Case and Review

Lipedematous Alopecia: Clinical, Histopathological, and Trichoscopic Findings of a Single Case and Review of the Literature

Ornella Accaputo^a Jonathan Michael White^a Athanassios Kolivras^b
Niloufar Sadeghi^c Evelyne Berlingin^d Véronique del Marmol^a

^aDermatology Department, Erasme Hospital, ULB, Brussels, Belgium; ^bDermatology Department, Saint-Pierre Hospital, ULB, Brussels, Belgium; ^cNeuroradiology Department, Erasme Hospital, ULB, Brussels, Belgium; ^dDermatology Department, CHU Ambroise Paré Hospital, ULB, Mons, Belgium

Keywords

Alopecia · Histology · Lipedema · Lipomatosis · Scalp · Trichoscopy

Abstract

A 66-year-old African woman suffered from a boggy thickening of the scalp in the frontoparietal area and the vertex accompanied by headache since infancy. In her thirties, she developed diffuse hair loss in the same area. Clinical and trichoscopic examination were unhelpful, but the histopathological and radiologic findings led to a diagnosis by demonstrating thick subcutaneous adipose tissue. First described in 1935 by Cornbleet [Arch Dermatol Syphilol. 1935;32:688], lipedematous scalp is a rare scalp disease marked by a soft thickening of the scalp. A similar clinicopathologic entity associated with nonscarring but permanent acquired alopecia was described in 1961 by Coskey et al. [Arch Dermatol. 1961 Oct;84(4):619–22] and termed lipedematous alopecia. In this article, we report a rare case of lipedematous alopecia with emphasis on a trichoscopic and histopathological description with review of the literature.

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Dr. Ornella Accaputo
Dermatology Department, Hôpital Erasme, ULB
808, route de Lennik
BE-1070 Bruxelles (Belgium)
E-Mail ornella.accaputo@erasme.ulb.ac.be

Our case highlights the importance of palpation during the clinical examination. It remains to be determined whether this is actually a specific disease entity or a combination of 2 separate conditions. Symptomatic treatment is advisable given the lack of robust published data on treatment. Only 40 cases of lipedematous alopecia and 52 cases of lipedematous scalp have been published in the literature. Our case is the 93rd.

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Case Report

A 66-year-old African woman reported diffuse hair loss for 35 years. She described her scalp as feeling like cotton wool since her childhood. There was no clinical improvement with local application of minoxidil for several months.

Given her African origin, we took special care to ask about hair-care practices. She had relaxed her hair on many occasions but only rarely braided her hair.

She had been taking β -blockers (nebivolol) for hypertension for almost 15 years, had suffered from recurrent headaches since childhood, and was treated with hydroxychloroquine for Sjögren's syndrome for 1 year. She reported that her daughter's scalp was also thicker, with a cotton wool-like feeling but without hair loss, and there was no other family history of androgenetic alopecia.

Physical examination revealed diffuse alopecia of the vertex and frontoparietal area with an edematous, boggy, and spongy consistency of the scalp. Only a few hairs persisted on the occipital region (Fig. 1a). The lesions were easily compressed down to the underlying bone but came back instantly to their initial form, like a balloon (Fig. 1b).

Trichoscopic examination showed the honeycomb network and pinpoint white dots normally seen in dark skin phototypes. No vellus hairs were observed, and persistent hairs seemed to be homogeneous, though very short (<2 cm).

On closer inspection, there was subtle erythema and hyperpigmentation around the remaining follicles and in some inter-follicular areas. No tufted hair, peri-follicular scaling, or other cicatricial trichoscopic (white dots, etc.) findings were present (Fig. 1c; Table 1).

Histopathological findings were characterized by an expansion of subcutaneous tissue accompanied by infiltration of fat into the superficial dermis. Some dilated lymphatic vessels were also seen. The number of hair follicles was decreased. Both dermis and subcutaneous tissue were devoid of inflammatory infiltrate and of scarring (Fig. 2a, b). Direct immunofluorescence was negative.

