Review

The kerion: an angry tinea capitis

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Abstract

Tinea capitis has a high incidence with a global changing pathogen distribution, making this condition a public health concern around the world. As the infection is initially asymptomatic, it is easily spread. Moreover, it is present in many fomites, including hairbrushes, pillows, and bedding. Prompt recognition and treatment is necessary for kerion, an inflammatory subtype characterized by tender boggy plaques with purulent drainage. Kerion is usually associated with infection by zoophilic dermatophytes, although other sources have been described. Treatment for this severe form of dermatophytic infection can be challenging. In addition to the use of topical treatments, oral administration of griseofulvin, terbinafine, itraconazole, or fluconazole is often required. Griseofulvin, the first-line treatment, may not completely eradicate pathogen colonization of the host and may contribute to reinfection and prevalence of infective but asymptomatic carriers. This review highlights new agents that are being evaluated for the treatment of kerion and typical tinea capitis, enhanced diagnostic criteria, and a grading system for kerion evaluation.

Introduction

Tinea capitis represents a growing public health concern due to changing geographic patterns of infection and high incidence. As one of the most common cutaneous infections in pre-pubertal children, incidence of infection is high globally, with the World Health Organization claiming it is the second most common dermatologic infantile infection after pyoderma.[1] In the United States, between 1995 and 2004, the prevalence rates of tinea capitis were reportedly up to 15%.[2,3] A more recent study in the United States determined a carriage rate of 6.6%. Infection rates at participating schools ranged from 0% to 19.4%, with black children demonstrating the highest rate of infection (12.9%).[2] Infection is associated with poor hygiene and low socio-economic status. Adding to the epidemic are three factors: late recognition of suspicious lesions, an incubation period of a few weeks in which patients are contagious but asymptomatic, and transmission from household pets.[4] Prompt treatment of an inflammatory subtype, the kerion, is necessary to preclude permanent scarring and alopecia. However, current treatment may not completely eradicate pathogens.

Etiology

Tinea capitis can be caused by any dermatophyte, except *Epidermophyton floccosum* and *Trichophyton concentricum*. The most commonly implicated dermatophytes are of the *Trichophyton* and *Microsporum* genera. The list of causative pathogens based on geographic area is shown in Table 1.[5]

While the incidence has been decreasing in the United States,[6] it has greatly increased in Europe and other developing countries.[7]

The development of tinea capitis and kerion in prepubertal age groups is likely due to a lack of sebum secretion. Low sebum production results in decreased fatty acids and increase in pH of the scalp, facilitating colonization and subsequent infection by dermatophytes. In addition, poor hygiene, playing in sand, crowded living conditions, and low socio-economic status have been

Table 1 Geographic distribution of pathogens causing tinea capitis

Location	Pathogen
Southern Europe	*M. canis*
Central Europe	*M. canis*, *T. verrucosum*, *T. mentagrophytes*, *T. violaceum*
Eastern Europe	*M. canis*, *M. audounii*, *T. violaceum*
United Kingdom, France	*M. canis*, *T. verrucosum*, *T. mentagrophytes*, *T. violaceum*
United States	*T. tonsurans*
Canada	*T. mentagrophytes*, *M. canis*
Mexico	*M. canis*, *T. tonsurans*
Caribbean	*T. tonsurans*, *M. canis*
India, Pakistan	*T. violaceum*
China	*T. violaceum*, *T. mentagrophytes*
Middle East	*M. canis*, *T. violaceum*, *T. schoenleinii*, *T. verrucosum*
Western Africa	*Microsporum audouinii*, *T. soudanense*, *T. yaoundei*
Eastern Africa	*T. schoenleinii*

associated with the development of tinea.[8] In particular, kerion is mostly associated with infection by zoophilic dermatophytes.[9] Tinea capitis is easily spread from the infected and often asymptomatic carrier to others, making family epidemics very common. Spores of *Trichophyton* spp. have been cultured from diverse sources, including combs, hats, and pillows. Practices such as the use of oils, frequency of hair washing, and tight braiding, have not shown to increase the risk of infection.[6]

