


Gray Patch Tinea Capitis Caused by *Microsporum canis*

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ABSTRACT

Background: Tinea capitis (TC) is the most common dermatophytosis of childhood. **Case**: A 3-year-old boy came with a three-week history of whitish rash and baldness. Physical examination found whitish, ill-defined patches and plaques with focal area of scaling and hyperkeratosis on the scalp (vertex region) and alopecia. Woods light examination revealed green fluorescence of infected hairs, and KOH prep revealed an ectothrix pattern of hair shaft invasion by fungal elements. Fungal growth by culture on Sabouraud dextrose agar with chloramphenicol and cycloheximide for 3 weeks found the growth of *M. canis*. The treatment wasgriseofulvin 1x375 mg and ketoconazole 2% shampoo twice weekly. Complete clinical clearance was obtained after 8 weeks.

Keywords: Griseofulvin, *Microsporum canis,* tinea capitis gray patch type

ABSTRAK

Latar Belakang: Tinea kapitis (TK) adalah dermatofitosis terbanyak pada anak. **Kasus**: Anak berusia 3 tahun dengan keluhan bercak putih dan botak di kepala sejak 3 minggu. Pemeriksaan fisik mendapatkan bercak keputihan, batas jelas dengan sisik tipis, dan hiperkeratosis di area vertex kepala dan alopesia tanpa pustul, erosi, indurasi, atau inflamasi berat. Pemeriksaan lampu Wood menunjukkan fluoresensi hijau terang dan pemeriksaan KOH menunjukkan pola *ectothrix* elemen jamur pada sampel batang rambut. Kultur jamur pada *Sabouraud dextrose agar* dengan kloramfenikol dan sikloheksimid selama 3 minggu ditemukan pertumbuhan jamur *Microsporum canis*. Terapi griseofulvin oral 1x375 mg dan shampo ketokonazol 2% dua kali seminggu. Pasien sembuh setelah pengobatan 8 minggu. **Febrina Dewi Pratiwi, Trisniartami Setyaningrum. Tinea Kapitis tipe *Gray Patch* Disebabkan *Microsporum canis***

Kata kunci: Griseofulvin, gray patch, *Microsporum canis,* tinea kapitis

INTRODUCTION

Tinea capitis (TC) or scalp ringworm is common in pediatric population with reported rates of infection ranging from 4% to 15%,[1] but is uncommon in adults.[2,3] Adult cases of *Trichophyton tonsurans* infection may be seen in adults with acquired immune deficiency syndrome (AIDS). The infection has a worldwide distribution.[1-3]

TC is a fungal infection of the skin and hair scalp characterized by scaling and patchy alopecia. A variety of dermatophytes may cause TC. These organisms may be anthropophilic (spread from humans, i.e., *T. tonsurans* and *T. violaceum*); zoophilic (spread from animals, i.e., *Microsporum canis* and *M. audouinii*); or geophilic (spread from soil). *T. tonsurans* is the most common etiology in the USA, while *M. canis* remains the most common cause of tinea capitis in Europe.[2-4] *T. rubrum* is rarely reported in countries with a temperate climate.[4] Except for two species, *Epidermophyton floccosum* and *T. concentricum*, all dermatophytes are able to invade hair. Infection of hair by dermatophytes follows 3 main patterns: ectothrix, endothrix, and favus.[5,6]

The clinical appearance is partly dependent on the responsible fungus, and other factors such as host immune response.[7] There is always hair loss with varying degrees of scaling and erythema.[8] Noninflammatory type is seen most commonly with anthropophilic organisms such as *M. audouinii* or *M. ferrugineum*. Alopecia may be imperceptible; in inflammatory cases there may be circumscribed erythematous scaly patches of nonscarring alopecia with breakage of hair ("gray patch" type).[9]

Black dot type is typically caused by anthropophilic endothrix organisms *T. tonsurans* and *T. violaceum*. Hair broke off at the level of the scalp, leave behind grouped black dots within patches of polygonal shaped alopecia with finger-like margins. Inflammatory type is generally more intense with zoophilic or geophilic fungi such as *M. canis*, *M. gypseum*, and *T. verrucosum*. Inflammation is the result of hypersensitivity reaction to the infection, ranges from follicular pustules to furunculosis or kerion. Intense inflammation may also result in scarring alopecia. This type is often associated with posterior cervical lymphadenopathy, which serves as a differentiating clinical feature of tinea capitis from other inflammatory scalp disorders.[8,9]

