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CASE REPORT

Lipedematous Alopecia in an Asian Woman: Is It an Advanced Stage of Lipedematous Scalp?

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Lipedematous alopecia (LA) is a rare disease entity that manifests as increased thickness of subcutaneous fatty tissue in the scalp with a variable degree of acquired, non-scarring alopecia. Although the pathogenesis of alopecia in LA is not clear, it is assumed that the increased thickness of subcutaneous fat retards hair growth by disturbing the peripheral microenvironment of the hair bulb. LA is clinically distinguishable from lipedematous scalp (LS) in that LS is not associated with any hair abnormalities, while LA is characterized by variable degree of hair loss. However, both LA and LS share increased scalp thickness. Here, we describe a rare case of LA on the frontal scalp of an Asian woman, further characterize the clinical and histologic features, and suggest applying an integrated diagnosis as lipedematous diseases of the scalp.
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-Keywords-
Alopecia, Lipedematous alopecia, Lipedematous scalp, Lipomatosis

INTRODUCTION

Lipedematous alopecia (LA) is a rare disease entity that manifests as increased thickness of subcutaneous fatty tissue in the scalp with a variable degree of acquired, non-scarring alopecia[1]. Coskey et al.[2] first introduced the term "lipedematous alopecia" in 1961 to describe cases with a thickened subcutaneous layer of the scalp along with an inability to grow hairs longer than 2 cm. Since then, only 16 additional cases of this rare condition have been described, including our case. The disease is known to affect most commonly the vertex and occiput areas of adult African American females[3]. LA is clinically distinguishable from lipedematous scalp (LS) in that LS is not associated with any hair abnormalities, while LA is characterized by variable degree of hair loss[4]. However, both LA and LS share increased scalp thickness. The underlying pathogenesis of LA remains elusive, but some authors have reported a possible association between LS and hyperlipidemia[5]. Here, we describe a rare case of LA on the frontal scalp of an Asian woman, further characterize the clinical and histologic features, and suggest applying an integrated diagnosis as lipedematous diseases of the scalp.

CASE REPORT

A 52-year-old woman presented to our clinic with a 2-month history of a 1×1 cm sized pink, shiny, dome-shaped nodule on her frontal scalp (Fig. 1A, Patient's consent form about publishing all photographic materials received). She did not experience any subjective symptoms such as pruritus or pain. She reported no history of trauma or topical treatment and no relevant family history. Her medical history included myotonia and spinal stenosis for 11 years, and hyperlipidemia and diabetes for 2 years. She was taking the medications for hyperlipidemia and

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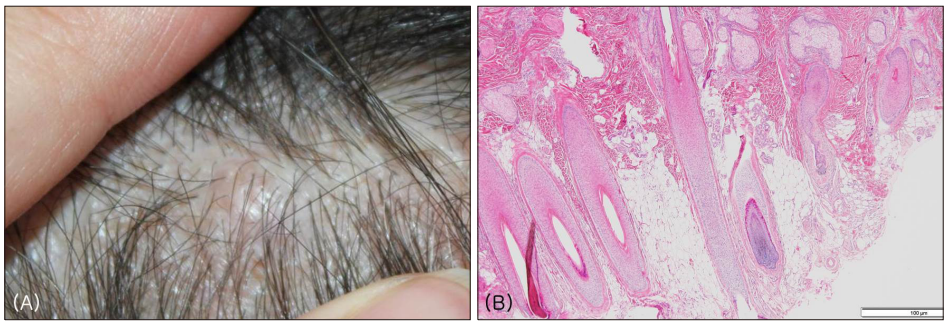

Fig. 1. (A) Clinical appearance of a pink, dome-shaped nodule with thinning of hairs. (B) Histopathologic findings. Striking hyperplasia of subcutaneous fat tissue composed of mature adipocytes. No evidence of encapsulation. Mild decrease in number of hair follicles (H&E, ×40).

diabetes.