Epidemiology

Tinea capitis most commonly infects children between 3 and 7 years of age. Infants and adults are rarely affected. Reports of infection in infants are usually related to the use of broad-spectrum antibiotics and immunosuppression.[10] Some studies have shown no gender predilection,[11] while others report an increased prevalence among boys,[12] and others report the highest prevalence among African–American girls.[2,6] Short hair is associated with higher incidence because fungal spores can access the scalp more easily.[6] A recent study also demonstrated a higher risk of kerion development in rural populations compared to suburban populations, perhaps due to greater contact with animals. This study postulated that a greater incidence in boys is related to their increased contact with farm animals compared to girls.[10]

In addition to children, there are several other groups that are at greater risk of developing tinea capitis and kerion. These include people with diabetes, anemia, and immunosuppression due to leukemia, organ transplant, and the use of immunosuppressants. The use of immunosuppressants may interfere with hair production and shaft strength, allowing for colonization by fungi.[13] Of note, tinea infections are not as common in patients with HIV, likely because of increased colonization with *Malassezia*, thus competitively inhibiting the colonization of dermatophytes.[14] Chronic users of topical or systemic corticosteroids may be more likely to develop infection. Women undergoing major hormonal changes, including during pregnancy and menopause, may also be more prone to infection because of reduced sebum excretion.[15] In fact, the majority of adults with tinea capitis are women, likely due to periods of hormonal changes, greater exposure to children, and increased visits to the hairdresser.[16]

Classification

Infection with tinea capitis can be classified into three patterns: endothrix; ectothrix; and endothrix favosa. The endothrix pattern is characterized by fungal spores dwelling within the hair shaft, thus allowing the cortex to remain intact. The ectothrix pattern of infection involves fungal spores attaching to the surface of the hair shaft, allowing for cuticle destruction. Endothrix favosa is characterized by the presence of hyphae and air bubbles within the hair shaft.[17]

Clinical presentation

Clinical presentation can be subdivided into two subtypes: inflammatory and non-inflammatory. Cervical and suboccipital lymphadenopathy are commonly found and may serve as a diagnostic clue.[17] Non-inflammatory presentation is characterized by scaling, a seborrheic form, and loss of hair. The ectothrix pattern commonly causes the non-inflammatory clinical presentation, with circumscribed patches of alopecia due to destroyed cuticles. The gray patch presentation is an ectothrix *Microsporum* infection with patchy circular alopecia and scaling. The black dot presentation is caused by an endothrix *Trichophyton* infection, which causes breakage of the hair shaft at the scalp, leaving behind black dots. The diffuse scale presentation is characterized by dandruff-like scaling of the scalp.[7]

The inflammatory subtype is characterized by tender plaques covered with broken hairs and pustules. This subtype can be further divided into the pustular form, favus, Majocchi granuloma, mycetoma, and kerion. The pustular form is associated with a patchy alopecia and scattered pustules or low-grade folliculitis. Favus presents with erythema around hair follicles and cicatricial alopecia. Eventually, scutula, or yellow-crusted cup-shape lesions, form with hair loss and scarring. Favus may also involve the skin and nails.[17] Majocchi granuloma is characterized by papular, pustular, or nodular lesions on the limbs or face. Mycetoma is characterized by nodular lesions overlying erythematous and scaly plaques, sinus tracts with purulent drainage, and pseudoalopecia.[18]

