Tinea Capitis Favus-Like Appearance: Problem of Diagnosis

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ABSTRACT
Background: Tinea capitis (TC) is an infection of scalp, hair follicles, and the surrounding skin, caused by dermatophyte fungi. Favus, a chronic inflammatory tinea capitis typically seen in *Trichophyton schoenleinii* infection. Favus is characterized by 'scutula'. Favus may result in cicatricial alopecia. **Purpose:** To understand the clinical manifestation and management of tinea capitis. **Case:** A girl, 8 year-old, 18 kg, with thick crust located at the center of the head since 3 months, became spreading overtime. There were itchy sensation, no fever, no pain. There was history of an itchy red patch on her neck which diminished with topical antifungal. Dermatological examination revealed multiple thick brown-yellow crust sharply marginated, there were erosion and alopecia area beneath the crust. There were no sign of inflammation, no pustule, no enlargement of cervical and occipital lymphnodes on palpation. **Discussion:** Wood's light examination showed no fluorescence and potassium hydroxide (KOH) showed no spores. Result of fungal culture was no colony growth. Patient was given griseofulvin 20 mg/kg body weight/day, wet dressing, and ketoconazole 2% shampoo twice weekly. Good clinical result shown after 10 weeks. **Conclusion:** Diagnosis of TC established based on history taking, clinical findings, Wood's light examination, KOH preparation, fungal culture. A negative culture may arise because antifungal treatment had been used prior to collection of the specimen. Griseofulvin still became the drug of choice for tinea capitis even there are new generations of antifungal.

Key words: diagnosis, favus-like, griseofulvin, tinea capitis.

ABSTRAK
Latar Belakang: Tinea kapitis adalah infeksi pada kulit kepala, folikel rambut, dan kulit disekitarnya yang disebabkan oleh jamur dermatofit. Favus merupakan infeksi kronis, dan merupakan tinea kapitis inflamasi yang biasanya nampak pada infeksi *Trichophyton schoenleinii.* Ciri khas favus adalah lesi 'scutula'. Favus dapat mengakibatkan alopesia sikatrikalis. **Tujuan:** Mengetahui gambaran klinis dan tatalaksana tinea kapitis. **Kasus:** Anak perempuan, 8 tahun, 18 kg, keluhan krusta tebal terbentuk pada bagian tengah kepala sejak tiga bulan sebelum ke rumah sakit dan menyebar seiring waktu. Gatal, tanpa ada nyeri dan demam. Riwayat bercak merah gatal pada leher dan menghilang dengan antijamur topikal. Pemeriksaan dermatologi menunjukkan krusta tebal kuning kecoklatan dengan batas jelas, apabila krusta diangkat nampak erosi dan area alopesia dibawahnya, tidak ada radang dan *pus*, tidak didapatkan pembesaran kelenjar limfa leher dan *occipital*. **Pembahasan:** Pemeriksaan lampu Wood tidak terdapat *fluorescence*, pemeriksaan potasium hidroksida (KOH) juga tidak ditemukan spora. Kultur jamur tidak didapatkan pertumbuhan koloni. Pasien diberikan terapi griseofulvin 20 mg/kg berat badan/hari, kompres basah, serta sampo ketokonazol 2% dua kali dalam seminggu. Perbaikan klinis nampak setelah 10 minggu. **Simpulan:** Diagnosis tinea kapitis dapat ditegakkan berdasarkan anamnesis, gambaran klinis, pemeriksaan lampu Wood, KOH, serta kultur jamur. Kultur jamur yang negatif dapat terjadi karena penggunaan antijamur sebelum pengambilan spesimen. Griseofulvin masih menjadi obat pilihan untuk tinea kapitis meskipun terdapat antijamur generasi lebih baru.

Kata kunci: diagnosis, *favus-like*, griseofulvin**,** tinea kapitis.

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INTRODUCTION

Tinea capitis (TC) is an infection of scalp, hair follicles, and the surrounding skin caused by dermatophyte fungi, usually species in the genera *Microsporum* and *Trichophyton*. Tinea capitis continues to be predominantly a disorder of prepubertal children.[1] These infections frequently spread among family members and classmates. Certain hairdressing practices such as shaving of the scalp, plaiting or the use of hair oils may promote disease transmission, but their precise role remains the subject of study. New cases of TC in Mycology Division in Dermatology and Venereology Outpatient Clinic of Dr. Soetomo General Hospital Surabaya (2001-2006) are 0,31-1,55% compared to other dermatomycosis. Most of the case affect children <14 year-old (93,33%), boys are higher (54,5%) than girls (45,5%), kerion is the most common type (62,5%),

gray patch 37,5%, and no black dot and favus type. Causative species include *Microsporum gypseum, Microsporum ferrugineum, Trichophyton mentagrophytes*.[2,3]

