Lipedematous Alopecia: Spongy Scalp Syndrome

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Lipedematous scalp and lipedematous alopecia are rare and closely related entities of unknown etiology. Almost all cases have occurred in black women. We report a case of lipedematous alopecia that is characterized by a boggy spongy thickening of the scalp, an increase in subcutaneous fat, short hairs, patchy alopecia, scarring, atrophy, and depigmentation. The histologic finding of an increase in subcutaneous adipose tissue is notable.


Case Report
An 83-year-old black woman noticed a gradual loss of scalp hair, beginning on the scalp vertex but eventually involving all areas of the scalp by one year. She complained of sensitivity and pruritus of her scalp and denied any other symptoms. There was no history of scalp trauma or hair pulling; no applied medications; and no use of physical devices, hot irons, cornrows, other braiding, or hair relaxers. Her medical history was positive for slight arthritis. There was no family history of alopecia or thyroid disease. Results of a physical examination revealed her entire scalp had short hairs (<2 cm) interspersed with areas of alopecia, scarring, and depigmentation (Figure 1). Other than the shortness, there were no morphologic abnormalities of the hairs. On palpation, there was a dramatic thick spongy feel to the scalp that was most prominent in the frontal area and vertex (Figure 2). There was no visible evidence of scalp inflammation. An ultrathin, firm, metal probe measured a scalp thickness of 16.5 mm (average, 5.5 mm). No abnormalities of the toenails and fingernails were present.

The only notable laboratory finding in the peripheral blood was an elevated antinuclear antibody titer of 1:640 (reference, 0). The results of complete blood count, general chemistry, thyroid profile, and urinalysis tests were otherwise unremarkable. The patient was not available for follow-up studies.

The histopathologic findings on excisional biopsy of a scalp specimen included an increase of subcutaneous adipose tissue, which was substantial (Figure 3). Abnormalities in the epidermis and follicular structures were not notable. There were some dilated blood vessels and focal lymphangiectatic vessels (Figure 4).

Treatment with various topical corticosteroid applications and oral antihistamines provided only temporary relief of the patient’s symptoms.

Comment
Lipedematous alopecia cannot be diagnosed on clinical observation alone; palpation is necessary. This report documents an additional case of this rare disorder and also emphasizes that the scalp should be palpated in all cases of alopecia, especially in black women.

In 1935, Cornbleet reported an unusual thickening of the scalp due to an increase in subcutaneous fat, which is now classified as lipedematous scalp. In 1961, Coskey et al reported 2 cases as lipedematous alopecia because they exhibited not only abnormal thickening of the scalp but also partial alopecia. The etiology of the increase in subcutaneous scalp fat is unknown. The precise mechanism of alopecia is unclear, but it is hypothesized that the increase in fat causes alopecia because of the increased pressure on the hair follicle and disruption of the normal growth cycle of the hair. Perhaps lipedematous scalp and lipedematous alopecia are variations or various stages of a single scalp entity, or perhaps they are distinct but closely related entities. We believe the latter. However, the issue would be settled if follow-up reports on previously recorded cases of lipedematous scalp disclosed that the disorder eventually progressed to lipedematous alopecia.
Few cases of either condition have been reported. We do not believe these disorders are as rare as the paucity of cases reported in the literature would indicate. Unless the scalp is palpated with moderate pressure, these conditions will go undetected. Therefore, in all cases of alopecia, the scalp should be palpated, especially when black women are involved. Of approximately 14 cases of lipedematous alopecia that have been reported, 10 cases have similar distinct findings (Table). From a clinician’s standpoint, the term spongy scalp syndrome is an appropriate and descriptive synonym.

In addition to those 10 cases, we present a similar case. The disorder occurs almost exclusively in black women, a fact that raises the possibility that lipedematous alopecia results from a cultural procedure associated with hair care, such as hot irons, cornrows, and hair relaxers, which we have not found to be the case. There is no abnormality of the hair shaft other than being shortened.

Specific clinical findings generally are present. When moderate pressure is applied to the involved areas of the scalp, it readily depresses and feels soft, boggy, thickened, and spongy, a reaction and feel that is similar to pressing the skin on an obese person’s shoulders or buttocks. The depression slowly reverses when the pressure is relieved, similar to the physical reaction of a sponge. The sponginess is not visually apparent and is detectable only on palpation. A patchy network of depigmented, often cobblestonelike, small areas of hairless scarring and atrophy is interspersed among other patches of short hair (<1 cm). Ridging or furrowing is not present. The disorder is slowly but steadily progressive, and there is no consistent satisfactory therapy. Coskey et al reported that oral prednisone resulted in increased hair length and decreased scalp thickness following 4 months of therapy. All regions of the scalp may be involved, but the disorder primarily affects the frontal area and vertex, at least initially. Black women predominately are affected; the average age of the women in the cases we cited was 56.6 years, with one 18-year-old woman and one 28-year-old woman.

