Tinea Capitis

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33.1 Introduction

The most common pediatric dermatophyte infection worldwide is that of the scalp. The incidence of tinea capitis is highest in 3- to 7-year-old children, although a recent retrospective study has found 47 % of tinea capitis occurring in individuals 13 and older [1]. In the same study, tinea capitis disproportionately affected the black population with black individuals comprising 87 % of all patients diagnosed with tinea capitis in a dermatology clinic during a 15-year period. It routinely affects individuals who are otherwise healthy, but there is increased susceptibility in immunocompromised individuals. The growth and transmission of the fungi are favored in warm, humid, and overcrowded environments. Incidence appears to correlate with low socioeconomic status, large family size, poor hygiene, and frequent person-to-person contact. The incidence of tinea capitis is increasing worldwide and has had considerable shifts in the causative organisms over the past 50 years. This infection is especially prevalent in Afro-Caribbean and African American children.

Tinea capitis is the most common superficial fungal infection in North America and in the United States. It affects 3–8 % of children in urban areas, especially those of African or Afro-Caribbean descent. Over the past 50 years, the epidemiology of tinea capitis has changed more than other tinea infections. *Microsporum canis* and *Microsporum audouinii* have been eradicated and replaced by *Trichophyton tonsurans* as the cause of tinea capitis in 95 % of the cases in the United States and Canada [2, 6]. Although *M. canis* is still the most predominant cause of tinea capitis in Europe, Mexico, South America, and China, trends are changing. The United Kingdom now reports 83 % of tinea capitis cases being caused by *T. tonsurans*, and cities like Paris and Amsterdam are showing similar trends [7]. *Microsporum audouinii* is the most prevalent organism in Africa; however, *T. violaceum* and *T. soudanense* are common organisms in Asia and Africa.

The dermatophytes causing tinea capitis are from the *Microsporum* and *Trichophyton* groups. *M. canis* is the most

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common cause worldwide. *M. canis* can be carried by pets in the household and is therefore difficult to eradicate. There has been an increasing incidence of *T. tonsurans*, which now comprises 95 % of all tinea capitis in the United States and Canada. Its rates are increasing similarly in Europe as well [2]. *T. soudanense* and *T. violaceum* are major causes of tinea capitis in parts of Africa.

Dermatophytes cause infection by entering keratinized tissues. The dermatophyte hyphae penetrate hair cuticles and grow downward into the hair, invading newly formed keratin, which becomes visible at the skin surface after 12–14 days of inoculation. The hair becomes brittle, causing breakage by 3–4 weeks.

33.2 Clinical Features

There are several clinical presentations of tinea capitis, including gray patch, moth eaten, black dot, diffuse scale, pustular, and kerion. The gray patch (Fig. 33.1) and moth-eaten (Fig. 33.2) presentations consist of areas of alopecia with superficial scaling, with scaling being more prominent in the gray patch presentation. The black dot presentation is seen due to breakage of hair shafts, leaving tiny black dots in the affected areas. Tinea capitis presenting with diffuse scale mimics generalized dandruff. The slight erythema of the scalp in the above presentations is difficult to appreciate on darker-skinned individuals. The presence of comma and corkscrew hairs on dermoscopy may be a reliable sign of tinea capitis in such patients [3]. Pustular tinea capitis often implies a superficial bacterial infection (Fig. 33.3). A kerion represents a delayed hypersensitivity reaction and granuloma formation due to immune reaction to the dermatophyte and is often mistaken for a bacterial abscess (Figs. 33.4 and 33.5). A kerion can be painful with associated adenopathy (Fig. 33.6) and can also leave scarring alopecia. In a study consisting of predominantly black children, scaling of the scalp presenting with adenopathy had a positive predictive value of 97 % for tinea capitis [4]. In another study, Hispanic children presenting with scalp hyperkeratosis were more likely than not to have tinea capitis [5]. There have been several case reports of erythema nodosum associated with kerions, which usually occurs at the height of infection or shortly after induction of therapy. It is thought to be due to delayed hypersensitivity to systemically absorbed fungal antigens, antigen release induced by treatment, or a large antigen load from the primary inflammatory response [6].

