Tinea Capitis in Adolescent: A Case Report

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ABSTRACT

Background: Tinea capitis is a dermatophyte infection of the scalp, eyebrows, and eyelashes with a propensity for attacking hair shafts and follicles, commonly affect children. The treatment remains the same between child, adolescent and adult **Purpose:** To evaluate the clinical manifestation, species causing agent, and management of tinea capitis. **Case:** An adolescent woman, 16 years old, body weight 33 kg with primary amennorhea came to Dermatovenereology Outpatient Clinic Dr. Soetomo Hospital because of baldness on her head since 3 weeks before admission. At first the lession was appeared as an itchy redness patches covered with thin scale followed by hairs turning into grey, lusterless and easily to pulled out leaving a patch of baldness. Dermatological examination demonstrated a well defined 10 cm x 10 cm alopecia with mild erythematous plaque covered with thin scale located in the parieto-occipitalis area. Wood's lamp examination revealed bright green fluorescence. Potassium hydroxide examination showed ectothrix spores. The positive result of fungal culture on Sabouraud's Dextrose Agar (SDA) was identified as *Microsporum audouinii.* Patient was diagnosed with tinea capitis greypatch type, and was treated with oral griseofulvin 125 mg microsize 2x3 tablets per day and ketoconazole 2% shampoo once a day. At 6 weeks of follow up, the lesion of her scalp was improved, the itchy was decreased, Wood's Lamp and Potassium hydroxide examination were negative. **Discussion:** In patient with amenorrhea, the progesterone level was low, the sebum production was decreased, as the consequence free fatty acid component which has fungistatic and fungisidal effect also low and increase the risk of tinea capitis. Griseofulvin remains the mainstay of treatment for tinea capitis caused by *Microsporum audounii*. **Conclusions:** The wood's lamp can detect *Microsporum* sp. infection with its greenish fluorescence. Definitive diagnosis and exact identification of the causative organism of tinea capitis could be determined by culture.

Key words: tinea capitis, *greypatch*, adolescent, *Microsporum audouinii* infection, griseofulvin.

ABSTRAK

Latar Belakang: Tinea kapitis adalah infeksi dermatofita pada kulit kepala, alis dan bulu mata yang cenderung menyerang rambut dan folikel, umumnya pada anak. Pada remaja dapat diberikan terapi sesuai terapi standar tinea kapitis **Tujuan:** Mengetahui gambaran klinis, spesies penyebab, dan penatalaksanaan tinea kapitis. **Kasus:** Remaja wanita, 16 tahun, berat badan 33 kg dengan amenore primer, datang ke Poli Kulit dan Kelamin RSUD Dr. Soetomo Surabaya karena kebotakan di kepalanya sejak 3 minggu sebelumnya. Awalnya berupa bercak kemerahan, gatal, tertutup sisik tipis. Rambut berubah menjadi abu-abu, kusam, mudah rontok sehingga menyebabkan kebotakan. Pemeriksaan dermatologis menunjukkan adanya alopesia diameter 10 cm x 10 cm dengan plak eritematosa ringan tertutup skuama tipis di daerah parieto-osipitalis. Pemeriksaan lampu Wood menunjukkan fluoresensi hijau terang. Pemeriksaan KOH menunjukkan adanya spora ektotrik. Hasil kultur Sabouraud Dextrose Agar (SDA) positif dan diidentifikasi sebagai *Microsporum audouinii*. Penderita didiagnosis dengan tinea kapitis tipe *greypatch*, diberikan griseofulvin 125 mg tablet mikron 2x3 per hari dan sampo ketokonazol 2% sehari sekali. Pada *follow-up* minggu ke-6, lesi membaik, gatal berkurang, pemeriksaan lampu Wood dan KOH memberikan hasil negatif. **Pembahasan:** Pada pasien ini, terdapat amenore primer, dimana kadar hormon progesteron rendah menyebabkan berkurangnya produksi sebum, sehingga komponen *free fatty acid* yang berfungsi fungistatik dan fungisidal juga rendah dan meningkatkan risiko tinea kapitis. Griseofulvin merupakan terapi pilihan untuk kasus tinea kapitis yang disebabkan oleh spesies *Microsporum audouinii.* **Simpulan:** Lampu Wood dapat digunakan untuk mendeteksi infeksi akibat *Microsporum* sp. yang akan memberikan fluoresensi hijau terang. Diagnosis pasti dan identifikasi pasti organisme penyebab tinea kapitis dapat dilakukan dengan kultur.

