



## Tinea Capitis Mimicking Cicatricial Plaque on the Scalp and Acne Keloidalis

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### Abstract:

Tinea capitis is a common infection caused by dermatophyte fungi in children and can sometimes occur in adults. The clinical features are variable due to the diverse immune responses produced by fungal infection<sup>1</sup>. Atypical presentations have been reported within which scarring alopecia due to tinea capitis has also been described<sup>2,3</sup>. We present a case of tinea capitis which manifests as cicatricial alopecia and acne keloidalis.

**Keywords:** dermatophyte fungi; cicatricial alopecia; acne keloidalis

### Introduction:

Tinea capitis is a common infection caused by dermatophyte fungi in children and can sometimes occur in adults. The clinical features are variable due to the diverse immune responses produced by fungal infection<sup>1</sup>. Atypical presentations have been reported within which scarring alopecia due to tinea capitis has also been described<sup>2,3</sup>. We present a case of tinea capitis which manifests as cicatricial alopecia and acne keloidalis.

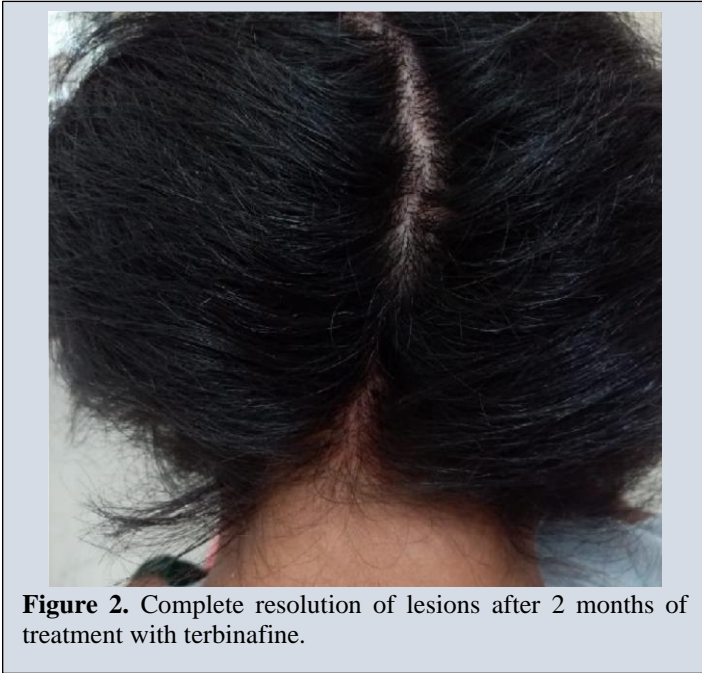
### Case report

A 32-year-old woman with a medical history of systemic lupus erythematosus presented for evaluation with a twelve-year history of itching and hair loss. Her medications included prednisone, hydroxychloroquine and enalapril. Physical examination revealed an erythematous cicatricial plaque with associated alopecia and diffuse dry scales on the scalp and firm confluent papules on the posterior neck (Figure 1).



**Figure 1.** Cutaneous finding of our patient. (A) Erythematous plaque with cicatricial aspect with associated alopecia and diffuse dry scales on the scalp. (B) Confluent papules on the posterior neck.

Trichoscopy showed clobbested cicatricial like papules with single hairs arising in some areas. Tufted hair were not observed. A scalp biopsy was performed. Histopathology revealed a mixed inflammatory infiltrate composed of lymphoplasma cells and fibrosis surrounded the hair shafts. Clusters of spores and hyphae within some hair shafts were found. Tissue culture isolated *Trichophyton tonsurans*. Therapy was started with oral terbinafine at a dose of 250mg per day for 2 months with complete resolution of lesions (Figure 2). After 6 months of follow-up, no recurrence was observed.



**Figure 2.** Complete resolution of lesions after 2 months of treatment with terbinafine.

## Discussion

*Tinea capitis* is a frequent dermatomycosis most commonly seen in children. Despite its frequency, TC can represent a diagnostic challenge due to the variation in clinical presentation. Some cases of TC due to *Trichophyton tonsurans* mimicking folliculitis, chronic furuncles, canalizing pyoderma and tufted hair folliculitis have been reported<sup>2,3</sup>. Likewise, a few cases of TC mimicking cicatricial alopecia have also been described. A case of a child with *tinea capitis* with apparent loss of follicular markings treated successfully with oral terbinafine was reported by Mirmirani<sup>4</sup>. In addition, Ferenczi described a woman with *acne keloidalis* caused by fungal infection with near-complete resolution after 2 months of treatment with griseofulvin<sup>5</sup>. In both cases, patients were diagnosed with long-standing *tinea capitis* due to *Trichophyton tonsurans* and showed good response to antifungal therapy. Clinical presentation of TC depends on the causative fungus and the immune status of the patient<sup>4</sup>. An immunohistochemical study found evidence that supports the involvement of both cell-mediated and humoral mechanisms in the chronic and non-inflammatory TC<sup>6</sup>. In a proposed model which could explain the long-standing non-inflammatory scalp infections in some patients, an immunocomplex formation between IgE and antigen produced by *T. tonsurans* would bind mast cells at the site of infection realising histamine. Histamine would activate H<sub>2</sub> receptors of T-suppressor

cells which could prevent T-helper cell proliferation and inhibit MIF expression resulting in immune anergy<sup>7</sup>. We believe patient treatment could additionally contribute to the immune anergy. The role of mast cells and histamine have been studied in the pathogenesis of primary cicatricial alopecia<sup>8</sup>. Furthermore, a study by Hoda found IL-1 $\beta$  and MIF cytokine network induce MMP-1 and contribute to the loss of interstitial dermal collagen<sup>9</sup>. We hypothesize histamine could also play a role in scarring alopecia due to *tinea capitis* and MIF expression could induce MMP which would lead collagen degradation explaining the excellent response to antifungal treatment, albeit further studies are needed to determine the exact pathway.

This case shows *tinea capitis* can mimic scarring alopecia and should be considered in the differential diagnosis of apparent cases of primary cicatricial alopecia.

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