Lichen planopilaris with Koebner phenomenon

Leena Alahmari, MD,[a] Rawan Almesned, MD,[a] Ahmed Alhumidi, MD,[b] and Abdullah Alkhalifah, MD[c]
Riyadh, Saudi Arabia

Key words: Lichen planopilaris; scarring alopecia.

INTRODUCTION

Lichen planopilaris (LLP) is a rare, progressive disease of the scalp and is an important cause of scarring alopecia.[1] LPP is classified clinically into 3 types: classic LPP, frontal fibrosing alopecia, and Graham-Little-Piccardi-Lassueur syndrome.[2,3] In classic LPP, patients commonly present with single or multiple patches of alopecia in the vertex of the scalp.[4] The exact pathogenesis of LPP remains unclear.[5,6] Here we describe a case involving one of the youngest reported adult subjects in which the trigger for LPP is unique and was not previously reported. The patient admitted a prolonged history of placing an excessive traction on the temporal scalp as she repeatedly pulled her hair up in a unique fashion. This excessive strain resulted in what we think is traction-induced LPP, particularly developed over the site of tension. After the patient stopped applying the traction and successful treatment with topical, intralesional, and oral corticosteroids, the patient experienced complete remission, so all medications were stopped. To our knowledge, this is the first report with history of positive traction being the eliciting factor of LPP.

Abbreviations used:

LLP: lichen planopilaris
CCCA: central centrifugal cicatricial alopecia

CASE REPORT

A 25-year-old Saudi otherwise healthy woman presented to our clinic with a 5-month history of bilateral focal temporal hair loss with concomitant pain, redness, itching, and burning sensation of the scalp. She reported a prolonged history of applying traction to the areas of hair loss. She denied using any topical or oral medication before the onset of hair loss. Her family history is positive for alopecia areata but with unremarkable family history of other hair or autoimmune disease. Upon examination, her scalp showed symmetrical bitemporal irregular patches of scarring alopecia with remarkable perifollicular erythema and scaling (Fig 1, *A* and *B*). There was no hair loss of the eyebrow, axilla, or groin area. Mucocutaneous and nails changes associated with lichen planus were not present. Her laboratory studies, including complete blood count, serum ferritin level, thyroid function test, and liver and renal function tests were all within normal ranges. Erythrocyte sedimentation rate was not elevated. A biopsy specimen of the scalp showed decreased hair follicle density with perifollicular fibrosis and lymphatic infiltrate of the isthmus part of the hair follicle consistent with LPP (Fig 2, *A* and *B*).

The patient was advised to avoid the traction hair care practice to prevent potential worsening of hair loss. She was started on clobetasol solution in combination with triamcinolone Acetonide injections (10 mg/mL) administered monthly, but her symptoms of pain, redness, and itching persisted. Systemic treatment with prednisolone was initiated (20 mg/d). At follow-up, the condition was fairly controlled, as further hair loss was halted with resolution of her symptoms; thus, prednisolone dose was tapered gradually to 5 mg/d. Other systemic medications that were prescribed include hydroxychloroquine (200 mg twice a day); however, the patient was not adherent so it was discontinued. A trial of tetracycline (500 mg twice a day) was attempted, but the patient was noncompliant because of the gastrointestinal side effects. The patient then experienced a complete remission, as

From the Department of Pathology,[b] College of Medicine,[a] King Saud University and Department of Dermatology, Prince Sultan Military Medical City.[c]
Funding sources: None.
Conflicts of interest: None disclosed.
Correspondence to: Rawan Almesned. E-mail: rawanalmisned@gmail.com.

