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ADVANCED KERION CELSI IN A FIVE-YEAR-OLD CHILD: DIAGNOSTIC AND THERAPEUTIC CHALLENGES IN A RESOURCE-LIMITED HOSPITAL SETTING

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ABSTRACT

Tinea capitis is a dermatophytic infection of the scalp, predominantly affecting pediatric populations, which may progress to severe inflammatory forms such as kerion celsi, carrying a significant risk of aesthetic and psychosocial sequelae, including permanent scarring alopecia. We report the case of a previously healthy five-year-old girl from the Itabapoana River Basin region in Brazil, who presented to local hospital facilities with fever and extensive, exudative, purulent scalp lesions. Initially diagnosed as a nonspecific bacterial infection, she received oxacillin therapy without clinical improvement of the cutaneous lesions. Subsequent evaluation at a university outpatient clinic raised clinical and epidemiological suspicion of inflammatory tinea capitis consistent with kerion celsi. In the absence of laboratory resources for specific mycological culture, empirical treatment was initiated with griseofulvin (25 mg/kg/day for 16 weeks), topical ketoconazole 2%, and a seven-day course of amoxicillin-clavulanate for secondary bacterial infection. The patient exhibited an excellent clinical response, with resolution of purulent exudate within one week and progressive regression of inflammatory lesions in the following weeks. Nevertheless, scarring alopecia developed in previously affected areas, attributable to delayed diagnosis and the intensity of the inflammatory response. This case underscores the critical importance of early recognition of inflammatory tinea capitis, particularly in settings with limited diagnostic resources, and highlights the efficacy of evidence-based empirical combination therapy when advanced diagnostic testing is unavailable.

KEYWORDS: Tinea capitis; Kerion celsi; Inflammatory dermatophytosis; Pediatric fungal infection; Delayed diagnosis.

INTRODUCTION

Tinea capitis is a dermatophytic infection of the scalp that demand significant clinical and psychosocial consequences, particularly among pediatric patients. The infection may lead to follicular destruction and permanent scarring, resulting in irreversible hair loss. Moreover, the stigma often associated with visible scalp lesions can adversely affect a child's psychological well-being, potentially impairing school attendance and socioemotional development. In severe manifestations of this dermatophytosis, such as kerion celsi, the risk of long-term sequelae is markedly heightened. Recognizing the gravity of this condition and ensuring appropriate therapeutic interventions are therefore essential not only for resolving the infectious process but also for preserving the patient's physical integrity and overall quality of life.

Recent epidemiological trends indicate considerable geographic variability in the prevalence of tinea capitis and in the predominant causative agents. In southern China, a notable increase in tinea capitis prevalence has

been observed among children under three years of age, with a male predominance and *Microsporum canis* as the primary etiological agent, followed by *Trichophyton mentagrophytes* and *Trichophyton violaceum*.[1] In Greece, demographic shifts due to immigration from Africa and the Middle East have altered the epidemiological landscape, with a decline in *Microsporum canis*–associated cases and a concomitant rise in infections caused by anthropophilic species such as *Trichophyton violaceum* and *Trichophyton tonsurans*.[2] These changes highlight how population dynamics and human-to-human contact patterns influence the relative distribution of dermatophyte species.

In Brazil, the etiological profile of tinea capitis varies regionally. *Microsporum canis* and *Trichophyton tonsurans* are the most frequently isolated agents. In the southern and southeastern regions, *Microsporum canis* predominates, as demonstrated in studies from the cities of São Paulo and Botucatu, where it accounted for 88.2% and 75% of cases, respectively.[3,4] *Trichophyton tonsurans* is more commonly isolated in the northern region of Brazil.[5] Notably, even in São Paulo, *Trichophyton tonsurans* have significant presence despite *Microsporum canis* remains the most common agent.[3,6] The recent emergence of *Microsporum audouinii* as a causative agent of tinea capitis in the city of Rio de Janeiro and neighboring areas[7,8] further illustrates the dynamic nature of dermatophyte distribution, probably influenced by migratory patterns.[2,8] In many settings, the precise etiology of tinea capitis remains poorly characterized, as clinical features often permit empirical treatment without species identification.

