CASE REPORT

Lipedematous alopecia: Clinicopathological correlation.

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ABSTRACT

Lipedematous alopecia (LA) is a rare disorder within the spectrum of lipomatous diseases of the scalp, with approximately 115 cases reported in the literature. We present the case of a 46-year-old woman with bilateral diffuse non-scarring parietotemporal alopecia associated with a spongy scalp consistency and a positive pitting sign. Histopathological examination demonstrated mature adipocyte hyperplasia extending into the deep dermis, lymphatic ectasia, and preservation of hair follicles. This case highlights the importance of systematic scalp palpation and multimodal clinicopathological correlation in establishing the differential diagnosis.[1-3]

INTRODUCTION

Clinical History

A 46-year-old mestizo woman from Quito, Ecuador, presented for evaluation. Her past medical history was significant for subclinical hypothyroidism, treated with levothyroxine 50 µg/day for the past three years, and mixed dyslipidemia, well controlled with atorvastatin 20 mg/day. Her gynecologic and obstetric history included gravida 2, para 2, with regular menstrual cycles. Family history was notable for androgenetic alopecia in her mother, with no history of systemic lymphedema, lipedema, or autoimmune disease.

She presented with a 2-year history of progressive hair loss, accompanied by a sensation of diffuse scalp swelling and bilateral parietotemporal dysesthesia.

Dermatological Examination

Diffuse bilateral non-scarring alopecia involving the parietotemporal scalp was observed. On palpation, the scalp exhibited a soft, spongy consistency, with a positive pitting sign, characterized by persistent indentation following digital pressure. No erythema, scaling, or regional lymphadenopathy was identified. The hair pull test was negative. Hair density was preserved along the frontal hairline and occipital scalp.

Trichoscopy

Trichoscopy revealed anisotrichosis with numerous vellus hairs. No yellow dots, black dots, exclamation mark hairs, or cadaverized hairs were identified. Follicular ostia were preserved.

Laboratory Investigations

Complete blood count and routine serum chemistry were within normal limits. Thyroid-stimulating hormone (TSH) was 4.2 mIU/L, and free thyroxine (fT4) was 1.1 ng/dL. Antinuclear antibodies (ANA) were negative. Serum ferritin was 38 ng/mL, and vitamin D was 22 ng/mL, consistent with mild insufficiency. The Venereal Disease Research Laboratory (VDRL) test was nonreactive.



Figure 1. (A–B) Diffuse bilateral parietotemporal alopecia.
Reduced hair density is observed over the parietal regions, with preservation of the frontal hairline.

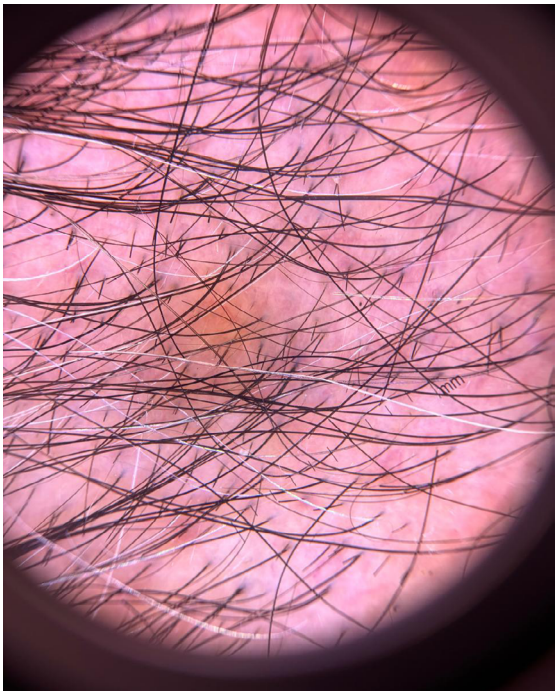


Figure 2. Trichoscopy showing anisotrichosis with numerous vellus hairs. No trichoscopic features suggestive of alopecia areata or scarring alopecia are observed.

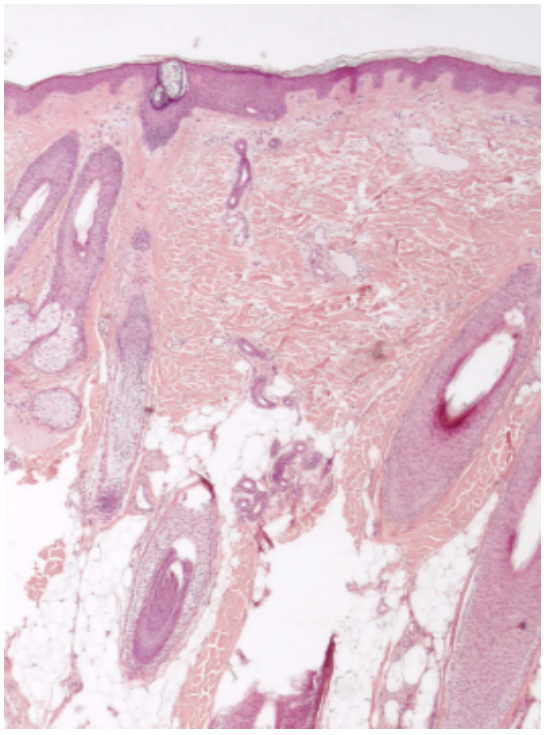


Figure 3. Hematoxylin and eosin staining demonstrating thickening of the subcutaneous tissue with mature adipocytes extending into the deep dermis. Hair follicles are preserved, without peribulbar inflammation.