Blood tests were within normal limits, except for positive antinuclear antibodies (titer at 1/320 with anti-SSA positive). Magnetic resonance imaging was performed to investigate chronic headaches. No parenchymal anomalies could be seen, but subcutaneous fatty infiltration was evaluated with a maximum thickness of 12 mm in the parietal region. Subcutaneous fat is best seen on T1- and T2-weighted images as an area of high signal intensity (Fig. 2c compares our patient with an age- and sex-matched control of the same ethnicity, Fig. 2d).

Discussion

Lipedematous scalp is a rare entity characterized by a thickened boggy scalp. Cornbleet [1], in 1935, was the first to describe this entity. Thereafter, in 1961, Coskey et al. [2] described 2 similar entities accompanied by short hairs and/or nonscarring but permanent alopecia and called it “lipedematous alopecia.” Müller et al. [3] objected to the terms “lipedematous alopecia” or “lipedematous scalp” and proposed a more general term, “localized lipomatosis of the scalp with or without alopecia,” to classify it among the group of conventional lipomatosis. He felt that the conditions were all on the same disease spectrum, each with a different localization.

Since then, 92 cases (40 lipedematous alopecia and 52 lipedematous scalp) have been reported in the literature (Table 2, Table 3). Most often located at the vertex and occiput, it generally spreads to the entire scalp. The disease progresses during months or years and then tends to stabilize spontaneously, generally without ever regressing.

Being almost invisible, this disease highlights the clinical importance of palpation and is consequently probably frequently undiagnosed and therefore underreported. The lesions are easily compressed down to the underlying bone, but come back instantly to their initial form, like a balloon. Accompanying symptoms, such as pruritus, pain, headaches, or paresthesias, have been described (Table 2, Table 3).

The subcutaneous tissue thickening, approximately double the normal, is the main abnormality found. Using the data from the different measurement methods on the 92 published cases as well as our case, we calculated a mean thickness of scalp fat (12.0 ± 5.6 mm; range 2.2–31.2 mm) (Table 2, Table 3). As a reference, 2 authors measured normal thickness of the scalp in healthy individuals (5.5 mm) [4]. We have doubts about the series of cases described by El Darouti et al. [5], given the small reported scalp thicknesses, and would suggest another diagnosis. A thickness of a minimum of 10 mm is necessary to evoke the diagnosis of lipedematous scalp/alopecia.

Isolated systemic diseases have been described, but no consistent associations are present (Table 2, Table 3). The etiology and pathogenesis remain unknown [3]. Predominantly described in female patients, hormonal factors have been suspected [6, 7]. At first reported in African-American patients, the proposed ethnic predilection seems to be a reporting bias, given the recent published cases of different ethnic origins (Table 2, Table 3). Mostly described in middle-age patients, a few young people, including 2 congenital cases, have also been reported [8, 9]. Family history of the same disease has been reported, which, along with congenital involvement, suggests genetic influences [7, 9, 10]. Nevertheless, hair styling habits should also be discussed.

Leptin, a hormone that regulates the structure and distribution of adipocytes, has also been implicated. A reactive or compensatory response to undetected stimuli, hormonal factors, or a genetic tendency is also a possibility [11]. Yasar et al.’s [7] case series shows a predominance of obese patients and proposes a mechanism of inadequate lipid distribution with impairment in the lymphatic flow.

Only 2 reports mention a trichoscopic examination with the finding of multiple linear telangiectasias [12, 13]. Not present in our patient, this may be explained by the dark skin phototype, making erythema difficult to see or possibly because of the late stage of the disease.

Normally with a whitish halo in dark-skin phototypes, the slight inter-/peri-follicular hyperpigmentation seen in our case may be a postinflammatory sign.