An accurate diagnosis remains a vital component of management. Clinicians that are unfamiliar with this condition often misdiagnose tinea capitis, especially inflammatory variants such as boggy kerions, leading to delay in diagnosis and inappropriate management. The clinical appearance of tinea capitis is highly variable, depends on the causative organism, type of hair invasion and degree of host inflammatory response. Common features are patchy hair loss with varying degrees of scaling and erythema. However, the clinical signs may be subtle and diagnosis can be challenging. A number of clinical patterns exist.[1]

On the basis of the type of hair invasion, dermatophytes are also classified as endothrix, ectothrix, or favus. In endothrix infection the fungus grows completely within the hair shaft, the hyphaes are converted to arthroconidia (spores) within the hair while the cuticle surface of the hair remains intact. In ectothrix infection hair invasion develops in a manner similar to endothrix except that the hyphaes destroy the hair cuticle and grow around the exterior of the hair shaft. Arthroconidia may develop both within and outside the hair shaft. Elongated hyphaes, parallel to the long axis of the hair, persist within the hair.[4]

Favus or tinea favosa is a chronic inflammatory dermatophyte infection of the scalp and, less commonly, nor the glabrous skin and nails. It has an insidious course, usually lacking an acute phase, and often the patient does not feel any need to consult a physician. The classic favus lesion is the 'scutulum', a concave, cup-shaped yellow crust on the scalp and glabrous skin that is associated with severe alopecia. These keratotic crusts contain fungal hyphae and can be highly infectious. The scutula form dense plaques, each composed of hyphae, neutrophils, and epidermis, secondary bacterial infections often occur in the plaques. The scutulum develops in a hair follicle, with the hair shaft in the center of the raised lesion. The scutula gradually increase in size, and adjacent scutula tend to form confluent masses of crusts. In addition to the typical scutular form of favus, atypical tinea favosa makes up about 5% of the cases. Pityroides, psoriasiform, follicular, and impetiginous forms have been observed both on scalps and on glabrous skin.[5] Both kerion celsi and favus can cause permanent alopecia.[6]

Although the clinical diagnosis of tinea capitis is often relatively accurate, when considered, laboratory investigations to confirm the diagnosis are advisable to isolate the causal organism and direct the choice of systemic therapy.[7] All specimens from cases of tinea capitis should be processed for microscopy and culture when possible, and the causal agent fully identified where isolated.[1]

CASE REPORT

A girl, 8 year-old, 18 kg, came with a complaint thick crust located at the center of her head since three months before visiting to the hospital and became spread overtime. There were itchy sensation, no fever, no pain. Firstly it was just an itchy red macule covered with thin scales, then became covered with thick crusts. There was a complaint of hair loss on the crusted area. The area of crusting became larger and spread overtime and the crust become thicker. History of an itchy red patch on her neck and had healed with an anti-fungal cream. She used to play with cats surround her house. This was the first time she got this disease. No family member was suffered from the same disease. No classmates were suffered from the same disease.

Patient for the last three months have had a numerous doctor visit and got various medication, both topical and oral. From the first doctor visit she got 3 types of ointment one of them was miconazole cream, and ketoconazole shampoo for 2 weeks. No clinical improvement was achieved hence the crust become thicker then spread to another area of the scalp. The second doctor visit she got oral ketoconazole 200 mg one times a day consumed for 2 weeks and continued the ketoconazole shampoo but also did not achieve a good clinical response. The third visit to dermatologist she got oral erythromycin and referred to Dr. Soetomo hospital for further management and laboratory examination.

General examination revealed a compos mentis condition, look well with no sign of anemic, icterus, cyanotic, or respiratory distress. The blood presure was 100/70 mmHg, pulse rate 80 times per minute, respiratory rate 20 times per minute, and body temperature was 36,5ºC. There was no abnormality on thorax and abdominal examination.

Dermatological examination found multiple thick brown-yellow crust sharply marginated, if the crust scratched there were erosion and alopecia area beneath, no inflammation, no pustule, with no enlargement of cervical and occipital lymphnodes on palpation. The remainder of the physical examination revealed no cutaneous abnormalities noted other than the scalp lesions. Blood and urine examination when she admitted were within normal limit. Haemoglobin was 12,3 g/dL, white blood cell 7,3 x 10^3/µL. From potassium hydroxide examination found no spores

both outside and inside the hair shaft (Figure 2). On Wood lamp examination found no fluorescence (Figure 3). The result of fungal culture on Sabouraud's Dextrose Agar (SDA) after six weeks showed no colony growth (Figure 4).