Several methods are available for identifying

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a TC infection.[10] The gold standard is fungal culture. Wood's light examination may be useful if the cause is an ectothrix organism. Demonstration of the fungus by potassium hydroxide wet-mount preparations of broken hairs or black dots, collected with a forceps, or skin scrapings from scaly areas may be useful. Microscopic examination of an infected hair will reveal tiny arthrospores surrounding the hair shaft in *Microsporum* infection (ectothrix infection) and chains of arthrospores within the hair shaft (endothrix infection) in *T. tonsurans* and *T. violaceum* infections.[11] The standard therapy for tinea capitis remains oral griseofulvin; depending on the specific pathogen identified, different treatment regimens may be employed.[12,13]

CASE REPORT

A 3 year-old Javanese child, came to the outpatient clinic of the Dermato-Venereology Department at Dr. Soetomo General Hospital Surabaya with baldness for 3 weeks. It appeared as a red patch accompanied with an itch, no pain and burning sensation; hair easily is broken and became grayish patch. The child has normal childhood development. No previous history of the same disease, no history of the same disease among family. The mother applied olive oil for several days but no improvement. No change of child's behavior since scalp eruption. The family had a cat that often slept next to the child during naps; the cat had never been checked by a veterinarian.

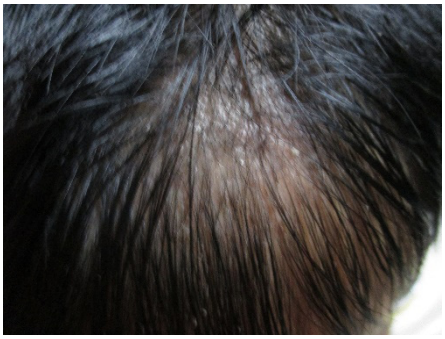

Figure 1. Child boy (3 years of age) presented with grayish, well-defined patch and thin plaques with scaling and focal hyperkeratosis on the scalp.

On general physical examination, a 3 year-old boy with respiratory rate 20/ minute, pulse rate 90/minute, and body temperature 37,4^{0}C, weight 13 kg. No signs of anemia, jaundice, cyanotic and respiratory distress. No enlargement lymph nodes in cervical area. No abnormality on his thorax, abdominal, and extremities.

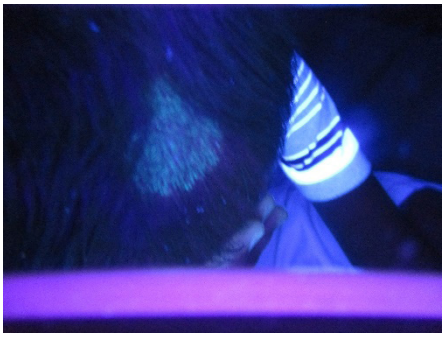

Figure 2. Woods light examination of scalp revealed bright fluorescence

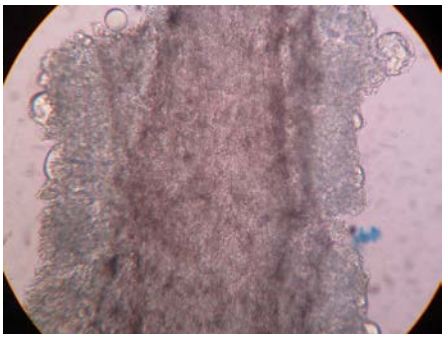

Figure 3.

Figure 4A.

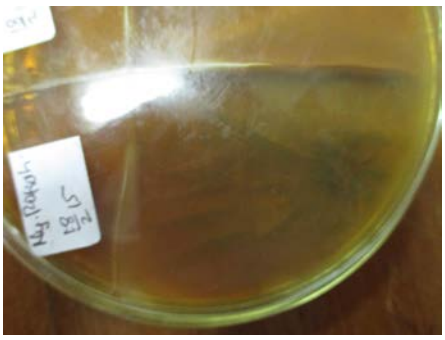

Figure 4B.

On dermatological examination, there was a grayish, well-defined patch, 3 cm diameter, with focal scaling and hyperkeratosis areas on the scalp, predominantly in vertex. No clinically noted alopecia (**Figure 1**). No areas of pustulation, erosion, induration, or boggy inflammation were noted on the scalp. No cutaneous abnormalities in other body parts.

The involved hair examination revealed bright green fluorescence with Wood's lamp (**Figure 2**). Samples of plucked hairs from the affected scalp region were examined by light microscopy (KOH prep). The sample was placed on object glass, followed by application of potassium hydroxide (KOH) 20% with heating. Scales and scalp hair samples were also cultured for fungi using Sabouraud dextrose agar with chloramphenicol and cycloheximide medium. Examination of the KOH prep revealed an actothrix pattern of hair shaft invasion by fungal elements with numerous spores located outside the surface of the hair shaft without long branched hyphae (**Figure 3**).