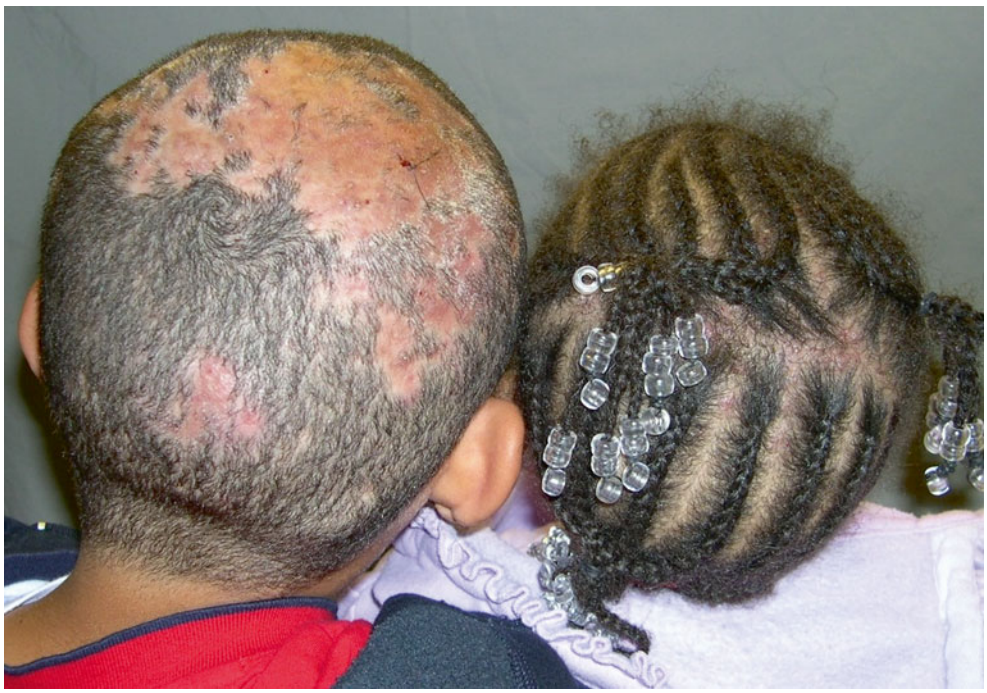

Fig. 33.1 A 12-year-old black male with *gray* patch tinea capitis (*left*) with his sister who also had a positive culture (*right*) (Photo courtesy of Dr. Tor Shwayder)

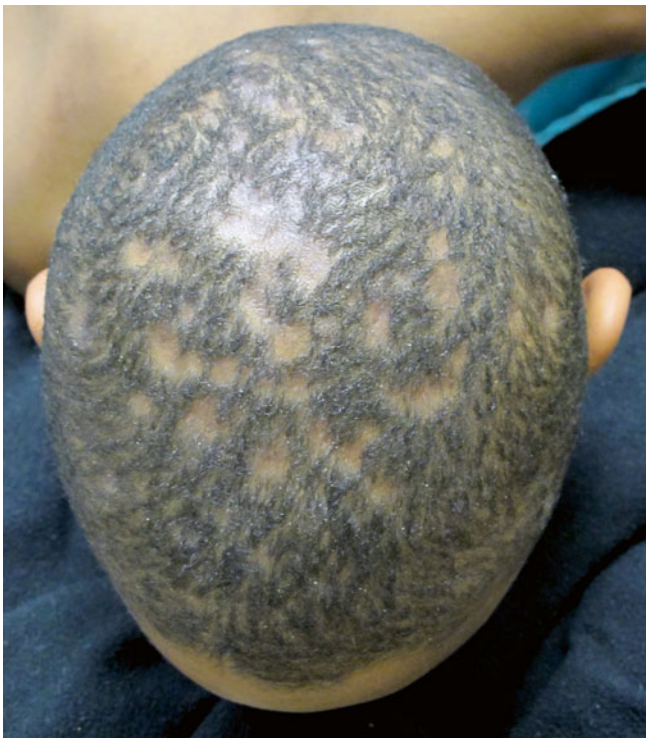

Fig. 33.2 A 6-year-old black male with moth-eaten tinea capitis (Photo courtesy of Dr. Tor Shwayder)

Fig. 33.3 A 5-year-old black female with pustular tinea capitis (Photo courtesy of Dr. Tor Shwayder)