Physical examination revealed sparse hair density and diffuse hair thinning. The remaining hairs in the involved area were thin, soft, and fragile. On palpation, the lesion was boggy and spongy. A hair pull test at several locations around the nodule was negative. Laboratory test results including a complete blood cell count, blood chemistry panel, and liver function tests were within normal ranges, while her glucose level was high at 198 mg/dl (reference range 74～107 mg/dl), her glycosylated hemoglobin was 6.2% (4.3～6.1%), her uric acid level was 6.1 mg/dl (3.2～7.0 mg/dl), and her triglyceride level was 268 mg/dl (28～199 mg/dl). High-density lipoprotein-cholesterol was 55 mg/dl (40～60 mg/dl) while low-density lipoprotein-cholesterol was checked as 89 mg/dl (66～159 mg/dl).

We excised the mass as we suspected pilomatricoma or epidermal cyst. However, the gross specimen showed no circumscribed masses, only diffuse thickness of the skin. Histopathologic findings revealed a normal epidermis and dermis with striking hyperplasia of the subcutaneous fat (Fig. 1B). Hair follicles in the anagen phase were distributed without peripheral inflammation. The hyperplastic subcutaneous fat tissue consisted of mature adipocytes with a normal size and shape, but was thickened up to the level of the sebaceous glands. No encapsulation or cystic structure was found. Clinical features and histopathologic findings were consistent with LA. Since the patient did not complain of any subjective symptoms and the lesion was completely excised, further diagnostic procedures were not performed.

DISCUSSION

Scalp thickness is clearly increased in both LA and LS patients. Garn et al.[6] measured the scalp thickness of 523 healthy adults at 5.8±0.12 mm on average. In contrast, González-Guerra et al.[1] reported the average scalp thickness of LA and LS patients to be 11.4 mm on average, which is about twice as thick as normal individuals. The thickening is mainly due to increased subcutaneous fat.

The nature of hyperplasia in subcutaneous fatty tissue remains unknown. Yip et al.[7] reported a potential role for leptin in the development of fatty hyperplasia. Leptin is known for its regulation of fat distribution and lipo-apoptosis through feedback mechanisms. Some authors suggested that inadequate lipid distribution could cause an expansion of the subcutaneous fat in LA[8]. Due to its predominance in the female population, hormonal influences are often discussed[9]. In addition, Bukhari et al.[5] presented a possible relationship between hyperlipidemia and LA. Metabolic diseases such as hyperuricemia, hyperlipidemia and diabetes mellitus are associated with multiple lipomatosis[10,11]. Our patient also had uncontrolled hyperlipidemia at the time of onset, yielding possible association with LA development. Therefore, we recommend having lipid profile test and checking medical history of hyperlipidemia in patients with LA or LS.

Reported cases of LA have shown variable degrees of hair abnormalities, ranging from almost normal hair follicle distribution to replacement of pre-existing follicles by fibrous tracts. Although the pathogenesis of alopecia in LA is not clear, it is assumed that the increased thickness of subcutaneous fat retards hair growth by disturbing the peripheral microenvironment of the hair bulb. Yaşar et al.[12] suggested that hair follicles surrounded by fat cells might have an inadequate blood supply, resulting in follicular atrophy or destruction. This hypothesis in combination with the clinical variety of hair loss suggests that alterations of the follicular microenvironment due to fatty hyperplasia can cause insidious destruction of the hair structure, not abrupt loss. The degree of alopecia may differ depending on the stage at which it is diagnosed or excised. In addition, LS and LA show similar distribution in ages of onset around the fourth decade and in affected areas of the vertex and occiput. Therefore, LS and LA may not be separate disease entities, but parts of the same disease spectrum.

In conclusion, we report a rare case of LA in an Asian woman. This rare, distinctive clinical feature of increased subcutaneous fat thickness with a variable degree of alo-

pecia should be on the differential diagnosis for scalp nodule. We suggest that LS and LA may coexist in a spectrum of lipomatous lesion of scalp with morphological variations. Further studies are necessary to elucidate the etiology and pathogenesis.

CONFLICTS OF INTEREST

The authors have nothing to disclose.

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