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Case Report

Beyond Appearances: Foreign Body Reaction of Hair Follicles as a Complication of Inflammatory Tinea Capitis (Kerion Celsi) Resolved

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Abstract: ***Background***: Tinea capitis is a common pediatric dermatophytosis involving the scalp, hair shafts, and follicles, most frequently caused by Trichophyton, Microsporum, and Nannizzia species. Although most cases are non-inflammatory, a subset progresses to kerion celsi. While appropriate antifungal therapy typically leads to resolution, persistent inflammation may occur despite microbiological clearance, suggesting additional pathogenic mechanisms. We report a 7-year-old male presenting with a chronic pseudoalopecic plaque on the scalp following inflammatory tinea capitis, with negative mycological studies but histopathological evidence of granulomatous dermatitis. Dermoscopic-guided removal of keratin plugs and ingrown hairs resulted in clinical improvement. This case highlights a novel mechanism of persistent inflammation in kerion celsi associated with hair shaft fragmentation, aberrant follicular penetration, and retention of keratinous debris acting as endogenous foreign bodies, triggering a chronic granulomatous response.

Keywords: Ingrown Hair, Kerion, Tinea Capitis, Mycosis, Keratin Plugs, Granulomatous Dermatitis.

INTRODUCTION

Tinea capitis is a superficial mycosis that affects the hair, hair follicles, and surrounding skin, and is potentially caused by any dermatophyte genus (except Epidermophyton) and, to date, has been attributed to Trichophyton, Microsporum, and Nannizzia [1]. However, up to 65% of cases may present bacterial overcolonization by Staphylococcus aureus, 48% by Staphylococcus aureus and 18% by gram-negative bacteria [2]. In México, this dermatophytosis represents 4–10% of all cases of tinea, with the dry form (90%) predominating over the inflammatory form (10%) [3], particularly in the central and northern regions of the country. Kerion Celsi is a severe inflammatory variant that is frequent in the Yucatán Peninsula, which can cause complications such as scarring alopecia [1], or a foreign body reaction of hair follicles and keratin plugs, perpetuating the inflammatory process even after eradication of the fungal agent.

CASE DESCRIPTION

We report a 7-year-old male patient from Mérida, Yucatán, who presented with a dermatosis localized to the scalp in the vertex region, characterized by a pseudoalopecic plaque measuring 12 × 10 cm, irregular in shape, well defined, with multiple keratin plugs within dilated follicles at the center (Fig. 1). Dermoscopy revealed broken hairs, comma hairs, corkscrew hairs, and anisotrichosis (Fig. 2). The patient reported a 6-month history of evolution, associated with pain, and had been previously treated with multiple systemic antibiotics (quinolones and cephalosporins) for 2 months, with remission of the initial pustules but persistence of pseudoalopecia. He was referred to the Centro Dermatológico de Yucatán, where direct examination with Chlorazol Black revealed no fungal structures; a biopsy revealed granulomatous dermatitis with fibrosis, reactive epidermal hyperplasia, and a peripheral lymphocytic infiltrate, without identification of hyphae (Fig. 3). After excluding persistent parasitic infestation, keratin plugs and hair shafts were removed under dermoscopic guidance (Fig. 4), resulting in clinical improvement."