The principal histopathological finding is the increased thickness of the subcutis, which is approximately doubled [7, 14–16]. In the majority of cases, no mucin deposits in the dermis or subcutis have been found. Lipedematous alopecia is characterized by the additional feature of reduction in the number of hair follicles, which is best seen on horizontal sections [14].

Different hypotheses have been made on the development of hair loss, such as a role of lymphangiectasia [6], an inadequate vascular supply and altered microenvironment created by adipocytes-coating follicles [2, 14]. However, there are features which mitigate these theories: in vitro fat cells show a positive effect on hair follicle cells [11, 17], leg lipedema, sharing a common histologic pattern, never causes hair abnormalities [14], and finally, lipedematous scalp never evolves to alopecia [18].

Scheufler et al. [16] suggest that lipedematous scalp is not a precursor of lipedematous alopecia, but these are 2 distinct diseases or 2 different phenotypic expressions of the same disease. Another possible assumption is that lipedematous alopecia does not really exist per se but is actually lipedematous scalp accompanied by a different underlying pathology of alopecia. Several differential diagnoses of alopecia can be evoked in our patient, including traction alopecia, androgenetic alopecia, or cicatricial alopecia, but these are difficult to confirm given the rather late diagnosis. Nevertheless, the most probable diagnosis is a central centrifugal cicatricial alopecia considering the compatible trichoscopy.

An important and frequently discussed differential diagnosis is cutis verticis gyrata, which shows increased thickness, but differs clinically with furrows or convolutions of the skin. Encephalocraniocutaneous lipomatosis is another differential diagnosis which is histopathologically similar but differs clinically by the neurocutaneous syndrome [19].

Therapeutic prognosis is very poor. Topical and locally injected steroids, surgery, and systemic therapies have shown slight or no improvement. Considering the paucity of published cases, the disease might have burnt itself out without any treatment, so there is insufficient evidence on which to evaluate treatments. Symptomatic treatment would seem to be the most pragmatic approach.

In conclusion, we report a rare case of lipedematous alopecia with emphasis on trichoscopic and histopathological description. Our case highlights the importance of palpation during the clinical examination. It remains to be seen whether this is a specific disease entity or a combination of 2 separate conditions. Symptomatic treatment is advisable given the lack of robust published data on different treatment approaches.

Statement of Ethics

The authors have no ethical conflicts to disclose.

Disclosure Statement

The authors have no conflicts of interest to declare.

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Fig. 1. Clinical examination showing diffuse hair loss in the vertex and frontoparietal area with hair persisting in the occipital region (a) and marked but transient edema on palpation (b). Trichoscopy (c) showing the honeycomb network (continuous black line, 1) and pinpoint white dots (small continuous black circles, 2). Slight erythema and hyperpigmentation around the remaining follicles (large discontinuous black circle, 3) and in inter-follicular areas (discontinuous black square, 4).

Fig. 2. Anatomopathological examination. **a** Vertical cut showing a normal epidermis, atrophic dermis, dilated vessels, slight perivascular mononuclear infiltrate and invasion of adipose tissue in the superficial dermis, and a thicker subcutaneous adipose tissue. **b** Horizontal cut showing reduced number of pilosebaceous units and no significant fibrosis or inflammatory infiltrate. Magnetic resonance imaging. **c** Sagittal T1-weighted image shows the subcutaneous fat as a high signal. Intensity layer with a maximum thickness of 12 mm measured at the parietal region. **d** Comparative sagittal T1-weighted image of a patient of the same sex, age, and ethnicity, showing a normal thickness of 3 mm.