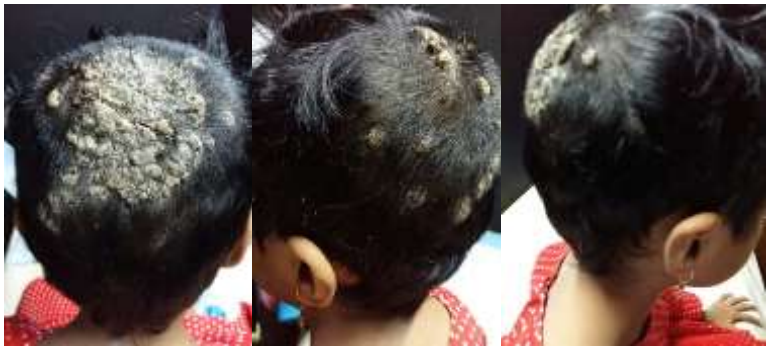

Figure 1. Tinea capitis favus-like appearance.

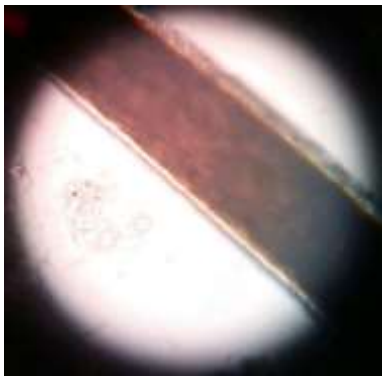

Figure 2. Potassium hydroxide 20% examination, there are no spore both outside and inside the hair shaft, magnification 40x.

Figure 3. No fluorescence on Wood's lamp examination.

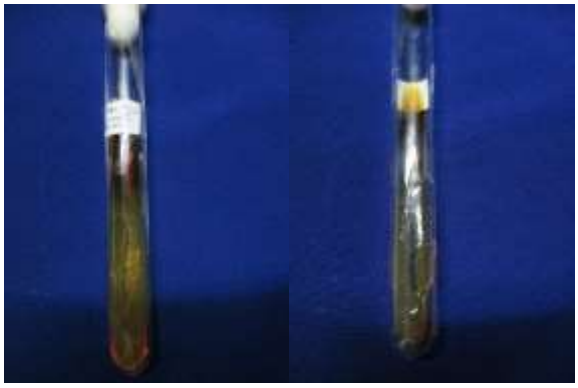

Figure 4. Fungal culture on Sabouroud's dextrose agar (SDA) showed no colony growth.

The patient was treated with griseofulvin dose 20 mg/kg body weight/day, wet dressing with normal saline and ketoconazole shampoo twice weekly. After one week of treatment the crust decreased with no new crusted area. After six weeks of treatment, there was no crust on the lesion, only thin scales covered the alopecia area. Atrophic alopecia was noted as well in occipital area and after ten weeks of treatment the atrophic alopecia area was still persisted (Figure 5).

Figure 5. Lesion after 10 weeks of treatment, A. atrophic alopecia area on occipital area. B,C, hair growth on alopecia area before, left, and right lateral side.

DISCUSSION

Tinea capitis (TC) is a superficial scalp dermatophyte infection, particularly seen during prepubertal children. TC is most commonly seen at the age of 5–10 years. Epidemiologic studies from North India, Bosnia, Tunisia, Spain, and Turkey showed that patients with TC are usually younger than 10 year-old. A retrospective study in Turkey show that TC was more common in patients less than 10 year-old, and the age groups of 1–5 years and 6–9 year had similar frequencies.[8] Nevertheless, this infection occurs across age and sex groups with high occurrence being reported among males. Tinea infections has remained a significant public health problem with poor hygiene, sharing of fomites, overcrowding and low socioeconomic being among some of the factors that predisposes populations to infections.[9] This is a case of TC in a girl, 8 year-old, with predisposing factors of disease because of living in crowded area with low socioeconomic condition. The source of contagious of this case could be from the cats she often plays with.

Four clinical infection patterns have been reported. Different clinical presentation may arise depending on the causative organism, the type of hair invasion, and the specific host T-lymphocyte inflammatory response.[10]

Non-inflammatory black dot pattern is clinically characterized by well demarcated areas of hair loss. Fungal arthrospores proliferate inside the hair shafts, weakening them. Hairs break off at or below the scalp surface, giving the characteristic appearance of black dots on the alopecic patch. Cell-mediated immunity to fungal antigen skin test is usually negative and adenopathy is often absent.