Kerion is commonly confused with bacterial abscesses due to the purulent drainage. It presents as a painful, crusty carbuncle-like boggy plaque that requires early diagnosis to prevent bacterial superinfection, folliculitis, and permanent alopecia (Figs 1 and 2). It usually occurs as a solitary lesion, most commonly in the occipital area of the scalp,[19] although it can occur as multiple lesions. It has been reported to occur in other sites, including the beard, eyebrow, and vulva.[20–22] The course of kerion begins with dermatophytic folliculitis and a dry lesion with scaling and short hairs. Development of kerion depends on the type of pathogen as well as the host immune status. Erythema, tenderness, and inflammation rapidly follow the initial lesions. Short hairs are eventually expelled and within 8 weeks, the infection usually resolves. Dermatophytid reactions, which are immunological reactions caused by infection or inflammation, commonly occur. The reaction can be localized or generalized and often presents as eczematous lesions with papules, scaly patches, vesicles, and pustules. Classically, these present as the "ear sign," in which erythematous papules and scaling are found overlying the helix, antihelix, and retroauricular regions. Several studies have demonstrated a high rate of association of dermatophytid reactions and kerion. In one study, 68% of patients with kerion had an accompanying dermatophytid reaction. These reactions tend to occur at the height of the infection

Figure 1 Clinical example of kerion. Note the alopecia surrounding the lesion as well as pustules and purulent drainage over multiple lesions
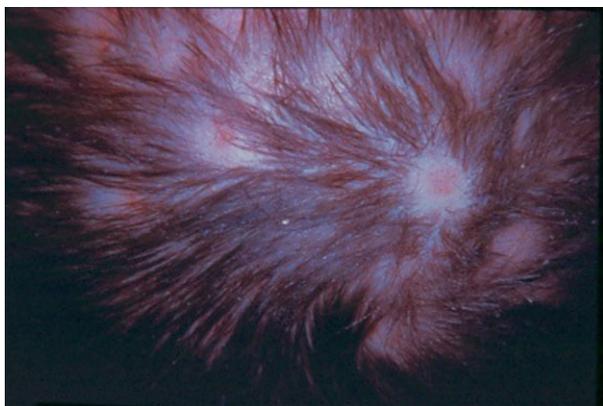
Figure 2 Clinical example of alopecia following infection

and therefore directly before or after initiation of antifungal therapy.[19] Therapy should not be discontinued.

Histopathology

Histologic analysis is useful in culture-negative patients. As most cases of kerion are culture negative, histopathology usually reveals fungal spores surrounding the hair follicle and hyphae within the hair shaft. Inflammatory infiltrate is found in the dermis, and giant cell reaction may occur due to follicular destruction.[17] In addition, a periodic acid-Schiff stain can be used to confirm diagnosis.

Kerion is specifically characterized by neutrophilic and granulomatous infiltrates in the early stages and fibrotic scar in later

Table 2 Major and minor features suggestive of kerion

Major criteria	Minor criteria
Tenderness to palpation	Dermatophytid reaction
Alopecia surrounding the lesion	Regional lymphadenopathy
Numerous pustules and purulent drainage	Short hairs on dermoscopy
	Boggy plaques
Scaling at the lesion	Clear dermarcation of borders
	Overlying erythema
	Pruritus

stages. There are four types of histopathological patterns associated with kerion. First, suppurative folliculitis is characterized by a perifollicular inflammatory infiltrate with spongiosis and infiltrates of neutrophils, lymphocytes, and plasma cells. Most hair follicles are in the catagen stage. The second pattern is suppurative folliculitis with suppurative dermatitis. This pattern is characterized by a perifollicular and perivascular infiltrate of neutrophils with edema in the papillary dermis. Hair follicles are in the catagen or telogen stage with few disrupted follicles. The third pattern is suppurative folliculitis with suppurative and granulomatous dermatitis, which is characterized by an infiltrate of neutrophils, lymphocytes, and plasma cells, and a granulomatous reaction. There is a decreased number of hair follicles. Finally, the fourth pattern is suppurative and granulomatous dermatitis with fibrosing dermatitis. In this pattern, there is an inflammatory infiltrate of neutrophils, lymphocytes, and plasma cells, with granuloma formation, increased number of collagen fibers, and absent hair follicles consistent with scarring alopecia. The latter two patterns have negative periodic acid-Schiff stains.[18]