Kata kunci: tinea capitis, *greypatch*, remaja, infeksi *Microsporum audouinii*, griseofulvin.

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INTRODUCTION

Tinea capitis is a dermatophyte infection of the scalp, eyebrows, and eyelashes with a propensity for attacking hair shafts and follicles.[1,2] The disease is a form of dermatophytosis which are classified into three genera namely *Tricophyton, Microsporum,* and *Epidermophyton.* Tinea capitis predominantly caused by *Tricophyton* or *Microsporum* species.[1,2,3]

The incidence of tinea capitis varies according to sex, but the degree of variation depends on the microorganism. Tinea capitis affects primarily pre-pubertal children between 6 and 10 years. When the etiological agent is *Microsporum audouinii*, the ratio between male and female is 5:1; with *M. canis*, the ratio varies considerably, but the infection in boys is usually higher. *Trichophyton* infections of the scalp affect girls and boys equally. Although most commonly seen in prepubescent children, tinea capitis can occur in adults. In adults, women are infected more frequently than men. There is no sufficient data about tinea capitis prevalence in adult. Transmission is higher in person with lack of hygiene, overcrowding and low socioeconomic status.[1,4-9] The incidence of new case of tinea capitis in 2001 – 2006 compared to dermatomycosis new cases in Dermato-Venereology Outpatient Clinic Dr. Soetomo Hospital Surabaya was 0.31% - 1.55%. The incidence was greater in children under 14 years old which was 93.33% of the patients, whereas the percentage of the boys were higher (54.5%) than the girls (45.5%). Kerion was most commonly found in 62,5% of the patients compared to gray patch type (37.5%). Black dot type was not found. [10]

A wide variety of tinea capitis presentation have been discribed depends on the type of organism, the type of hair invasion, level of host resistance and the degree of inflammatory host response. The major clinical types of tinea capitis include non inflammatory or grey patch, inflammatory type, black dot, and favus. A prominent cervical or occipital lymphadenopathy may occur in all types of tinea capitis.[1,2,3] 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 hyphae 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 hyphae destroy the hair cuticle and grow around the exterior of the hair shaft. Arthroconidia may develop both within and outside the hair shaft. Elongated hyphae, parallel to the long axis of the hair, persist within the hair. Favus is characterized by production of hyphae, which are parallel to the long axis of the hair shaft. When the hyphae degenerate, long tunnels are left within the hair shaft.[1,2,5]

If tinea capitis is suspected, specimens should be taken to confirm the diagnosis as systemic therapy will be required. Positive microscopy (when the hair or scales are seen to be invaded by spores or hyphae) confirms the diagnosis and allows treatment to commence at once. Culture allows accurate identification of the organism involved, and this may alter the treatment schedule. Culture is more sensitive than microscopy; results may be positive even when microscopy is negative, but may take up to 4 weeks to become available. In addition, fluoresence patterns under Wood's light examination may support a clinical suspicion. This is useful for certain ectothrix infections, e.g. those caused by *M. canis, M. rivalieri* and *M. audouinii*, which cause the hair to fluorescense bright green.[11,12]

The aim of treatment is to achieve a clinical and mycological cure as quickly as possible. Oral antifungal therapy is generally needed to penetrate the hair follicle, such as griseofulvin, terbinafine, itraconazole, fluconazole, and ketoconazole. Griseofulvin is fungistatic, and inhibits nucleic acid synthesis, arresting cell division at metaphase and impairing fungal cell wall synthesis. It is also antiinflammatory. Terbinafine acts on fungal cell membranes and is fungicidal. Itraconazole exhibits both fungistatic and fungicidal activity depending on the concentration of drug in the tissues, but like other azoles, the primary mode of action is fungistatic, through depletion of cell membrane ergosterol, which interferes with membrane permeability. Fluconazole and ketoconazole have occasionally been assessed for tinea capitis but its use has mainly been limited by side effects. [13]