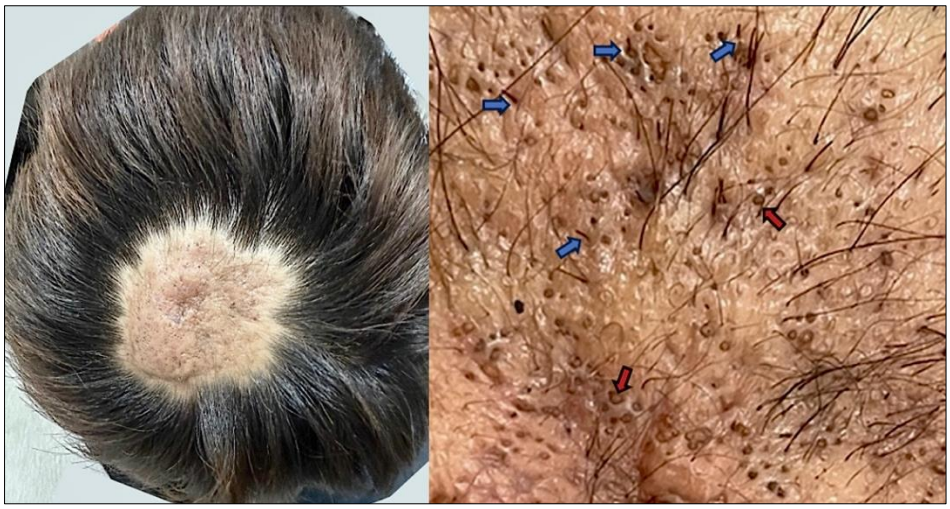

Fig. 1: Pseudoalopecic plaque measuring 12 cm in diameter with irregular shape, showing multiple keratin plugs (red arrows) and broken hairs (blue arrows) in the center.

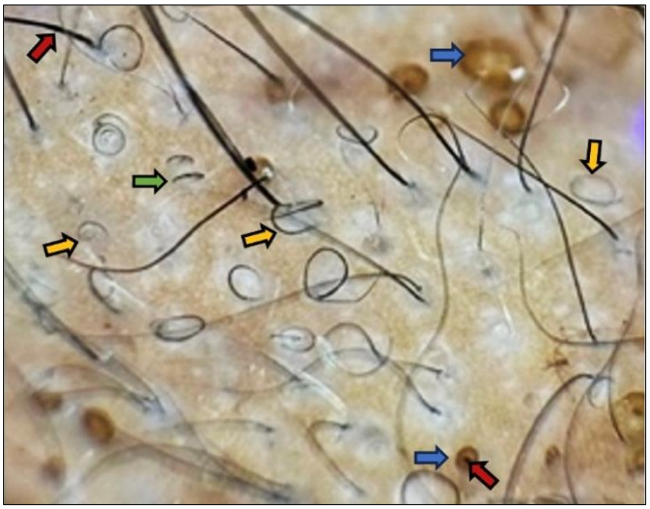

Fig. 2: Dermoscopy revealing anisotrichosis, broken hairs (red arrow), comma hairs (green arrow), corkscrew hairs (yellow arrow), and keratin plugs (blue arrow).

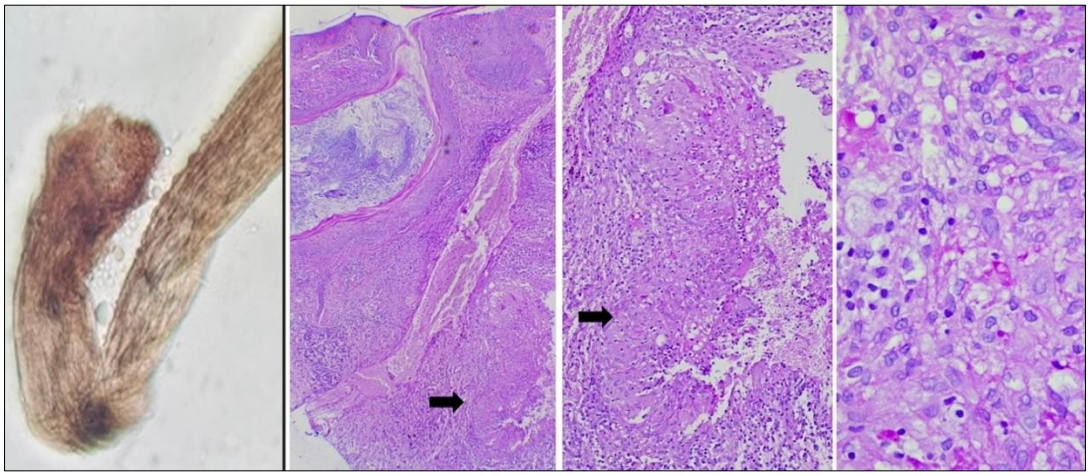

Fig. 3: A. Direct hair examination negative for fungal elements. B. Keratin plugs, naked granuloma (black arrow) with fibrosis and reactive epidermal hyperplasia (Hematoxylin & eosin, ×10). C. Naked granuloma (black arrow), peripheral lymphocytic infiltrate, and multinucleated giant cells (Hematoxylin & eosin, ×40). D. Epithelioid histiocytes with abundant cytoplasm and lymphocytes (Hematoxylin & eosin, ×100).

