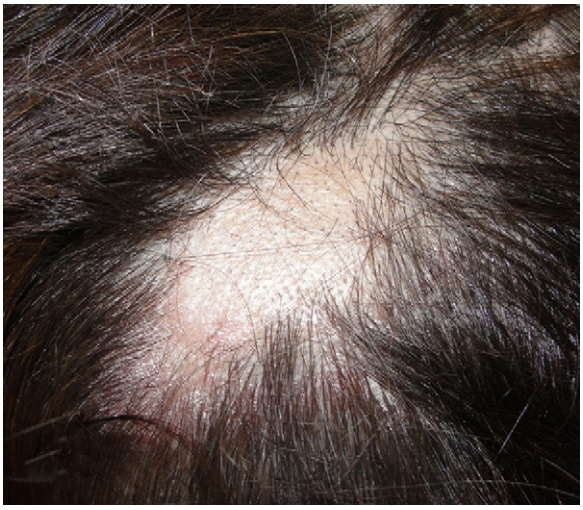

Figure 2 Clinical image of an alopecic plaque in the left parietal region.

subcutaneous layer (3.12 cm) in the vertex and left occipital area (Fig. 1).

On physical examination, the skin of the parieto-occipital area was found to be edematous and boggy, and 2 hairless patches each measuring about 2×2 cm and similar in appearance to alopecia areata plaques were observed in the left parietal region. There were also several psoriatic lesions on the scalp, which the patient said had been stable without treatment for several months (Fig. 2).