Clinically, tinea capitis manifests in diverse forms, usually categorized as non-inflammatory and inflammatory. Non-inflammatory presentations are typically subtle, often leading to delayed diagnosis. These include fine scaling with single or multiple circular alopecic patches (“gray patch” variant), diffuse or irregular fine white adherent scales resembling generalized dandruff with mild hair shedding, and the “black dot” variant, characterized by well-demarcated alopecic areas with broken hairs at the scalp surface, producing a black-dot appearance.[9] Non-inflammatory forms tend to be more chronic but less likely to cause scarring compared to inflammatory variants such as kerion celsi and favus, which may result in permanent alopecia if untreated.[10,11]

Inflammatory forms include kerion and favus. Kerion represents a severe inflammatory response, presenting as a tender mass with pustules and crusting, commonly triggered by zoophilic dermatophytes such as *Microsporum canis*.[9,10] Favus, a rarer clinical form, is characterized by yellow, disc- or shield-shaped crusts (scutula) and is typically associated with *Trichophyton* species.[9,12] The clinical expression of tinea capitis is highly dependent on both the infecting dermatophyte species and the host’s immune response.[10,13] Understanding the pathogenesis and implementing effective therapeutic strategies, particularly for inflammatory forms, is crucial for timely diagnosis and management, especially in pediatric populations where the disease is most prevalent.[11,14,15]

This manuscript presents a case report of the kerion form of tinea capitis in a five-year-old child managed in the Itabapoana River Valley region of Brazil.

CASE REPORT

A previously healthy 5-year-old girl, accompanied by her parents, was admitted to the Pediatric Emergency Department of a hospital in the Itabapoana River Valley region, Brazil, in July 2025 with complaints of fever and extensive, exudative, and purulent scalp lesions. On admission, a provisional diagnosis of nonspecific bacterial scalp infection was established based on clinical assessment. Due to the severity of the patient condition, hospitalization was indicated, and empirical antibiotic therapy with oxacillin was initiated. After several days of inpatient care, fever resolved and the antibiotic course was completed. She was subsequently discharged with referral for specialized dermatologic evaluation, as her cutaneous lesions presented no significant improvement with the administered treatment. Her parents denied any pre-existing comorbidities.

In August 2025, the patient was scheduled for a clinical evaluation at the Pediatric Dermatology Outpatient Clinic of a neighboring university-affiliated hospital for etiological investigation and targeted management. The history was limited regarding the precise onset of lesions, as patient’s parents were unable to determine the exact start date or duration of symptom evolution. On initial physical examination, erosive lesions involving the dermis were observed in focal areas, characterized by an erythematous base, thick purulent crusts, yellow-green exudate, and associated alopecia. The lesions were extensive, spanning from the frontoparietal to the occipital region of the scalp. Epidemiological inquiry included questions about contact with pets, exposure to individuals with similar skin lesions at school, or contact with sand (potential reservoirs of dermatophytes); all responses were negative. General physical examination revealed the patient to be in good overall condition, with mild pallor, slight scleral icterus, and left cervical lymphadenopathy. No other systemic abnormalities were noted.

Laboratory investigations revealed microcytic hypochromic anemia (consistent with iron deficiency), leukocytosis, borderline thrombocytopenia, isolated elevation of indirect bilirubin, low serum iron and ferritin levels, vitamin D deficiency, normal transaminases, and elevated C-reactive protein, indicating an active systemic inflammatory process.

Mycological culture and etiological identification were not feasible due to the unavailability of appropriate laboratory resources at the referring institution, specifically the lack of dermatophyte-specific culture media in the microbiology department.