Fungal culture assessed at week 3 revealed positive growth showing flat, white to light yellow, coarsely hairy colonies with closely spaced radial grooves (**Figure 4A**) and yellow to reverse orange pigment (**Figure 4B**). Microscopic morphology features revealed numerous thick walled and echinulate spindle shaped macroconidia with terminal knob (**Figure 5**). It was consistent with *M. canis*.

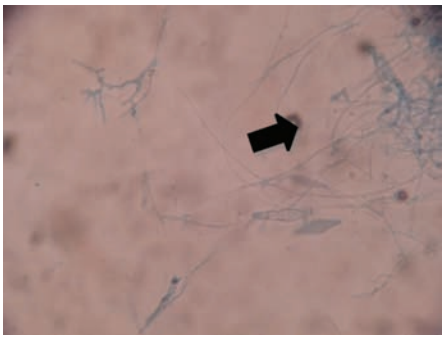

Figure 5. Microscopic morphology features revealed numerous thick walled and echinulate spindle shaped macroconidia with a terminal knob.

The child was treated with griseofulvin tablet 25mg/kg/day taken together with a lipid-contained meal. Ketoconazole 2% shampoo was used twice weekly; with instructions to avoid sharing combs, brushes, and towels and immediately get rid of the family cat or to be checked by a veterinarian.


After 2 weeks of treatment, the improvement was noted, and complete clinical clearance was obtained after 8 weeks (**Figure 6A, B, C**). An additional KOH prep obtained from the previously affected scalp, including some hairs in the region at the end of treatment (8 weeks) was negative for any fungal elements (**Figure 7**). No adverse effects from medication were reported by the mother.

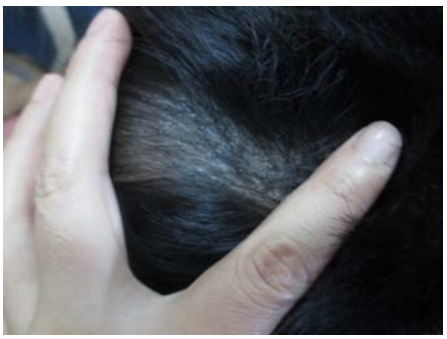

Figure 6A.

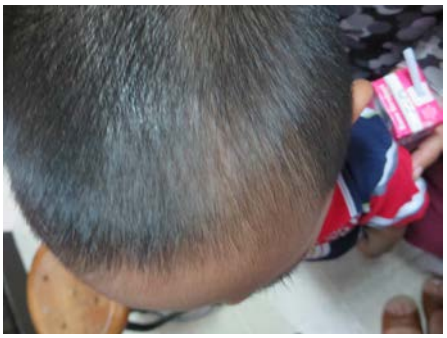

Figure 6B.

Figure 6C.

Figure 6A - 6C. After 4 weeks treatment

DISCUSSION

Tinea capitis (TC) is a fungal infection of the skin and hair of the scalp characterized by scaling and patchy alopecia.[5] TC is the most frequently seen among prepubescent children, especially those between the ages of 3 and 7 years, relatively rare among infants.[6] TC is uncommon in adults. The infection has a worldwide distribution.[7,8] The reason for increased resistance to tinea capitis after puberty is unknown, but may be related to a higher content of fungistatic fatty acids in the sebum of postpubertal individuals.

Dermatophytes have a short incubation period (generally 1-3 weeks) and infect boys more commonly. Predisposing factors for TC include large family size, crowded living conditions, and low socioeconomic class. In addition to transmission from other humans or animals, transmission via hairbrushes, combs, hats, and contaminated grooming instruments) is well documented.[6-8] This patient was 3 year-old Javanese boy and had a family cat that could be a potential source of infection.

Table 1. Causative species of tinea capitis[7]

Pathogens associated with clinical types of tinea capitis	
Inflammatory	■ *MIcrosporum audouinii* ■ *MIcrosporum Canis* ■ *MIcrosporum Gypseum* ■ *MIcrosporum Nanum* ■ *Trichophyton Interdigitale* ■ *Trichophyton Schoenleinii* ■ *Trichophyton Tonsurans* ■ *Trichophyton verrucosum*
Noninflammatory	■ *M. audouinii* ■ *M. canis* ■ *MIcrosporum ferrugineum* ■ *T. tonsurans*
Black dot	■ *T. tonsurans* ■ *T. violaceum*
Favus	■ *T. schoenleinii* ■ *Tricophyton violaceum* ■ *Tricophyton mentagrophytes*

The clinical manifestations of TC vary, depending on the causative species and other factors such as the host immune response.[9] Although the clinical appearance is in part dependent on the fungus responsible for the infection, there is always hair loss, with varying degrees of scaling and erythema.[7,8] A single dermatophyte may have more than one presentation (**Table 1**)