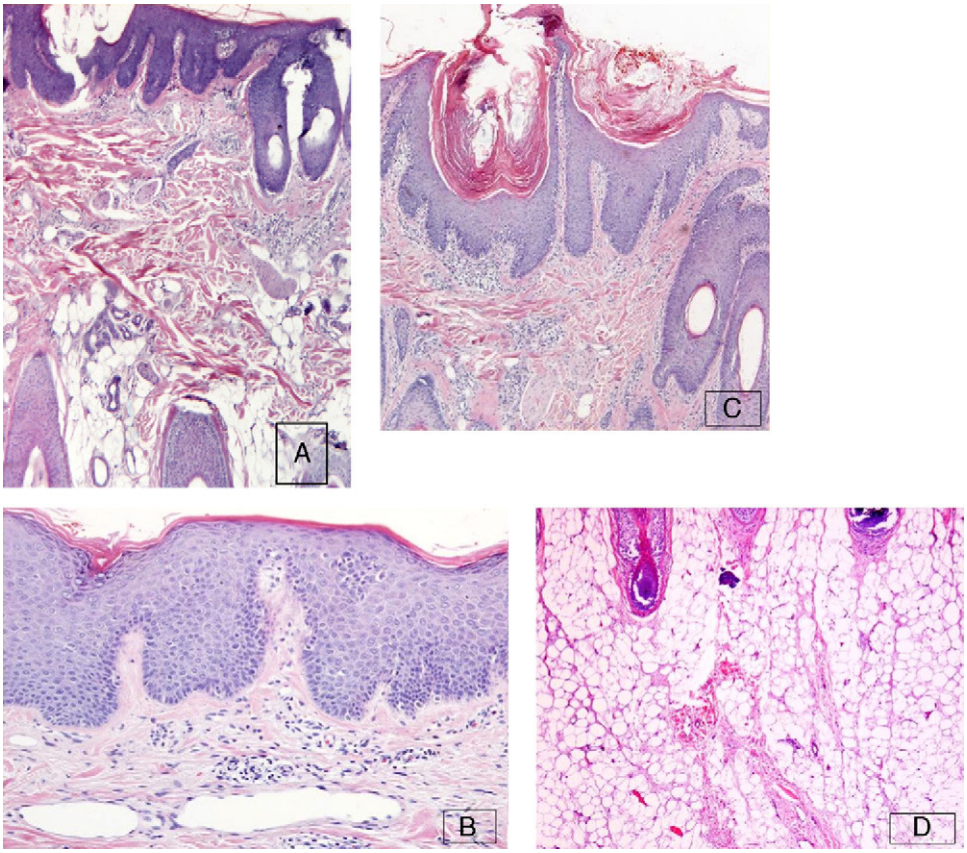

Figure 3 A, Biopsy of the scalp showing the thickened layer of subcutaneous cell tissue with normal follicle morphology (H&E, original magnification x4). B, Epidermal hyperplasia with keratin plugs in the follicular infundibula (H&E, original magnification x20). C, Dilated dermal blood vessels with a telangiectatic appearance (H&E, original magnification x10). D, No evidence of abnormal deposition or panniculitis (H&E, original magnification x10). Abbreviation: H&E, hematoxylin-eosin.

Table 1 Reported Cases of Lipedematous Scalp and Lipedematous Alopecia.

Author/Year	Condition	Patient Characteristics	Duration	Thickness, mm	History
Cornbleet[2]/1935	LS	F/Blk/44 y	6 y	-	None
Coskey et al.[3]/1961	LA	F/Blk/28 y	2 y	15	Diabetes mellitus
	LA	F/Blk/75 y	1 y	10	
Curtis and Heising/1964	LA	F/Blk/62 y	15 y	15	Skin and joint hyperelasticity
Lee et al./1994	LS	F/Blk/32 y	3 y	10.7	None
Kane et al./1998	LA	F/Blk/49 y	4 mo	12.6	None
Fair el al.[4]/2000	LA	F/Blk/18 y	6 mo	9	None
Bridges et al.[8]/2000	LA	F/Blk/48 y	6 y	12	Kidney failure
Ikejima et al.[6]/2000	LA	M/Asn/30 y	7 y	16	None
Tiscornia et al.[5]/2002	LA	F/Wh/69 y	6 mo	10	None
Scheufler et al./2003	LS	F/Wh/51 y	1 y	15	Neck rigidity
Bukhari et al.[7]/2004	LS	F/Asn/57 y	-	19.2	None
Martín et al.[9]/2005	LA	F/Wh/77 y	1 y	11	None
	LA	F/Wh/59 y	1 y	9.2	Sjogren syndrome
	LS	F/Wh/48 y	2 mo	10.8	None
High and Hoang/2005	LA	F/Blk/57 y	10 y	12-15	Discoid lupus
	LS	F/Blk/55 y	-	10-15	None
Piraccini et al./2006	LA	M/Wh/48 y	10 y	11	Androgenic alopecia
	LA	M/Wh/53 y	4 y	12	Androgenic alopecia
Rowan et al./2006	LS	F/Blk/9 y	6 mo	9.8	None
Yasar et al./2007	LA	F/Wh/45 y	-	10	None
	LA	M/Wh/49 y		12	
	LS	F/Wh/62 y		18	
	LA	F/Ira/45 y	5 y	10.7	None
El Darouti et al./2007	LA	F/Egy/50 y	5 y	8.5	None
	LA	F/Egy/30 y	2 y	4.6	
	LA	F/Egy/40 y	2 y	4.6	
	LS	F/Egy/36 y	2 y	5.1	
	LS	F/Egy/17 y	2 mo	4.8	
	LS	F/Egy/21 y	1 y	3.1	
	LS	F/Egy/39 y	2 y	2.5	
	LS	F/Egy/11 y	1 y	4.5	
	LS	F/Egy/35 y	-	2.2	
	LS	F/Egy/40 y	2 y	4.6	
Martínez-Morán et al[1]/2007	LS	F/Wh/77 y	6 mo	15	None
González-Guerra et al.[10]/2008	LA	F/Wh/52 y	5 y	15	Polycystic ovary
Case described	LA	F/Wh/49 y	3 mo	31 mm	Scalp psoriasis Breast cancer

Abbreviations: Asn, Asian; Blk, Black; Egy, Egyptian; F, female; Ira, Iranian; LA, lipedematous alopecia; LS, lipedematous scalp; M, male; Wh, White.