Imaging Studies

Neither magnetic resonance imaging (MRI) nor high-frequency ultrasonography was performed. Their omission was justified by the concordance among the clinical, trichoscopic, and histopathological findings, which was considered sufficient to establish the diagnosis. Furthermore, imaging was not expected to influence therapeutic decision-making. These modalities are best reserved as complementary diagnostic tools when uncertainty persists or when quantitative assessment of subcutaneous tissue thickening is required.[2,4]

Histopathological Findings

Histopathological examination demonstrated mature, non-neoplastic adipocyte hyperplasia extending into the deep dermis. Hair follicles were preserved. Mild dermal edema and lymphatic ectasia were observed, accompanied by a sparse superficial perivascular lymphocytic infiltrate. There was no evidence of panniculitis, lipoma, dermal mucin deposition, or perifollicular fibrosis. These findings were consistent with lipedematous alopecia.

Diagnosis and Treatment

The patient was diagnosed with lipedematous alopecia. The differential diagnosis included androgenetic alopecia, diffuse alopecia areata, cutis verticis gyrata, follicular mucinosis, and myxedema, all of which were excluded based on the clinicopathological findings.[1,3,5,6]

Treatment consisted of clobetasol propionate 0.05% scalp solution applied nightly, 5 days per week for 4 weeks, followed by gradual tapering, together with cholecalciferol 4,000 IU daily. Mycophenolate mofetil (500 mg twice daily) was considered as a potential systemic therapeutic option.[7] Follow-up included trichoscopic evaluation every three months.

DISCUSSION

Lipedematous scalp (LS) and lipedematous alopecia (LA) are currently regarded as two manifestations within the same spectrum of lipomatous scalp disorders, with approximately 115 cases reported worldwide to date.[1] The term lipedematous alopecia was introduced by Coskey in 1961 to describe scalp thickening associated with impaired hair growth beyond 2 cm, whereas the non-alopecic variant (lipedematous scalp) had previously been described by Cornbleet in 1935. The most recent narrative review reported a marked female predominance (approximately 83%) and a mean age at presentation of 42.1 years,[1] findings consistent with our 46-year-old patient. The largest published case series, including 31 patients, likewise confirmed the female predominance and the broad clinical variability of this disease spectrum.[8]

The pathogenesis remains poorly understood. Several mechanisms have been proposed, including leptin dysregulation affecting adipose tissue distribution, local lymphatic dilatation contributing to follicular damage, and hormonal influences, given the striking female predominance.[1,3,9] Familial occurrence in sisters has also been reported, providing additional evidence for a possible genetic predisposition.[10] Metabolic comorbidities, including obesity, hyperlipidemia, and diabetes mellitus, have been described incidentally; however, no causal association has yet been established.[1,3,9–11] Our patient had concomitant subclinical hypothyroidism and dyslipidemia. More recently, the coexistence of lipedematous scalp with lichen planopilaris has been reported, raising the possibility that subcutaneous edema may represent a reactive process rather than an isolated disease entity.[12]

Clinically, the most distinctive feature is a soft, spongy scalp with compressibility or a positive pitting sign, as observed in our patient, emphasizing the importance of systematic scalp palpation during dermatologic examination.[1,9,11,13] Although the vertex and occipital scalp are the most commonly affected sites, our patient exhibited predominant bilateral parietotemporal involvement. Trichoscopy may reveal anisotrichosis and numerous vellus hairs; linear telangiectasias and, in isolated cases, tufted hair follicles have also been described.[2,9,13,14] Nevertheless, these findings are nonspecific, making clinicopathological correlation—and, when diagnostic uncertainty persists, MRI or high-frequency ultrasonography—essential for accurate diagnosis.[2,4,13]

The hallmark histopathological finding is subcutaneous tissue thickening to approximately twice its normal thickness, caused by hyperplasia of mature, non-neoplastic adipocytes in the absence of underlying panniculitis.[1,3,15] In lipedematous alopecia, replacement of hair follicles by fibrous tracts without significant inflammation, together with dermal edema and lymphatic ectasia, may also be observed.[3,11] Our case demonstrated these classic features, including adipocyte expansion into the deep dermis with preservation of follicular architecture, thereby confirming the diagnosis. Importantly, coexisting treatable alopecias, particularly alopecia areata arising on a background of lipedematous scalp, should be excluded because both conditions may coexist and the latter is potentially reversible.[5,6]

Management remains challenging because no standardized treatment guidelines or controlled clinical trials are currently available.[1,3,12] Reported therapeutic approaches are limited to case reports and small case series, including high-potency topical corticosteroids, intralesional triamcinolone, and systemic mycophenolate mofetil, the latter having shown partial hair regrowth in isolated reports.[1,7,9] Early recognition through careful scalp palpation and multimodal assessment—including

clinical examination, trichoscopy, histopathology, and imaging when indicated—is essential to distinguish this condition from disorders such as cutis verticis gyrata, follicular mucinosis, and myxedema.[3,9,11] Our case adds to the limited body of evidence and underscores the importance of maintaining a high index of suspicion in patients presenting with non-scarring alopecia associated with an abnormally soft scalp.

CONCLUSIONS

Lipedematous alopecia should be considered in patients presenting with non-scarring alopecia associated with a soft, spongy scalp and a positive pitting sign. Systematic scalp palpation, together with clinicopathological and trichoscopic correlation, is essential for establishing the diagnosis and excluding other causes of alopecia. Imaging studies represent a valuable adjunct in diagnostically challenging cases but are not invariably required when clinicopathological findings are conclusive. Early recognition of this rare entity may prevent misdiagnosis and unnecessary treatment.

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