Diagnosis

Tinea capitis is often misdiagnosed, thus delaying proper treatment and allowing spread of infection. There are no established clinical guidelines for kerion, and it is often confused with a bacterial infection. We propose major and minor diagnostic criteria for kerion (Table 2). If these criteria are present, a dermatology consult should be requested before surgically manipulating the site or labeling the infection origin as bacterial. In addition, we devised a grading system for kerion to ease communication between practitioners about the severity of the lesion (Table 3). While the grade of the lesion does not change treatment, it may be helpful in determining treatment response and prognosis. In addition, it would be of interest to determine risk factors that correlate to the grade of lesion, e.g., if immunosuppression predisposes an individual to a higher-grade lesion.

A culture for fungi is the gold standard to confirm the diagnosis. A simple method that has demonstrated good efficacy is the hairbrush culture method.[23] After obtaining a sample of hair, hair and scalp scale, or scalp brushings, the sample is placed

Table 3 Grading of kerion lesions

Grade	Characteristics
1	A few pustules overlying erythematous plaque; most hairs in catagen phase but still intact; histology shows suppurative folliculitis
2	Pustules and papules overlying erythematous plaque; scaling at the periphery of plaque; few disrupted hair follicles; histology show suppurative folliculitis with suppurative dermatitis
3	Pustules, papules, scaling, and erythema present; vesicles may be present; clear patches of alopecia; histology shows suppurative folliculitis with suppurative and granulomatous dermatitis; negative PAS stain
4	Pustules, scaling, and erythema present; permanent patches of alopecia with scarring; histology shows suppurative and granulomatous dermatitis with fibrosing dermatitis; negative PAS stain

PAS, periodic acid-Schiff.

in Sabouraud liquid medium or dermatophyte test medium. At least one plate should also include cycloheximide–chloramphenicol to prevent mold growth and bacterial contamination that may suggest false findings. Using a cytobrush, which is a sterile tool with softer bristles, improves sample quality and minimizes discomfort. Cultures often require 2 weeks of growth before examination. With *Trichophyton verrucosum*, cultures should be incubated for 3 weeks. Other culture methods include growth on rice grains, urease test, and hair perforation test.[24] Of note, patients with kerion have a high false-negative culture rate with conventional methods of sampling, likely because the sample represents the inflammatory response. Therefore, gauze swabs or use of a moistened bacterial swab from the pustular area in addition to directly pressing the agar plate on to the kerion may yield more positive cultures.[7] Samples should also be examined under a microscope using 10–30% potassium hydroxide with or without calcofluor.

Dermoscopic examination of tinea capitis is increasingly becoming a quicker and faster method of diagnosis. Comma hair is a classic finding, seen in both endothrix and ectothrix patterns of infection.[25] Corkscrew hair is also a classic finding, observed in dark-skinned patients. Less specific findings include black dots, broken hair, pigtail hair, and zigzag hairs.[25] More recently, bar code-like hair has been reported in the ectothrix pattern of infection.[26] Dermoscopic examination is particularly important in differentiating tinea capitis from alopecia areata. Specific trichoscopic findings of alopecia areata include yellow dots, exclamation mark hairs, and short vellus hair.[27] Antiseptic precautions with the use of a film between the dermoscope and lesion should be employed to minimize spread of infection.

Wood lamp examination is useful if the infection is caused by *Microsporum canis*, which exhibits green fluorescence under the light, or tinea capitis favosa, which exhibits faint blue fluorescence. However, *Trichophyton* infections, except for *T. schoenleinii*, do not fluoresce.[28]

Table 4 Differential diagnoses of kerion and tinea capitis

Diagnosis	Differentiating features
Alopecia areata	No epidermal changes; exclamation point hairs; no crusting, inflammation, or pustules
Atopic dermatitis	Personal or family history of asthma, hay fever, sensitive skin; usually no lymphadenopathy; usually no alopecia
Bacterial scalp abscess	Plucking hair produces pain; usually no alopecia
Psoriasis	Family history of psoriasis; gray or silver scale; other systemic involvement
Seborrheic dermatitis	Greasy scale; usually no alopecia and lymphadenopathy
Trichotillomania	No scaling; hairs are varying lengths