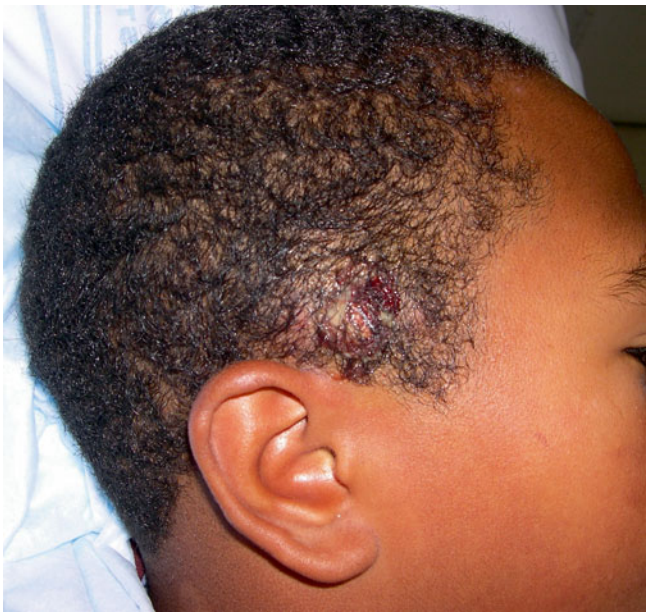

Fig. 33.4 An 11-year-old black male with a kerion (Photo courtesy of Dr. Tor Shwayder)

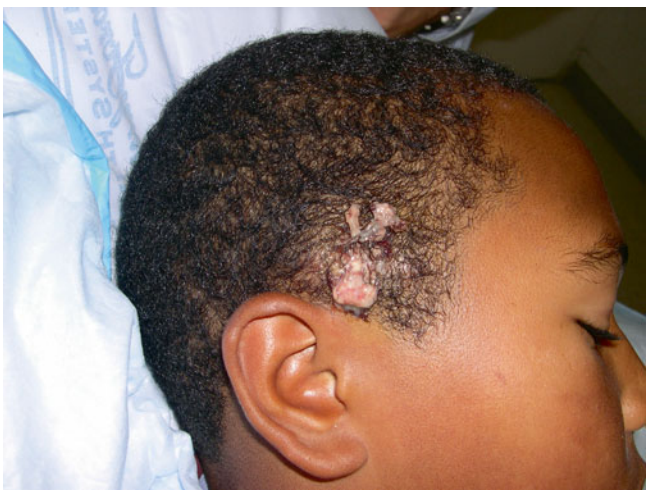

Fig. 33.5 Same patient as Fig. 33.4 with expressed contents of the kerion (Photo courtesy of Dr. Tor Shwayder)

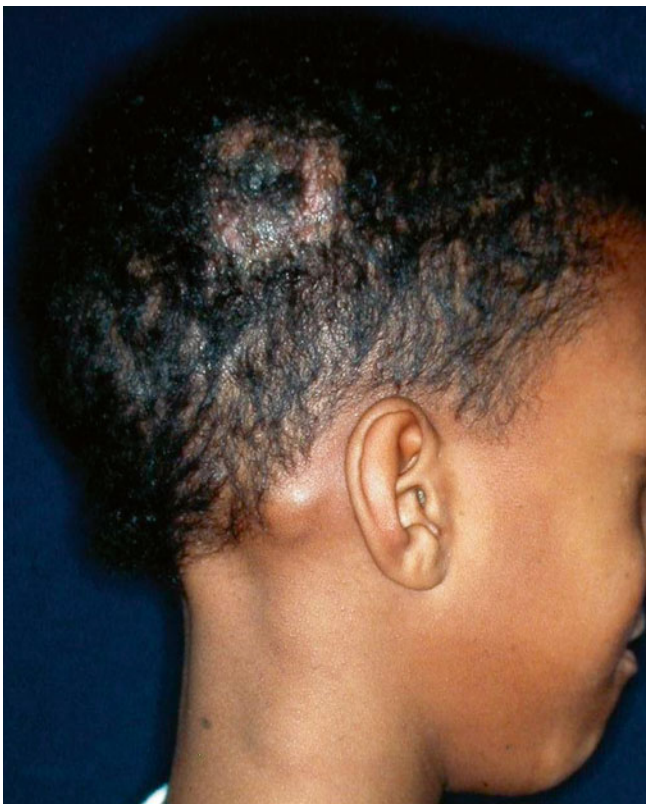

Fig. 33.6 Tinea capitis with lymphadenopathy (Photo courtesy of Dr. Tor Shwayder)

33.3 Natural History and Prognosis

With early diagnosis and appropriate treatment, transmission and permanent sequelae of tinea capitis, including scarring and alopecia, can be avoided.