JAAD Case Reports 2018;4:848-50.
2352-5126

https://doi.org/10.1016/j.jdcr.2018.05.009

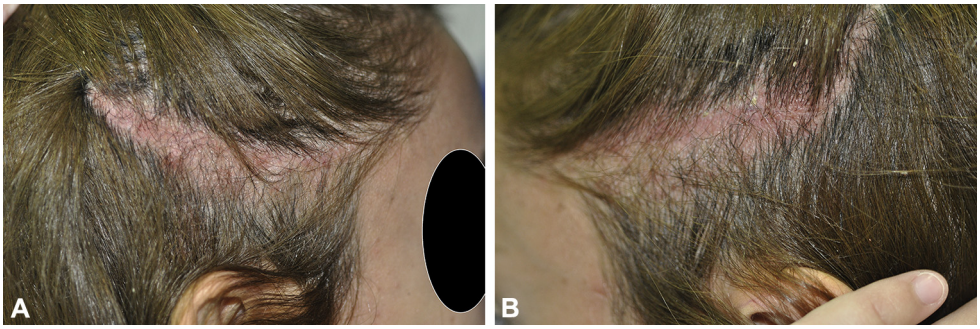

Fig 1. **A** and **B**, Bilateral linear patch of scarring alopecia following the line of hair traction.

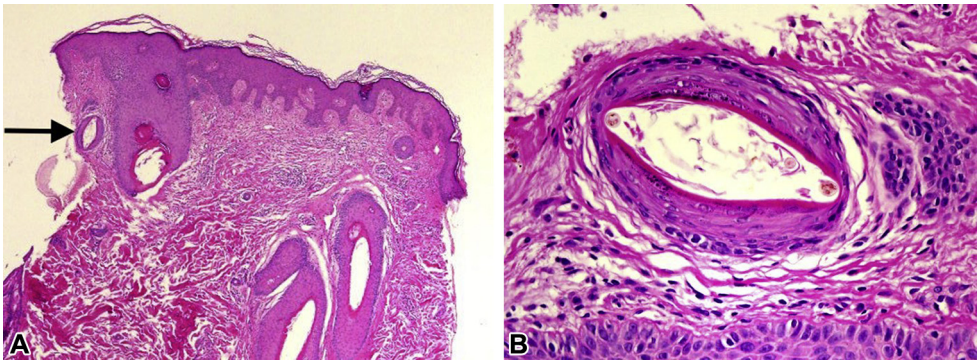

Fig 2. **A**, Low-power view shows a low follicular density. **B**, A higher magnification shows an altered follicle with focal perifollicular fibrosis with a few lymphocytes at the level of the infundibulum. (**A** and **B**, Hematoxylin-eosin stain.)

she became symptom free for around 3 months, so all medications were stopped.

DISCUSSION

We describe a case in which the trigger for LPP is unique and was not previously reported. Ostensibly, the patient had a prolonged history of placing excessive traction on the temporal scalp as she repeatedly pulled her hair up in a unique fashion. This excessive strain resulted in what we think is traction-induced LPP, particularly developed over the site of tension. To our knowledge, this is the first report with history of positive traction being the eliciting factor of LPP. A recent report shows that scalp trauma in one patient resulted in LPP developing only in the areas of trauma.[7] An important differential diagnosis is central centrifugal cicatricial alopecia (CCCA).[8] There are some similarities between LPP and CCCA, as they both are classified as lymphocytic types of primary cicatricial alopecias, so they both have a common end result: follicular scarring.[8] Treatment options are quite similar in both disease entities.[9,10] However, differences do exist around the epidemiologic and clinical presentation.[8,11] Although LPP can occur in individuals with any racial background, CCCA distinctively occurs in women with African descent.[8,12] Moreover, even though the etiology for both diseases remain debated, CCCA has been classically attributed to certain hair practices such as braiding, use of chemical and lye-containing relaxers, hot comb, and excessive heat application.[13] It is important to note that these mentioned hair care practices were not reported to be associated with LPP. In addition, the typical sites of scalp involvement in LPP include the frontal and parietal areas and the vertex, then spreading anywhere.[1,14] Conversely, in CCCA the area that is first to be affected is the central scalp, after which it starts expanding centrifugally.[8] Histopathologic features are overlapping between CCCA and LPP.[8,15] It is noteworthy that the isolated involvement of the temporal area—the site at which the hair has been under large amounts of strain in our patient—further supports that traction was the triggering factor of LPP. This case is reported to show that unusual triggers for LPP exist. Careful history, clinical assessment, and histopathologic evaluation can prevent the delay in diagnosis. Prompt initiation of treatment can improve the prognosis. Clinicians should be able to distinguish LPP from CCCA.