There are four clinical types of TC, including noninflammatory type, inflammatory type, black dot type, and favus. Examination of involved hair bearing areas, such as the scalp or beard, with a Wood's lamp (365 nm) may reveal pteridine fluorescence of hair infected with particular fungal pathogens. Hairs that fluoresce should be selected for further examination, including culture. While ectothrix organisms *M. canis* and *M. audouinii* will fluoresce on Wood's light examination, the endothrix organism *T. tonsurans* will not fluoresce.[11,12]

Diagnosis based only on presenting clinical symptoms is often difficult, and potentially misdiagnosed. If TC infection is suspected, both KOH prep and fungal culture are important for diagnosis. Fungal culture is the gold standard.[12,13]

In this patient, the presentation was whitish, well-defined patches and hyperkeratotic plaque of alopecia. Remaining hair shafts and scales exhibited a bright green fluorescence when examined with a Wood's lamp. To confirm the diagnosis, we performed a light microscopy examination by KOH prep and fungal culture. Samples of affected hair were placed on a microscope, and we added 20 % KOH with heating. Microscopic examination presenting the ectothrix pattern of hair invasion with multiple round spores lined up on the outer surface of the hair shaft was considered to be a positive result.

Table 2. Treatment of tinea capitis[7]

Disease	topical treatment	Systemis treatment
Tinea capitis	■ Only as adjuvant ■ Selenium sulfide 1% or 2,5% ■ Zinc pyrithione 1% or 2% ■ Povidone iodine 2,5% ■ Ketoconazole 2%	Adults: ■ Griseofulvin, 20-25 mg/kg/day x 6-8 weeks ■ Terbinafine, 250 mg/kg/day x 2-8 weeks ■ Itraconazole, 5 mg/kg/day x 2-4 weeks ■ Fluconazole, 6 mg/kg/day x 3 weeks Children: ■ Terbinafine, 6-6 mg/kg/day x 2-8 weeks All others are the same

Tabel 3. Choice of drug according to organism isolated[13]

Trichophyton tonsurans	Terbinafine
Trichophyton violaceum, soudanense	Terbinafine
Microsporum canis	Griseofulvin or itraconazole
Microsporum audouinii	Griseofulvin or itraconazole

For culture, the hair samples were placed on Sabouraud dextrose agar with chloramphenicol and cycloheximide. Before obtaining a sample for culture, the area was cleaned with alcohol to avoid cross contamination with bacteria. Then the media was incubated in a warm, moist environment at 28^{0}C and examined regularly to detect fungal growth. Fungal growth needed 3 weeks in this case. This positive culture was subsequently examined for specific identification of genera and species. Specification of superficial fungi is based on macroscopic, microscopic and metabolic characteristics of the organism. In this case, colony morphology of culture revealed flat, white to light yellow, coarsely hairy, with closely spaced radial grooves. Yellow to reverse orange pigment (**Figure 4**). On microscopic morphology we found numerous thick walled and echinulate spindle shaped macroconidia (**Figure 5**). The organism was identified as *M. canis*.

The differential diagnosis of TC includes seborrheic dermatitis, psoriasis, alopecia areata.[14-16] Seborrheic dermatitis is characteristically found in regions of body with high concentrations of sebaceous follicles and active sebaceous glands including the face, scalp, ears, upper trunk, and flexures (inguinal, inframammary, and axillary), presents with pink to erythematous, superficial patches and plaques with yellow, branny (small husklike) and sometimes greasy scale. Psoriasis is a chronic disorder, presents with erythematous plaques and white scaly surface, with less scale in intertriginous sites, while alopecia areata has a typical clinical picture. A sudden (overnight or several days) appearance of one or more round or oval well-circumscribed non-scarring, clearly defined patches of hair loss. Alopecia areata is thought to be a tissue-restricted autoimmune disorder with an attack by T lymphocytes on follicle after immune privilege.[15-17]

Management of TC involves more than simply selecting the right medication (**Table 2**). Consideration of the specific causative organism with regard to the selection of therapy, daily dose, and anticipated duration of treatment, incorporation of adjunctive topical antifungal therapy, and handling of fomites (inanimate objects or substance, such as clothing, furniture), which may promote transmission to others are significant aspects.[18,19] Griseofulvin remains the gold standard of oral antifungal treatment for TC in the United States and is approved for this indication by the FDA.

Treatment regimen varies according to the dermatophyte involved (**Table 3**). A prolonged course or a change of agent may be required in cases of treatment failures, or if an unexpected fungus is identified on culture.[13]

In this case, microscopic KOH examination was repeated after 8 weeks of oral griseofulvin treatment to confirm that the infection had resolved. Repeat KOH examination was negative and oral griseofulvin was discontinued.

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