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Hair Regrowth in a Recent-Onset Scarring Alopecia Associated with Kikuchi-Fujimoto Disease

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Dear Editor:

Kikuchi-Fujimoto disease (KFD) is a rare autoimmune disease characterized by cervical lymphadenopathy and fever[1]. It is a self-limiting disease that is more common in Asian populations[1]. Typical cutaneous features include erythematous papules and plaques, and there has been one case of alopecia associated with KFD[2]. Scarring alopecia involves permanent injury to hair follicles and is associated with infection and autoimmune diseases such as lupus erythematous and polymyositis[3]. Since the lesions are refractory to various treatments, early recognition of the disease is helpful in stalling disease progression.

A 44-year-old female presented with an 8 cm pigmented hairless patch on the occiput area with multiple inflammatory nodules that developed 1 month ago (Fig. 1A, B). She experienced mild fever and neutropenia with swollen cervical lymph nodes 4 months ago and was diagnosed with KFD after a lymph node biopsy. Two weeks of oral steroid treatment alone improved tender lymph nodes, leaving a hairless patch. Blood tests were negative for antinuclear antibodies, thyroid dysfunction, and syphilis. Histological examination showed miniaturized hair follicles and a prominently dense infiltration of

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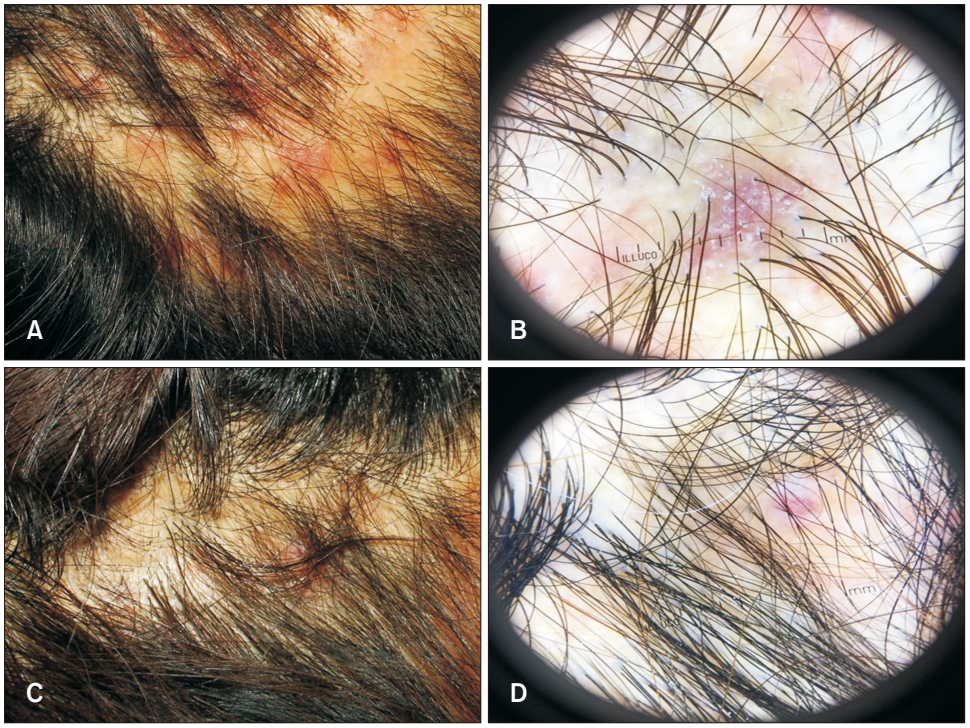

Fig. 1. (A) Clinical image of a hair loss patch on the occiput. (B) Dermoscopic view. (C) After 3 months, inflammatory nodules, erythema, and pigmentation improved with hair regrowth. (D) Dermoscopic view. We received the patient's consent form about publishing all photographic materials.

numerous inflammatory cells consisting of lymphocytes and histiocytes around the destructed hair follicle structures with fibrotic stroma (Fig. 2A~C). Immunohistochemistry showed that the infiltrated cells around the hair follicle structures were CD4-, CD8-, CD68-positive (Fig. 2D~F). Based on both clinical and histological findings, the patient was diagnosed with scarring alopecia associated with KFD. Treatment comprised doxycycline 200 mg daily for 3 months and corticosteroid injections at 2-week intervals. Inflammatory nodules and erythema improved, with slight hair growth within the center and margin of the lesion (Fig. 1C, D).

In the first reported case of scarring alopecia associated with KFD, histological examination of the bald patch revealed CD4, CD8, and CD68 positivity, similar to the current case[2]. The numbers of CD4- and CD8-positive cells vary depending on the disease stage, and positivity for CD68 (which is a marker of histiocytes and plasmacytoid monocytes) is helpful for the diagnosis of KFD[1].