Further studies are needed to better understand precipitating factors and pathologic mechanisms behind LPP.

REFERENCES

1. Meinhard J, Stroux A, Lünnemann L, et al. Lichen planopilaris: epidemiology and prevalence of subtypes-a retrospective analysis in 104 patients. *J Dtsch Dermatol Ges [Internet]*. 2014;12(3):229-235. Available from: http://ovidsp.ovid.com/ovidweb.cgi?T=JS&PAGE=reference&D=emed11&NEWS=N&AN=2014167985＼nhttp://www.ncbi.nlm.nih.gov/pubmed/24533855.
2. Olsen EA, Bergfeld WF, Cotsarelis G, et al. Summary of North American Hair Research Society (NAHRS)-sponsored Workshop on cicatricial alopecia, Duke University Medical Center, February 10 and 11, 2001. *J Am Acad Dermatol*. 2003;48(1):103-110.
3. Baibergenova A, Donovan J. Lichen planopilaris: update on pathogenesis and treatment. *Skinmed [Internet]*. 2013;11(3):161-165. Available from: http://www.ncbi.nlm.nih.gov/pubmed/23930355.
4. Tan E, Martinka M, Ball N, Shapiro J. Primary cicatricial alopecias: clinicopathology of 112 cases. *J Am Acad Dermatol*. 2004;50:25-32.
5. Misiak-Galazka M, Olszewska M, Rudnicka L. Lichen planopilaris in three generations: grandmother, mother, and daughter - a genetic link? *Int J Dermatol [Internet]*. 2016;55(8):913-915. Available from: http://www.ncbi.nlm.nih.gov/pubmed/26694527.
6. Atanaskova Mesinkovska N, Brankov N, Piliang M, Kyei A, Bergfeld WF. Association of lichen planopilaris with thyroid disease: a retrospective case-control study. *J Am Acad Dermatol*. 2014;70(5):889-892.
7. Montpellier RA, Donovan JC. Scalp trauma: a risk factor for lichen planopilaris? *J Cutan Med Surg [Internet]*. 2014;18(3):214-216. Available from: http://www.ncbi.nlm.nih.gov/pubmed/24800713.
8. Sundberg JP, Hordinsky MK, Bergfeld W, et al. Cicatricial Alopecia Research Foundation meeting, May 2016: Progress towards the diagnosis, treatment and cure of primary cicatricial alopecias. *Exp Dermatol*. 2018;27(3):302-310.
9. Chiang C, Sah D, Cho BK, Ochoa BE, Price VH. Hydroxychloroquine and lichen planopilaris: efficacy and introduction of Lichen Planopilaris Activity Index scoring system. *J Am Acad Dermatol [Internet]*. 2010;62(3):387-392. Available from: https://doi.org/10.1016/j.jaad.2009.08.054.
10. Herskovitz I, Miteva M. Central centrifugal cicatricial alopecia: challenges and solutions. *Clin Cosmet Investig Dermatol*. 2016;9:175-181.
11. Filbrandt R, Rufaut N, Jones L, Sinclair R. Primary cicatricial alopecia: diagnosis and treatment. *CMAJ: Can Med Assoc J*. 2013;185(18):1579-1585.
12. Olsen EA, Callender V, McMichael A, et al. Central hair loss in African American women: incidence and potential risk factors. *J Am Acad Dermatol*. 2011;64(2):245-252.
13. Dlova NC, Salkey KS, Callender VD, McMichael AJ. Central centrifugal cicatricial alopecia: new insights and a call for action. *J Investig Dermatol Symp Proc*. 2017;18(2):S54-S56.
14. Cevasco NC, Bergfeld WF, Remzi BK, de Knott HR. A case-series of 29 patients with lichen planopilaris: The Cleveland Clinic Foundation experience on evaluation, diagnosis, and treatment. *J Am Acad Dermatol*. 2007;57(1):47-53.
15. Tandon YK, Somani N, Cevasco NC, Bergfeld WF. A histologic review of 27 patients with lichen planopilaris. *J Am Acad Dermatol*. 2008;59(1):91-98. Available from: http://www.ncbi.nlm.nih.gov/pubmed/24800713.