Non-inflammatory seborrhoeic dermatitis type is a diffuse or patchy, fine, white, adherent scale affecting the scalp. This is the most difficult to diagnose because it resembles dandruff and only one third of patients have a positive potassium hydroxide examination.

In inflammatory tinea capitis (Kerion type), there are one or multiple tender, inflammed, alopecic nodules with pustules on their surface. Fever, occipital adenopathy, leukocytosis, and even a diffuse, morbilliform rash may occur. Most patients have a positive skin test to fungal antigen, suggesting that the patient's immune response may account for the intense inflammation. Favus is a rare type of inflammatory TC characterized by typical honey colored, cup-shaped, follicular crusts called scutula. Kerion celsi and favus have the potential to cause scarring and permanent alopecia.[11,13]

Favus can be classified into three main stages according to infection severity: (1) First stage shows only erythema of the scalp primarily around follicles. The hairs are not loose or broken, (2) Second stage shows scutula along with the beginning of hair loss, (3) Third stage is the most severe stage, large areas of the scalp are involved, with at least one-third of the scalp affected. There is extensive hair loss, atrophy, and scarring. Formation of new scutula at the periphery of plaques is common.[5]

In this case the clinical appearance of scalp lesions showed a favus-like type with multiple yellow-brown crust attached to the scalp, with hair pierced the crust. There were no sign of inflammation

such as pain, fever, and enlargement of lymphnode. The clinical stage of this patient also resembled the favus type lesion with the erythema of the scalp which latter become the multiple thick brown crust, with one-third area of the scalp affected, hair loss, and athropic alopecia.

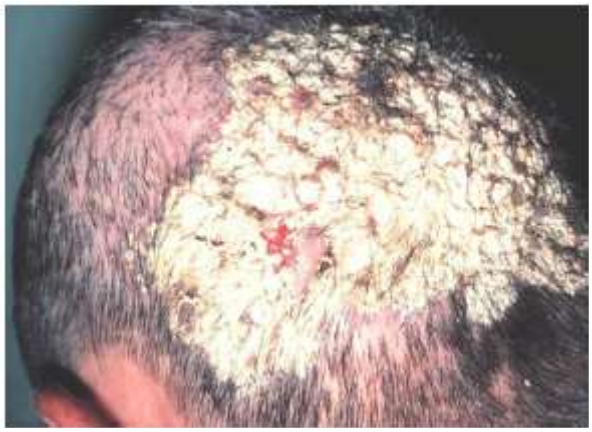

Figure 6. Tinea favosa of the scalp with yellowish-white scutula of 20-year-old patient (Courtesy of Prof Dr H. R. Memisoglu).[5]

There are two accepted methods for confirming tinea capitis: microscopic analysis of scalp debris, and fungal culture. Wood's lamp examination could be useful in some case which the causative agent shows fluorescence. It will be negative in cases of tinea capitis involving *Tricophyton* species. In this case the Wood's lamp examination did not show any fluorescence.

Potassium hydroxide 20% examination from plucked hair stubs using sterile forceps to include the hair roots. The presence of fungal element (hyphae and/or spores) within and/or around hairshaft under microscope magnification was considered to be a positive test. Although a microscopic examination of potassium hydroxide (KOH) preparations of clinical skin scraping or hair affords a rapid diagnostic test, some limitations have been noted. Some clinicians have little experience interpreting KOH preparations, thus reducing the value of the test. Artifacts including heterologous fibers, fat globules, and environmental contaminants may result in false positives. A false-negative KOH preparation may be the result of early or inflammatory lesions, or it may be observed in patients partially treated with topical agents. Thus a negative KOH result cannot rule out the diagnosis of tinea capitis.[14] In this case we did not found spores outside and inside the hair shafts. A negative result can be observed because of topical antifungal treatments used before.