Given the clinical severity of the lesions and the high suspicion of inflammatory scalp dermatophytosis consistent with kerion celsi, empirical treatment was initiated based on clinical and epidemiological criteria, taking into account the patient's socioeconomic context. The therapeutic regimen included topical ketoconazole 2% shampoo (applied twice daily with a 5-minute contact time before rinsing), oral amoxicillin-clavulanate suspension (125 mg/31.25 mg per 5 mL, three times daily for 7 days), and oral griseofulvin (25 mg/kg/day as a single daily dose for 16 weeks). Additionally, iron and vitamin D supplementation were prescribed to address the associated microcytic hypochromic anemia and vitamin D deficiency. Temporary school exclusion was recommended due to the potentially contagious nature of the condition, and a medical certificate was issued in accordance with local regulations.

Seven days after initiating therapy, coinciding with completion of the amoxicillin-clavulanate course, nearly complete resolution of purulent exudate was observed. A follow-up complete blood count showed a marked reduction in leukocytosis, supporting the diagnosis of secondary bacterial infection and confirming the efficacy of the antibiotic regimen, although leukocyte counts remained slightly elevated and anemia persisted. Over the subsequent weeks, the patient demonstrated excellent clinical improvement, with progressive regression of inflammatory lesions and partial repigmentation of alopecic areas. However, due to delayed diagnosis and the intensity of the inflammatory response, the child developed permanent scarring alopecia in several affected regions—a well-recognized complication of untreated or late-treated inflammatory forms of tinea capitis.

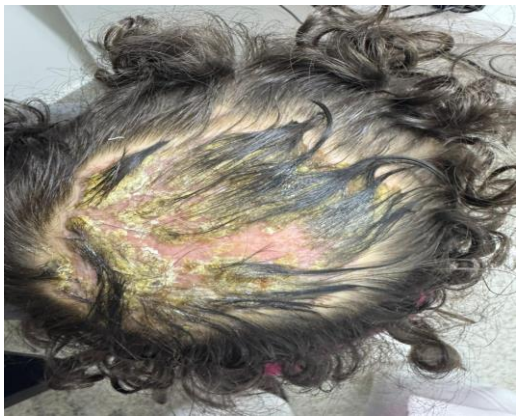

Figure 1: On initial outpatient evaluation, an extensive erythematous and scaling area was observed, featuring crusted plaques with a purulent appearance, associated alopecia, and exudate moistening the hair shafts.

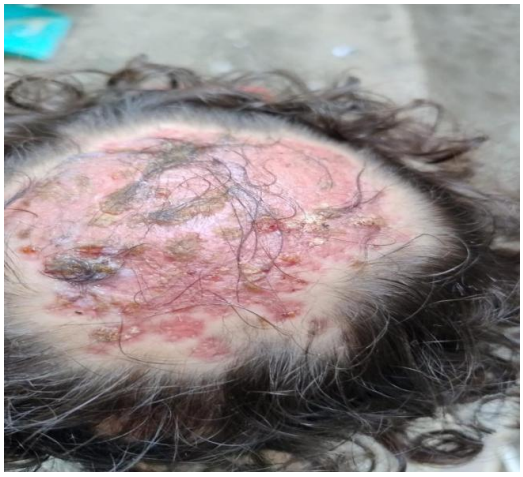

Figure 2: After one week of treatment, purulent plaques and yellowish exudate were no longer present. However, spontaneous detachment of dermal tissue along with tufts of hair was observed, likely resulting in areas of permanent scarring alopecia.