A particularly thick area of the subcutaneous tissue in the left occipital area was biopsied. Histology showed thickened subcutaneous fatty tissue, dermal edema, and a mild perivascular lymphocytic infiltrate in the superficial dermis. There was no evidence of an increase in mucin deposition in the dermis or subcutaneous tissue. Epidermal hyperplasia and keratin plugs in the follicular infundibula were observed. The number of terminal hair follicles in the anagen phase was normal, and dermal blood vessels were telangiectatic (Fig. 3). No significant abnormalities were found in the blood count, biochemistry, antinuclear antibody test, or thyroid profile. On the basis of this evidence, the diagnosis was lipedematous scalp with areas of lipedematous alopecia.

The alopecic plaques and psoriatic lesions were treated with a topical corticosteroid, and hair regrowth was evident within 6 weeks. Oral nonsteroidal antiinflammatory drugs were prescribed for pain control, to be taken as needed.

On follow-up at 9 months, no alopecia was evident, and the pain had lessened considerably. At her final visit, the patient reported that she had had surgery for chronic right-arm lymphedema (a side effect of her breast cancer surgery) with a very satisfactory functional outcome some 6

weeks earlier, and that the scalp discomfort had disappeared entirely.

Lipedematous scalp was first described in a black woman in 1935 by Cornbleet.[2] In 1961, reporting the cases of 2 black women, Coskey et al.[3] coined the term *lipedematous alopecia* to refer to thickening of the subcutaneous tissue on the scalp coinciding with the inability of the hair to grow more than 2 cm. To date only 19 cases of lipedematous alopecia and 16 cases of lipedematous scalp have been reported (Table 1), none associated with any pathology of relevance. In addition to a localized or generalized thickening of subcutaneous tissue, patients may report diffuse pain, numbness, and itching. The thickness of subcutaneous tissue can be measured using ultrasound, magnetic resonance, or computed tomography; a normal thickness in healthy adults is 5.8 $\pm$0.12 mm at the bregma. Lipedematous scalp and lipedematous alopecia are probably underdiagnosed; indeed, there is still no agreement on whether they are different processes or simply different stages of the same process.[1]

Histological findings for lipedematous alopecia are a thickening of the subcutaneous cell tissue, variable dermal edema, a perivascular lymphocytic infiltrate, an absence of abnormal deposition, no evidence of panniculitis, epidermal hyperplasia, and keratin plugs in the follicular infundibula. Terminal hair follicles may be reduced in number or preserved. Dermal blood vessels are often dilated and telangiectatic in appearance.[4]

The pathogenesis of lipedematous scalp and lipedematous alopecia is unknown. Some authors have suggested that there may be a hormonal factor because most cases are diagnosed in women. Race, once thought to be a key factor, is now considered less important because of the growing number of cases reported in white,[5] Asian,[6] and Middle Eastern[7] women.

One theory proposed to explain hair loss is that the thickening of the subcutaneous tissue may place pressure on the hair follicles, restricting hair growth or shortening anagen cycles.[8] Lymph vessels were dilated in our patient, and Martín et al[9] reported the same for 2 patients with lipedematous alopecia, suggesting that this effect might play an important role in the pathogenesis of the alopecia. Regarding the association with other systemic diseases, cases have been reported of patients with a history of diabetes mellitus,[3] joint and skin hyperelasticity, and acute kidney failure.[8]

No case has been reported to date of lipedematous alopecia in a patient with comorbid scalp psoriasis or a history of malignancy. Noteworthy in our patient was the intense scalp pain, the very thick subcutaneous layer, the rapid resolution of the alopecic plaques, and the favorable course after surgical treatment of the chronic lymphedema of the arm. Since the patient was already responding to treatment we do not know if the apparently favorable post-surgical response was a coincidence or whether, given the histological evidence of dilated lymph vessels, there was an actual causal relationship between the events.

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