A more recent method of diagnosis is the use of real-time polymerase chain reaction (PCR) test or PCR reverse-line blot assays to detect *T. tonsurans* ribosomal DNA in fluid used to rinse the patient's hairbrush. This test takes 5 h.[23]

Differential diagnosis

The differential diagnosis of kerion is vast. This complication of untreated tinea capitis should be distinguished from diagnoses with differentiating features listed in Table 4.[29] Kerion may be confused with dissecting cellulitis, which is characterized by tender, suppurative nodules that produce draining sinus tracts and scarring. Unlike tinea capitis, dissecting cellulitis usually occurs in African American adults as a deep lesion. It is important to perform cultures and swabs to confirm fungal infections in cases of atypical presentation.[30]

Treatment

For any case of tinea capitis, systemic antifungal agents are indicated as topical agents will not penetrate the hair shaft.[7] However, use of topical agents is being assessed, as described below. Griseofulvin is the first-line treatment for tinea capitis since 1958.[31] Eight weeks of treatment are necessary for *Microsporum* infections, and 12–18 weeks of treatment are necessary for *Trichophyton*.[32,33] It should be avoided in pregnant patients. It is also contraindicated in patients with lupus erythematosus, porphyria, and severe liver disease. One study reported that griseofulvin does not eradicate *Trichophyton* in most infected children but simply causes symptom resolution. As such, persistent infection and spread of infection may occur.[34]

Newer agents, including terbinafine, itraconazole, and fluconazole, are equally effective according to recent clinical trials. In addition, the newer antifungal agents are more concentrated in the hair and therefore may provide fungicidal concentrations even after treatment is completed, allowing for shorter treatment duration and lower rates of reinfection. Terbinafine is a

fungicidal drug that acts on the cell membrane. A meta-analysis of randomized clinical trials did not demonstrate significant difference in efficacy of terbinafine compared to griseofulvin.[35] In addition, terbinafine requires 2–4 weeks of treatment for *Trichophyton* infections.[36] A newer granule formation of terbinafine that can be sprinkled on food is approved for use in the United States. However, the cost is high in developing countries, precluding its use. It is not recommended for the treatment of *Microsporum* infection. Side effects include gastrointestinal discomfort and rashes.

Itraconazole is a fungistatic and fungicidal drug, depending on its tissue concentration. Treatment duration is between 2 and 4 weeks. It is shown to have comparable efficacy to griseofulvin and terbinafine.[37,38] It is also available in the liquid form with fewer side effects. However, it has several drug interactions with warfarin, antihistamine, antipsychotics, anxiolytics, digoxin, cyclosporine, and simvastatin and must be used cautiously in patients taking multiple medications.[7] Fluconazole serves as a valid alternative; it is present in the liquid and rectal forms, persists for several weeks after administration, and must only be given once weekly. Multiple studies have demonstrated that fluconazole is comparable to griseofulvin in terms of use, side effects, compliance, and efficacy.[39–41] However, its high cost limits its use. Voriconazole shows greater activity against pathogens than griseofulvin does, but its high cost and adverse effects are often prohibitive.[42]

The use of topical squalamine, a natural aminosterol isolated from dogfish shark, has also been reported. While this drug does not cause complete eradication of fungi, it provides partial clinical response with some hair growth in areas of the lesion. It is suggested to be used as an adjuvant therapy to decrease treatment duration and increase compliance.[43] In addition, use of 1% or 2.5% selenium sulfide, 2% ketoconazole, 1–2% zinc pyrithione, and 2.5% povidone iodine shampoos may reduce transmission if used within the first 2 weeks of infection.[44,45] Use of prednisone during the first week of infection may alleviate pain and swelling accompanying inflammatory subtypes.[46]