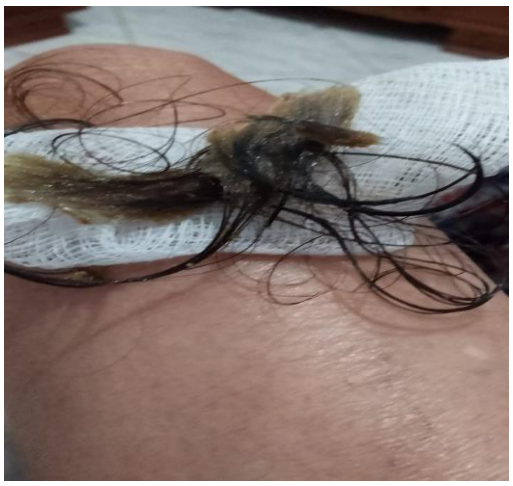

Figure 3: After one week of treatment, purulent plaques and yellowish exudate were no longer present. However, spontaneous detachment of dermal tissue along with tufts of hair was observed, likely resulting in areas of permanent scarring alopecia.

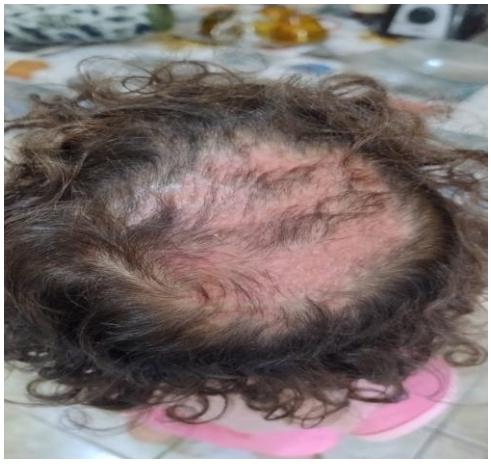

Figure 4: After 14 weeks of treatment, the patient demonstrated clear therapeutic success; however, areas of scarring alopecia were already evident.

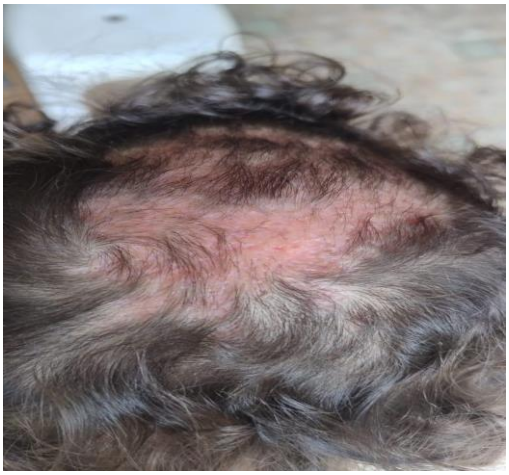

Figura 5: After 16 weeks of follow-up and completion of treatment, the final clinical record of the patient shows an extensive area of scarring alopecia.

DISCUSSION

The pathogenesis of kerion involves an exaggerated host immune response to dermatophyte infection, leading to the formation of tender, purulent plaques, pustules, and scalp abscesses.[16,17] This inflammatory reaction is particularly pronounced in infections caused by zoophilic dermatophytes, especially those of the genus *Microsporum*, and can result in significant tissue damage if not promptly treated.[10,18]

The case described herein represents a severe inflammatory form of tinea capitis presenting as kerion celsi in a previously healthy child. The progression to extensive, exudative, purulent, and deeply inflammatory lesions, with systemic manifestations evidenced by fever, leukocytosis, and elevated inflammatory markers, underscores the critical importance of early recognition of this dermatophytosis. Notably, the initial diagnosis was erroneously attributed to a primary bacterial infection, delaying appropriate antifungal therapy.

Diagnosis of kerion demands careful differentiation from other causes of scalp swelling, particularly bacterial infections such as abscesses or folliculitis.[19] Several studies highlight that kerion is frequently misdiagnosed as a bacterial abscess, a confusion compounded by the common occurrence of secondary bacterial superinfection, which may mask the underlying fungal etiology.[19,20,21,22,23,24,25] *Staphylococcus aureus* is the predominant bacterial pathogen implicated in secondary infections complicating kerion celsi. Its invasion of hair follicles and sebaceous glands promotes abscess formation and exacerbates the inflammatory response, manifesting clinically as localized erythema, warmth, pain, and sometimes systemic symptoms such as fever and malaise.[25,26] Often, the lack of complete clinical resolution following antibiotic therapy serves as the pivotal clue leading to the correct diagnosis[24,25], as occured in the present case from the Itabapoana River Valley region.