Table 1. Characteristics of our case

Dots	Vessels	Skin	Hair shaft
Pinpoint white dots	Not observed (contrast is hard because of dark skin phototype)	Honeycomb network	No vellus hair
No cicatricial white dots		Slight erythema and hyperpigmentation around follicles and interfollicular areas	Same range of diameter (homogenous)

Table 2. Reported cases of lipedematous scalp and lipedematous alopecia

First author [ref.]	Year	Diagnosis	Age, years	Gender	Ethnicity	Family history	Thickness, mm	Measurement	Clinical	Associated disorders
Cornbleet [1]	1935	LS	44	F	B	(–)	(–)	(–)	P/I	(–)
Coskey [2]	1961	LA	28	F	B	(–)	15	N	P	Heart murmur
		LA	75	F	B	(–)	10	N	I/Pa	Diabetes mellitus
Curtis [cited in 7]	1964	LA	62	F	B	(–)	15	N	I	Skin hyperelasticity and joints hyperlaxity
Lee [cited in 7]	1994	LS	32	F	As	(–)	10.7	CT	A	None
Kane [cited in 7]	1998	LA	49	F	B	(–)	12		Pa	None
Fair [14]	2000	LA	18	F	B	No	9	B	P	None
Bridges [cited in 7]	2000	LA	48	F	B	No	10	N	I	Chronic renal failure
Ikejima [cited in 7]	2000	LA	30	M	As	No	16	MRI	A	ANA +
Tiscornia [cited in 7]	2002	LA	69	F	W	(–)	10	CT	I	None
Scheufler [16]	2003	LS	51	F	W	(–)	15	N	H	(–)
Bukhari [cited in 7]	2004	LS	57	F	Ar	No	21.2	CT	A	Tachycardia/hypercholesterolemia/HTA
Mohsin [cited in 7]	2004	LA		F	W	(–)				(–)
Martín [6]	2005	LS	48	F	W	(–)	10.8	US	A	(–)
Martín [6]	2005	LA	77	F	W	No	11	US	I	No
		LA	59	F	W	(–)	9.2	US	P/I	Sjögren's syndrome
High [cited in 7]	2005	LA	57	F	B	(–)	15	N	(–)	Discoid lupus erythematosus
High [cited in 7]	2005	LS	55	F	B	(–)	15	CT		(–)
		LA	48	M	W	(–)	11	B	A	(–)
Piraccini [12]	2006	LA	53	M	W	(–)	12	B	I	(–)
Rowan [8]	2006	LS	10	F	Maori	(–)	9.8	CT	Pa	Asthma, tension headaches
Yaşar [10]	2007	LS	62	F	W	Yes: her daughter	18	MRI	(–)	Seborrheic dermatitis
Yaşar [10]	2007	LA	45	F	W	No	14	MRI	(–)	None
		LA	49	M	W	No	11	MRI	(–)	None
Mansur [15]	2007	LS	46	F	(–)	No	13	MRI	(–)	Nevus lipomatosus superficialis
Moravvej-Farshi [cited in 7]	2007	LA	45	F	Ar	No	10.7	US	A	Freckles and café au lait spots on trunk
Scott [18]	2007	LA	83	F	B	No	16.5	N	P/I	Arthritis, ANA +
El Darouti [5]	2007	LS	36	F	Ar	(–)	5.1	US	P/H	Used tight head covers
		LS	17	F	Ar	(–)	4.8	US	P	Used tight head covers
		LS	21	F	Ar	(–)	3.1	US	P	Used tight head covers
		LS	39	F	Ar	(–)	2.5	US	P	Used tight head covers
		LS	11	F	Ar	(–)	4.5	US	P	Used tight head covers
		LS	35	F	Ar	(–)	2.2	US	P	Used tight head covers
		LS	40	F	Ar	(–)	4.6	US	P	Used tight head covers
		LA	50	F	Ar	(–)	8.5	US		Used tight head covers
		LA	30	F	Ar	(–)	4.6	US		Used tight head covers
		LA	40	F	Ar	(–)	4.6	US		Used tight head covers

LA, lipedematous alopecia; LS, lipedematous scalp; B, black; W, white; As, Asiatic; Ar, Arabic; La, Latin American; (–), not mentioned; N, needle; MRI, magnetic resonance imaging; US, ultrasound; CT, computed tomography scan; Rx, X-ray standard; B, biopsy; P, pain; Pa, paresthesias; I, itching; H, headache; A, asymptomatic; ANA, antinuclear antibodies; HTA, hypertension.