Kerion must be treated emergently with griseofulvin, as failure to treat can lead to permanent scarring and alopecia. Terbinafine should only be used if *Trichophyton* is documented as the cause of infection. Other treatments, including itraconazole pulsed therapy, show an initial improvement followed by recurrent and worse lesions.[47] It is important that surgical drainage of kerion is not performed, despite the similarities with bacterial abscesses. In addition, treatment with steroids has not been shown to reduce scarring or confer long-term advantages but may reduce itching and pain.[46]

Patients should be monitored every 14 d starting with the fourth week of treatment to determine treatment response.[17] Post-treatment clearance of infection should be verified with culture. For infections with no signs of clinical improvement, the treatment regimen should be altered by increasing the dose or changing the agent.

Prevention

Prevention of epidemic spread of tinea capitis requires patient education about proper hygiene techniques and prompt treatment. One study provided patients with a video, emphasizing frequent cleaning and washing of rooms and training clothes, immediate showering after sports practice, and recognition and immediate reporting of lesions. The video resulted in a significant decrease in culture-positive rates.[23] In addition, patients should be informed to restrain from sharing hairbrushes, combs, hats, dress-up clothes, and other hair accessories with siblings or classmates who are infected. Additional hand washing should be emphasized. Blankets and bedding should be changed daily to prevent spread. Curtains should be washed regularly. Screening of children and staff at schools has also been proposed to prevent epidemics.

Children who are receiving appropriate treatment are allowed to return to school. However, all family members should be screened and treated if necessary to prevent recurrence. Asymptomatic carriers with high fungal spore load should be treated with oral drugs. In patients with low spore load, topical treatment can be used with repeat tests to ensure clearance.[48]

Conclusion

Kerion is a subset of inflammatory tinea capitis that can result in permanent alopecia and scarring. Prompt recognition and treatment is desirable. We have delineated features suggestive of this diagnosis as well as a grading system. Diagnosis includes culture, dermoscopic examination, Wood's lamp examination, or PCR testing. Treatment requires the use of oral antifungal agents with adjuvant topical treatments to prevent the spread of infection. As tinea capitis remains a public health concern, patients should be educated on preventative measures and lesion appearance, particularly of the angry subtype, the kerion.

Questions (answers provided after references)

1 Risk factors for the development of tinea capitis include all of the following except:
 a Prepubertal age
 b Low socio-economic status
 c Tight hair braiding
 d Rural living conditions
2 Of the following groups of immunosuppressed patients, which are least likely to develop tinea capitis infections?
 a Patients with HIV
 b Chronic steroid users
 c Patients with leukemia
 d Organ transplant patients

3 True or False: Patients with dermatophytid reactions should undergo discontinuation of current treatment with continuation only after resolution of the reaction.

4 How is the presentation of kerion different from that of bacterial abscesses?

a Presence of purulent drainage

b Accompanying tenderness

c Presence of alopecia

5 What is the gold standard of culture for diagnosis in kerion?

a Culture growth on rice grains

b Hairbrush culture method

c Urease test

d Hair perforation test

6 Findings of kerion on dermoscopy include all of the following except:

a Comma hairs

b Corkscrew hairs

c Zigzag hairs

d Exclamation mark hairs

7 What is the first-line treatment of kerion?

a Terbinafine

b Griseofulvin

c Itraconazole

d Fluconazole

8 True or False: Children with kerion should be allowed to return to school after completion of treatment.

9 Major diagnostic criteria of kerion include all of the following except:

a Tenderness to palpation

b Purulent drainage

c Boggy plaque

d Scaling of lesion

10 True or False: Prevention of epidemic spread of tinea capitis requires patient education about proper hygiene techniques and prompt treatment.

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Answers

1 c; 2 a; 3 False – therapy should not be discontinued; 4 c; 5 b; 6 d; 7 b; 8 True; 9 c; 10 True.