Definitive diagnosis of kerion is typically confirmed through mycological culture.[16,17,18,22,23,25] Sabouraud dextrose agar is considered the optimal culture medium for isolating dermatophytes from scalp specimens suspected of kerion, owing to its support of keratinophilic fungi such as *Trichophyton* spp. and *Microsporum* spp., the most common etiological agents.[27] When supplemented with antibacterial agents to suppress bacterial overgrowth, Sabouraud agar enhances the isolation and identification of dermatophytes from clinical samples, serving as an effective diagnostic tool.[28] This is especially relevant in kerion, where pruritus may lead patients to scratch lesions, introducing secondary bacterial contaminants via fingers or nails.[25] Moreover, culture methods using Sabouraud agar generally exhibit higher sensitivity compared to direct microscopy, further emphasizing their diagnostic value in dermatophytoses, including kerion.[23,25]

In the present case, the absence of direct mycological examination or fungal culture represents a diagnostic limitation commonly encountered in hospital settings with restricted laboratory infrastructure. Nevertheless, the clinical-epidemiological suspicion—coupled with a favorable response to griseofulvin, a first-line systemic antifungal for scalp dermatophytoses[29,30,31], strongly supports the diagnosis of kerion-type tinea capitis. The presence of secondary bacterial infection, a frequent complication in inflammatory forms due to disruption of the epidermal barrier, explains both the initial partial response to broad-spectrum antibiotic therapy (amoxicillin-clavulanate) and the systemic inflammatory laboratory findings. The successful outcome following a multimodal regimen, comprising prolonged systemic antifungal therapy, adjunctive topical antifungal, and short-course antibiotic coverage for bacterial superinfection, aligns with current literature recommendations advocating combined therapeutic strategies for inflammatory tinea capitis.[19,24,25,32] Despite therapeutic success, the delayed diagnosis resulted in permanent scarring alopecia, highlighting the need for heightened clinical awareness and timely intervention, particularly in resource-limited settings.

CONCLUSIONS

This case report describes a severe, advanced form of kerion celsi–type tinea capitis in a five-year-old child, initially misdiagnosed due to clinical overlap with secondary bacterial infection. The progression to extensive, inflammatory, and exudative scalp lesions, accompanied by systemic manifestations and subsequent scarring alopecia, underscores the critical need for heightened clinical suspicion in pediatric patients presenting with inflammatory scalp disorders, even in the absence of classic epidemiological risk factors such as exposure to pets or known dermatophyte reservoirs.

The favorable response to a combined therapeutic regimen, comprising first-line systemic antifungal

therapy (griseofulvin), a short course of antibiotic treatment for secondary bacterial infection, and adjunctive topical antifungal, aligns with current evidence-based recommendations for managing inflammatory tinea capitis.

Furthermore, this case highlights the diagnostic challenges encountered in healthcare settings with limited laboratory infrastructure. The unavailability of mycological culture facilities delayed etiological confirmation and likely contributed to a suboptimal prognosis. In such resource-constrained environments, early clinical recognition of atypical or severe presentations of tinea capitis becomes paramount. The implementation of accessible, low-complexity diagnostic protocols, and greater clinician awareness, could significantly reduce diagnostic delays and mitigate long-term complications.

Although the resulting scarring alopecia appears irreversible, it might have been partially prevented with earlier, targeted intervention. Recognizing kerion celsi not merely as a superficial skin infection but as a potentially disfiguring and psychosocially impactful condition is essential. Timely diagnosis and management are crucial not only for preserving skin integrity but also for safeguarding the child's psychological well-being and social development, both in the present and throughout.

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