33.4 Histopathological Features

Dermatophytes are usually visible within and around the hair shaft and can be elucidated by using periodic acid-Schiff or methenamine silver stains. Multinucleated giant cells with surrounding chronic inflammatory infiltrate are sometimes present along the degenerated hair follicles. A mild perivascular lymphocytic infiltrate may be present in the dermis. Kerions may have perifollicular dermal mononuclear infiltrate, follicular pustules, interfollicular neutrophilic infiltrate, and chronic inflammatory infiltrate.

33.5 Diagnosis and Differential Diagnosis

The diagnosis of tinea capitis must be confirmed by microscopic visualization of branching hyphae, culture, or polymerase chain reaction (PCR). Samples can be collected using scalp brushings or swabbing in cases of pustular tinea capitis and kerions. Potassium hydroxide preparation and fluorescent stains such as calcofluor of the skin and hair are both rapid but can have false-negative results, especially in early disease. Cultures are more time consuming, requiring at least 6 weeks of incubation to establish negative results, but are more sensitive and can give antifungal susceptibilities. Although expensive, PCR is useful when the patient has been pretreated with antifungals. Wood's lamp can be used to detect infection with *Microsporum* spp. which fluoresce bright green.

Differential diagnoses of tinea capitis include seborrheic dermatitis and psoriasis when presenting with erythema and hyperkeratosis of the scalp. Alopecia areata may be considered for tinea capitis presenting with patchy hair loss. Dermoscopy may be useful to distinguish tinea capitis from alopecia areata. Pustular tinea capitis can look like impetigo, which begins as vesicles that evolve into erosions with superficial honey crusting, and is difficult to distinguish from a primary bacterial infection due to possible bacterial superinfection of the primary dermatophyte infection. Bacterial abscess should be considered in the differential diagnosis for a kerion.

33.6 Treatment

Tinea capitis requires treatment with oral antifungals to appropriately penetrate the hair shaft and eradicate disease. Griseofulvin and terbinafine are common treatment options. Azole antifungals are off-label therapies for tinea capitis. In a prospective, non-blinded, cross-sectional study, griseofulvin was found to be the most effective treatment option with a cure rate of 96 %. The cure rates for terbinafine and fluconazole were 88 and 84 %, repsectively [7]. Other studies have suggested that griseofulvin is the most efficacious for *Microsporum* spp. and terbinafine appears to the most efficacious for *T. Tonsurans* [8].

In the United States, griseofulvin, given at a dose of 20–25 mg/kg/day for 8 weeks, remains the mainstay of therapy with low risk of adverse effects. However, there are reports of resistance developing toward griseofulvin as well as issues with compliance due to the long duration of therapy. The main side effects of griseofulvin are gastrointestinal symptoms. Terbinafine is given at 250 mg daily for 4 weeks and is the mainstay of therapy in European countries. The shorter course makes this therapy more convenient, and it can be used in pregnancy. However, terbinafine can cause liver toxicity, and patients should be monitored for the occurrence of this rare side effect. Gastrointestinal side effects can also occur with terbinafine. Azoles are administered at 6–8 mg/kg/week. Caution must be taken with patients on multiple medications due to cytochrome P450 metabolism of azoles.

There have been debates over the management for kerions. Some argue in favor of intralesional corticosteroids to decrease the host inflammatory response. A recent retrospective study showed successful cure rates of kerions with oral antifungals alone and no evidence that intralesional corticosteroids help clear the infection [9]. There is also questionable data for advocating the use of prophylactic ketoconazole shampoo for kerions [10].

In addition to medical therapy, the individual's social settings should be considered. Reinfection can occur through carrier pets, clothing, bedding, toys, combs, and phones. In some infections, especially with *T. tonsurans*, some recommend treatment for all household members. Lastly, patient education and close follow-up should be stressed in every patient.

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