



Letter to the Editor

Increased circulating Th17 cell in a patient with tinea capitis caused by *Microsporum canis*



Dear Editor,

Tinea capitis is a relatively common superficial fungal infection. In the infection of *Microsporum canis* (*M. canis*), the lesion is around a hair shaft and eventually the hairs break off 1–3 mm above the scalp. The patients sometimes experience permanent hair loss, especially after longstanding inflammation. However, the detail mechanism of severe inflammation leading to subcutaneous pustular formation and hair loss caused by tinea capitis remains unclear. Recently, IL-17-producing Th17 cells, which are comprised in established CD4⁺ T helper cell subsets, exacerbate skin inflammation,

leading to pustular formation in some disease condition, such as pustular psoriasis,¹ bacterial infection.² Herein, we report a possible role of Th17 cells in tinea capitis.

A 66-year-old woman developed erosive crusted plaques with pustules on her scalp 2 months before our initial examination. Despite treatment with systemic antibiotics and oral methylpredonisolone, the lesions gradually enlarged. She kept three cats in her home. On physical examination, pyodermic, ulcerative plaques covered with pustules and crusts were seen on her scalp (Fig. 1A). Bacterial culture evaluation was negative. Fungal culture revealed the presence of the *M. canis*. Laboratory examinations

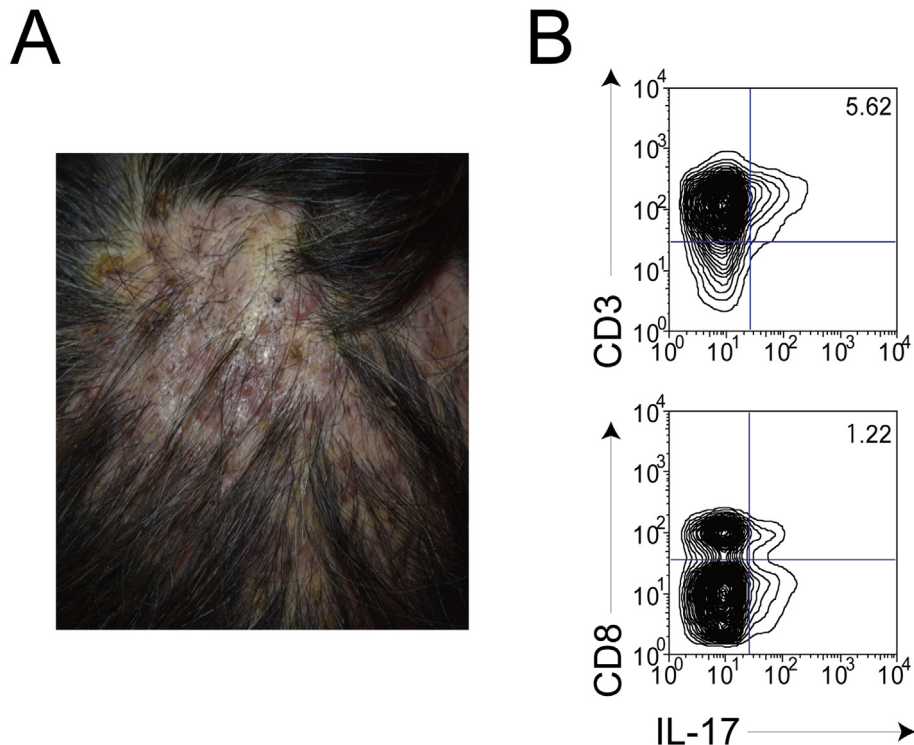


Fig. 1. Clinical manifestation and circulating Th17 cells. (A) Erosion and ulcers with pustules and crusts on the backhead. (B) Representative flow cytometric analysis of IL-17⁺ cells in peripheral blood lymphocytes.

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revealed neutrophilia (15,518/ μ l). We diagnosed the patient's eruption as tinea capitis caused by *M. canis*. She was treated with oral itraconazole 100 mg per daily without topical application of antifungal agent, which gradually improved her skin lesions and led to re-epithelization of the erosive skin 2 months after the treatment. To address the possible participation of Th17 cells in the pathogenesis of tinea capitis, we examined the frequency of Th17 cells in a peripheral blood mononuclear cells (PBMCs) in our patient by a method as previously described.³ The frequency of Th17 cells in our patient (4.4% of CD4⁺ IL-17⁺ cells in PBMC) was calculated as the difference between that of CD3⁺ IL-17⁺ cells (5.62% of CD3⁺ IL-17⁺ cells in PBMC) and CD8⁺ IL-17⁺ cells (1.22% of CD8⁺ IL-17⁺ cells in PBMC) (Fig. 1B). This rate was higher than that of a healthy subject (approximately 0.4%) (Data not shown).

Recently, it has been reported that Th17 cells play an important role in an infection protective mechanism against fungi. However, severe hair follicular inflammation sometime exacerbates hair loss in tinea capitis patients. We demonstrated a high frequency of circulating Th17 cell in a patient with tinea capitis caused by *M. canis*. Although the detail mechanism of severe skin inflammation with subcutaneous pustules and broken hair follicle remains unclear, we suggested that Th17 cells might have systemically activated neutrophils and exacerbate skin inflammation in tinea capitis through a Dectin-1 dependent manner, because *M. canis* activates inflammatory cells through Dectin-1 stimulation.⁴ Therefore, it is important for early detection and treatment for tinea capitis to regulate severe skin inflammation and hair loss. Further analysis is necessary to clarify the pathogenesis of tinea capitis.

Conflict of interest

The authors have no conflict of interest to declare.

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