Table 3. Reported cases of lipedematous scalp and lipedematous alopecia (continued)

First author [ref.]	Year	Diagnosis	Age, years	Gender	Ethnicity	Family history	Thickness, mm	Measurement	Clinical	Associated disorders
Kavak [cited in 7]	2008	LS	50	F	(-)	(-)	10.8	US	A	HTA
Yip [11]	2008	LA	67	F	W	(-)	(-)	US	A	(-)
González-Guerra [19]	2008	LA	52	F	B	No	15	CT	I	Ovarian cysts
Fernandez-Torres [cited in 7]	2008	LS	55	F	W	(-)	22	MRI	I	Adenomyoma, Hashimoto's thyroiditis, hyalinizing cell carcinoma of salivary gland
Fernandez Mego [cited in 7]	2008	LS	47	F	La	Yes: father/sister	13	CT	I	(-)
Martínez-Moran [cited in 7]	2008	LS	77	F	W	(-)	15.2	CT	Pa/I/P	HTA
Wylie [cited in 7]	2009	LS	56	F	W	(-)	12	MRI	(-)	(-)
da Cunha Filho [cited in 7]	2010	LA	13	F	W	No	9.1	US	I	(-)
Ko [20]	2011	LA	18	F	As	No	16.9	MRI	(-)	(-)
Bosschaert [21]	2011	LS	16	F	W	No	8	MRI	A	(-)
Yasar [cited in 7]	2011	23 LS 8 LA	Mean 43 (±14)	F: 25 M: 6		2 family history + 29 no	9–18	MRI	Pa/P: 5 H: 3	1 pulmonary hyaline granulomatosis 1 acromegaly; 2 Cushing's disease; 1 migraine; 1 psoriasis vulgaris; 1 pachydermoperiostosis; 2 lipoma 22 no accompanying systemic disease
Zeng [22]	2011	LS		M	As	(-)	(-)	(-)		Heterochromia of the scalp hair
Müller [3]	2012	LS	15	M	Ar	(-)	(-)	(-)	A	None
Fuentelsaz-del Barrio [23]	2012	LA	49	F	W	(-)	31.2	CT	P	Scalp psoriasis, breast cancer
Peter [24]	2014	LS LS	57 40	F F	(-) (-)	(-) (-)	16.2 22.4	Rx CT	I I	None None
Lee [9]	2015	LA LA	6 10	F F	(-) (-)	(-) (-)	(-) (-)	(-) (-)	(-)	Nevus flammeus left flank Café au lait spot on back
Cabrera [13]	2015	LA	51	M	(-)	(-)	(-)	(-)	I	(-)
Keum [25]	2015	LS	40	F	(-)	(-)	15–20	MRI	H	Overweight, congenital mental retardation
Carrasco-Zuber [26]	2016	LS	48	F	La	(-)	10	US	Pa/P	HTA
Wang [27]	2016	LA	20	M	As	No	15	CT	I	None
Gonul [28]	2017	LA	31	F	W	(-)	12.5	US	(-)	ANA +
Chaplain [29]	2017	LS	36	F	B	No	12.5	MRI	P/H/I	None
Our case	2018	LA	66	F	B	Yes: her daughter	12	MRI	H	HTA/ANA +

LA, lipedematous alopecia; LS, lipedematous scalp; B, black; W, white; As, Asiatic; Ar, Arabic; La, Latin American; (-), not mentioned; N, needle; MRI, magnetic resonance imaging; US, ultrasound; CT, computed tomography scan; Rx, X-ray standard; B, biopsy; P, pain; Pa, paresthesias; I, itching; H, headache; A, asymptomatic; ANA, antinuclear antibodies; HTA, hypertension.