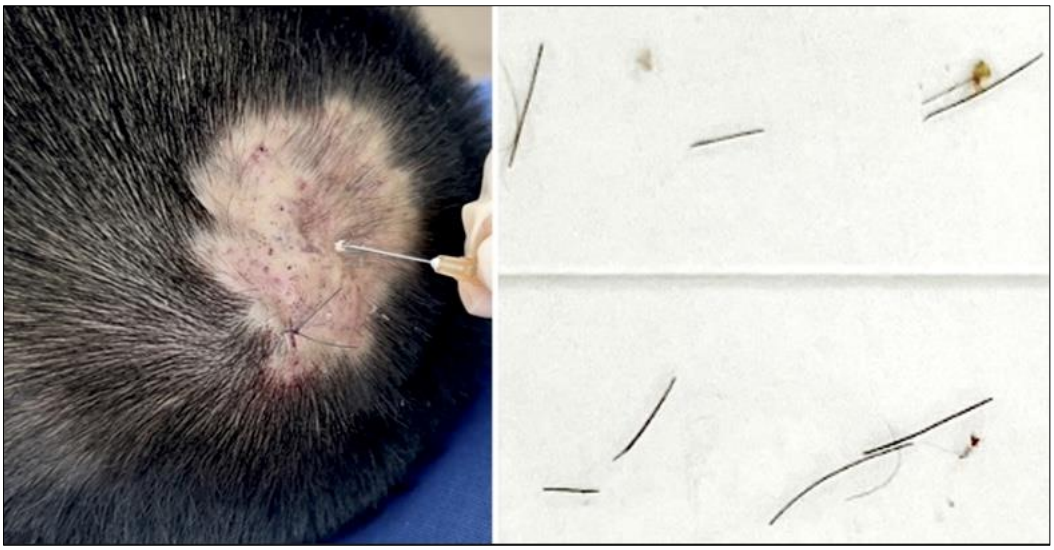

Fig. 4: Performance of biopsy and extraction of hairs and keratin plugs under dermoscopic guidance using a needle and Adson forcep.

DISCUSSION

This chronic granulomatous inflammatory reaction is generated as a host response to fragmented hair remnants and keratin material that perpetuate the inflammatory process after eradication of the fungal agent. Persistent kerion has been described as presenting more extensive involvement due to the reaction caused by ingrown hairs, which can act as foreign bodies and vary in extension, provoking local inflammation [4]. In another recent study conducted by Yao QH and collaborators at Hangzhou Third People's Hospital in China, it was demonstrated that ingrown hairs induced by fungal infection may worsen the persistent course of tinea capitis and that these can show significant clinical improvement once mechanically removed under dermoscopic guidance [5].

On dermoscopy, erythema, broken hairs, black dots, perifollicular scaling, pustules, yellowish crusts [6], cigarette-shaped hairs, comma hairs, corkscrew (spiral) hairs, zigzag hairs, and bent hairs have been observed. The nonspecific destruction of the keratin structure caused by keratinophilic fungi, which explains the morphological changes of the hair, has also been confirmed by scanning and transmission electron microscopy: interrupted and poorly compacted ends in cigarette-shaped hairs; bent hair shafts; asymmetrically disrupted cuticular layers [5].

The high incidence in children can be attributed to several factors: immature skin barrier function, insufficient secretion of fatty acids that inhibit fungal growth, and lack of hygiene, which may lead to easier exposure to sources of infection [7]. On the other hand, there is genetic susceptibility attributable to Ala12Thr polymorphisms in the KRT75 gene [8], which could favor abnormal hair penetration into the skin.

In this context, minor surgical procedures using a dermatoscope and sterile instruments to localize and extract ingrown hairs offer a simple and cost-effective treatment option, particularly for patients whose conditions persist or worsen with conventional therapy [5].

CONCLUSIONS

This is the first report describing the relationship between ingrown hairs and keratin plugs in inflammatory tinea capitis. Early detection and removal of short hairs and impacted keratin may improve clinical outcomes and prevent progression toward scarring alopecia and persistent follicular foreign body reaction, as these can perpetuate local inflammation and mimic therapeutic resistance. Since this complication has not been described in the literature, further research is required to develop diagnostic criteria and standardized therapeutic strategies. We therefore propose considering these abnormalities in cases of persistent kerion as part of a comprehensive clinical approach.

Research Data Availability: Does not apply.

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Conflicts of Interest: The authors declare that there are no conflicts of interest at the time of publication of this article.

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