Hair regrowth in scarring alopecia is not common, since the epithelial stem cells situated at the outer root sheath are permanently destroyed; however, some hair regrowth has been observed in cases of scarring alopecia associated with chronic cutaneous lupus erythematosus with a recent onset[3]. In addition, hair regrowth had been reported in scarring alopecia patients with discoid lupus erythematosus[4] and folliculitis decalvans[5]. The patient in this case showed hair regrowth and improvements in inflammatory nodules and pigmentation after transient administration of antibiotics and intralesional steroids.

Further studies are required to investigate the possible connection between KFD and scarring alopecia. This case emphasizes the importance of including KFD in the differential diagnosis of scarring alopecia, since KFD is relatively common in Asian populations and often underdiagnosed. Early recognition is critical to initiate hair growth in recent-onset alopecia and to prevent irreversible aggravation of hair loss. To the best of our knowledge, this was the first report of scarring alopecia with KFD showing clinical improvement of symptoms while also demonstrating the usefulness of specific immunohistochemical analyses to confirm the diagnosis.

CONFLICTS OF INTEREST

The authors have nothing to disclose.

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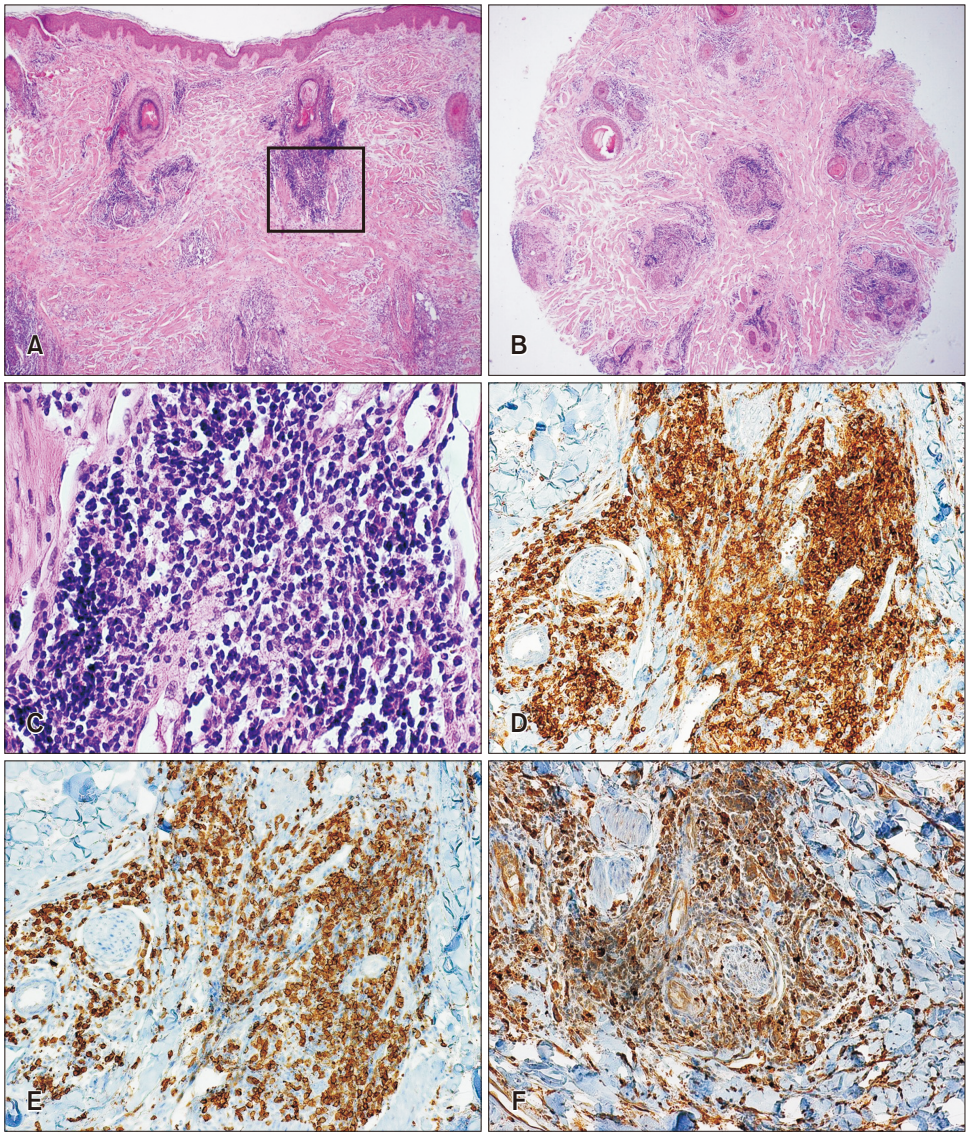

Fig. 2. Histologic findings of an alopecia lesion in a vertical section (H&E, ×40) (A) and transverse section (B) showed destructed hair follicles with a dense inflammatory cell infiltration and fibrotic stroma (H&E, ×40). (C) Higher magnification of the black box in (A) showed extensive nuclear debris and lymphohistiocytic infiltration, with no neutrophils observed around the destructed hair follicle structures (H&E, ×400). Immunohistochemistry showed CD4- (D), CD8- (E), and CD68- (F) positive cells infiltrating the hair follicle structures (×200).

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