High and Hoang suggested that lipedematous alopecia may represent a sign of discoid lupus erythematosus or a nonspecific pattern due to long-term...
Figure 3. Prominent subcutaneous adipose tissue advancing into the reticular dermis of the scalp (H&E, original magnification ×1). Photograph courtesy of Clay J. Cockerell, MD, and Carlos A. Cerruto, MD, both from Cockerell and Associates, Dallas, Texas.

Figure 4. Dilated blood vessels and focal lymphangiectatic vessels within the subcutaneous fat and dermis (H&E, original magnification ×10). Photograph courtesy of Clay J. Cockerell, MD, and Carlos A. Cerruto, MD, both from Cockerell and Associates, Dallas, Texas.

Injury. No history of trauma could be elicited in our case, but an elevated antinuclear antibody titer of 1:640 was present. Of the 10 other cases cited in this report, only one case report noted the results of an antinuclear antibody test, which was a titer of 1:20.6 The average scalp thickness in the 10 reported cases was 12.36 mm; scalps were measured using various techniques. The vertex scalp thickness in our case, as measured by an ultrathin, firm, sterile, metal probe, was 16.5 mm. Although the thickness of the scalp varies in different locations, it averages 5.5 mm.11,12

Lipematous alopecia (spongy scalp syndrome) is a rare scalp disorder primarily affecting black females. The diagnosis will be overlooked unless the clinician palpates the scalp in all patients with alopecia.

Acknowledgments—We are indebted to Clay J. Cockerell, MD, and Carlos A. Cerruto, MD, both from Cockerell and Associates, Dallas, Texas, for their report on the histology and for the photomicrographs.

Summary of Similar Cases of Lipematous Alopecia*

<table>
<thead>
<tr>
<th>Case (Year)</th>
<th>Age, y/Sex</th>
<th>Race</th>
<th>Duration</th>
<th>Clinical Presentation</th>
<th>Distribution</th>
<th>Scalp Thickness, mm</th>
</tr>
</thead>
<tbody>
<tr>
<td>Coskey et al2 (1961)</td>
<td>28/F</td>
<td>B</td>
<td>2 y</td>
<td>Shortened hairs</td>
<td>Diffuse</td>
<td>15.0</td>
</tr>
<tr>
<td></td>
<td>75/F</td>
<td>B</td>
<td>1 y</td>
<td>Shortened hairs</td>
<td>Diffuse</td>
<td>10.0</td>
</tr>
<tr>
<td>Curtis and Heising3 (1964)</td>
<td>62/F</td>
<td>B</td>
<td>15 y</td>
<td>Alopecia Shortened hairs</td>
<td>Diffuse</td>
<td>15.0</td>
</tr>
<tr>
<td>Kane et al4 (1998)</td>
<td>49/F</td>
<td>B</td>
<td>4 mo</td>
<td>Alopecia</td>
<td>Diffuse</td>
<td>12.6</td>
</tr>
</tbody>
</table>

TABLE CONTINUED ON PAGE 324
### Lipedematous Alopecia

#### Table. (continued)

<table>
<thead>
<tr>
<th>Case (Year)</th>
<th>Age, y/ (Sex)</th>
<th>Race</th>
<th>Duration</th>
<th>Clinical Presentation</th>
<th>Distribution</th>
<th>Scalp Thickness, mm</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bridges et al(^5) (2000)</td>
<td>48/F</td>
<td>B</td>
<td>6 y</td>
<td>Alopecia</td>
<td>Diffuse</td>
<td>10.0</td>
</tr>
<tr>
<td>Ikejima et al(^6) (2000)</td>
<td>30/M</td>
<td>A</td>
<td>7 y</td>
<td>Shortened hairs</td>
<td>Vertex</td>
<td>16.0</td>
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<tr>
<td>Fair et al(^7) (2000)</td>
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<td>B</td>
<td>6 mo</td>
<td>Alopecia</td>
<td>Parietal/Vertex</td>
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<tr>
<td>Tiscornia et al(^8) (2002)</td>
<td>69/F</td>
<td>W</td>
<td>6 mo</td>
<td>Alopecia</td>
<td>Temporal/Occipital</td>
<td>10.0</td>
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<tr>
<td>Martin et al(^9) (2005)</td>
<td>77/F</td>
<td>W</td>
<td>1 y</td>
<td>Alopecia</td>
<td>Frontal/Vertex</td>
<td>11.0</td>
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<tr>
<td>High and Hoang(^10) (2005)</td>
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<td>B</td>
<td>10 y</td>
<td>Alopecia</td>
<td>Vertex</td>
<td>15.0</td>
</tr>
</tbody>
</table>

*F indicates female; B, black; M, male; A, Asian; W, white.

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**REFERENCES**