All specimens should be cultured on Sabouraud agar with at least one agar plate containing cycloheximide to inhibit non-dermatophyte mould growth. Plates should be incubated for at least 2 weeks.[1] The two most common media are Sabouraud's agar and Mycobiotic agar containing chloramphenicol and cycloheximide to suppress the growth of bacterial saprophytic contamination. Dermatophyte test medium (DTM) is similar to Mycobiotic agar but contains a color indicator that changes from yellow to red in the presence of dermatophyte fungi.[4] Where exposure to cattle is documented and an infection caused by *T. verrucosum* is suspected, plates should be incubated for up to 3 weeks and examined very carefully at the end of this period for the presence of the slow-growing and inconspicuous colonies of this species. Any dermatophytes growing should be identified and reported.[1] The time required for growth of dermatophytes and the identification of genus and species may be prolonged. Although detailed information concerning the infecting dermatophyte is valuable for epidemiologic purposes, determination of the genus and species is not necessary because treatment options for all of the dermatophytic molds that are associated with tinea capitis require oral therapy and are standard.[14] We performed culture on sabouraud's agar and until 6 weeks of inoculation showed no colony growth. A negative culture in this case may arise because of antifungal treatment had been used prior to collection of the specimen.

The future direction for laboratory diagnosis of superficial fungal infections is likely to be a molecular one, and diagnosis of tinea capitis is unlikely to be an exception. Real-time polymerase chain reaction (PCR) tests and PCR reverse-line blot assays, designed for dermatophyte infections, have generally performed well on clinical specimens including hair from patients with tinea capitis.[15,16] A method for the

detection of *T. tonsurans* from hairbrushes has also been described.[17] Larger scale comparative studies of PCR, microscopy, and culture are required to determine whether DNA detection of dermatophytes will improve the diagnosis of tinea capitis.[1] We did not perform the PCR test in this case because the test did not available in our laboratory.

It is reasonable to begin treatment on the basis of one or more cardinal signs, while awaiting confirmatory mycology (strength of recommendation B; level of evidence 2++). Clear evidence has now emerged to show that the optimal treatment regimen varies according to the dermatophyte involved.[18] Treatment protocols should therefore reflect local epidemiology and be based on the most likely culprit organism[19] (strength of recommendation A; level of evidence 1+). 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.[1]

Griseofulvin is known as the gold standard therapy for tinea capitis, its efficacy seems to have decreased over the years, requiring larger doses and longer duration of treatment. The reduction in the efficacy of griseofulvin has been attributed to various factors, including changes in patterns of epidemiology, and fungal genetic mutations resulting in decreased susceptibility. Current treatment requires a 6-8 weeks course of oral griseofulvin given once daily.[20] The standard licensed treatment protocol for those aged >1 month is 1 gram in children weighing >50 kg, or 15-20 mg/kg daily in single or divided doses for 6-8 weeks if <50 kg. Taking the drug with fatty food may increase absorption and improve bioavailability. Dosage recommendations vary according to the type of formulation used and how easily it is absorbed. It may be necessary to use doses up to 25 mg/kg daily for more prolonged periods in resistant cases.[1]

Adjunctive topical therapies such as selenium sulphide, zinc pyrithione, povidone iodide or ketoconazole shampoos as well as fungicidal creams or lotions have been shown to decrease the carriage of viable spores responsible for the disease contagion and reinfection and may shorten the cure rate with oral antifungal. The shampoo should be applied to the scalp and hair for 5 minutes twice-weekly for 2-4 weeks or three times weekly until the patient is clinically and mycologically cured.[4]

In this case griseofulvin given with dose 20 mg/kg body weight/day, wet dressing with normal saline and oleum coccos to help removing the crusts and ketoconazole shampoo twice weekly were giving good clinical outcome shown after ten weeks.

Recent studies have investigated the use of newer oral antifungal agents including terbinafine, fluconazole, and itraconazole in the treatment of tinea capitis. Potential advantages over griseofulvin include better tolerability, improved efficacy as well as a shorter treatment course with associated cost savings and improved compliance. During the last decade several studies have been published suggesting various dosages and regimes of treatment in patients of tinea capitis.[20]

There are number of options for treating tinea capitis, and it seems that these options are unchanged over years, in particular favus. In favus, resolution of infection has been accomplished with the long-term use of some drugs. Clearing away the scalp debris, removing the crusts, and general improvement of scalp hygiene and aid in clinical management are essential in treating this infection. It is also important to became the outcome depends to some extent on the stage at which the disease is arrested.[5]

Although the potential risk of transmission of infection to unaffected classmates has led some authorities to recommend exclusion from school, most experts consider this impractical and suggest that children receiving appropriate systemic and adjunctive topical therapy should be allowed to attend school or nursery.[1]

Regardless of the clinical form, lesions evolve in all cases into ultimate scarring alopecia, which imposes an early diagnostic and therapeutic management. Indeed, if treated at an early stage of evolution, tinea favosa will cured without sequelae.[21] However, if the treatment is late established it will not prevent the ultimate scarring alopecia, which was the case of our patient. After 10 weeks of treatment the atrophic alopecia area was noted on occipital area.

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