CASE REPORT

A 16 years old adolescent Javanese girl, body weight 33 kg, came to Dermato-Venereology Outpatient Clinic Dr. Soetomo Hospital Surabaya on March, 14th 2016 with chief complain baldness on her head since 3 weeks before admission. At first the lession was appeared as an itchy redness patches accompanied with papule and covered with thin scales followed by hairs turning into grey, lusterless and easily to break off leaving a patch of baldness on her scalp. Initially the baldness was small, measuring about 4 cm in diameter and increased in size as the hairs easily to break off. There was no history of prior trauma to the site. She often plays with kitten around her house. No family member, no neighbours, classmates nor any other close contacts suffered from

the same disease. Her brother had applied topical ointment from general practitioner which she forgot the name but as there was no clinical improvement.

Based on general physical examination on the first day admission, she had diagnosed with rheumatic heart disease since 8 years old and she routinely visit cardiology department, with therapy ramipril and bisoprolol. Respiratory rate 20 times per minute, pulse rate 80 times per minute and body temperature 36.4 C. There was no sign of anemic, icterus, cyanotic or respiratory distress. No abnormality on abdominal examination and no enlargement of lymphnode. She has not have a menarche yet.

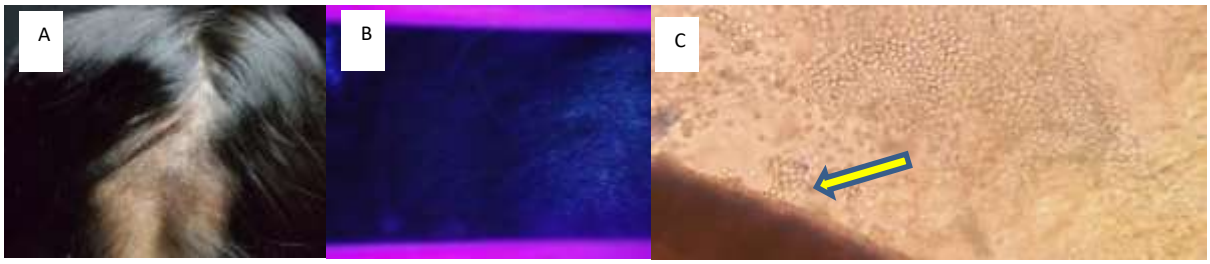

Figure 1. (A) Tinea capitis gray patch type, there was an alopecia which hairs were greyish in colour, lusterless and easily pulled out of the hair follicle. (B) Wood's lamp examination, there was bright green fluorescence. (C) Pottasium hydroxide 20% examination, there were spores outside the hair shaft, magnification 40x (arrow sign).

Laboratory examination revealed that haemoglobin was 13,4 g/dL, and white blood count was $9.63x10^3/\mu L$. The result of urinary test was normal. Serum glutamic oxaloacetic transaminase (SGOT) was 34 U/L, serum glutamic pyruvate transaminase (SGPT) was 28 U/L. Dermatological examination demonstrated a well defined 10 cm x 10 cm alopecia based on mild erythematous plaque covered with thin scale located in the parieto-occipitalis area. The hair was greyish in colour, lusterless and easily pulled out of the hair follicle (Figure 1a). Wood's lamp examination revealed bright green fluorescence (Figure 1b). Samples were promptly collected at day 1 for direct microscopic examination in a 20% solution of potassium hydroxide and subsequent culture on Sabouraud's Dextrose Agar (SDA). The direct microscopic examination showed spores outside the hair shafts (Figure 1c).

The established diagnosis was tinea capitis greypatch type, and then patient was treated with oral griseofulvin 125 mg microsize 2x3 tablets per day and ketoconazole 2% shampoo everyday. She was asked not to share combs, towels, and other hair products with others. The positive result of fungal culture on SDA at 37º C within 10 days was identified as *Microsporum audouinii*, which colonies were flat, spreading, greyish-white in colour, and had a dense suede-like surface. The microscopic examination of the culture growth showed element of fungi: Macroconidia and Microconidia, pectinate (comb like) hyphae, and racquet hyphae (Figure 2).

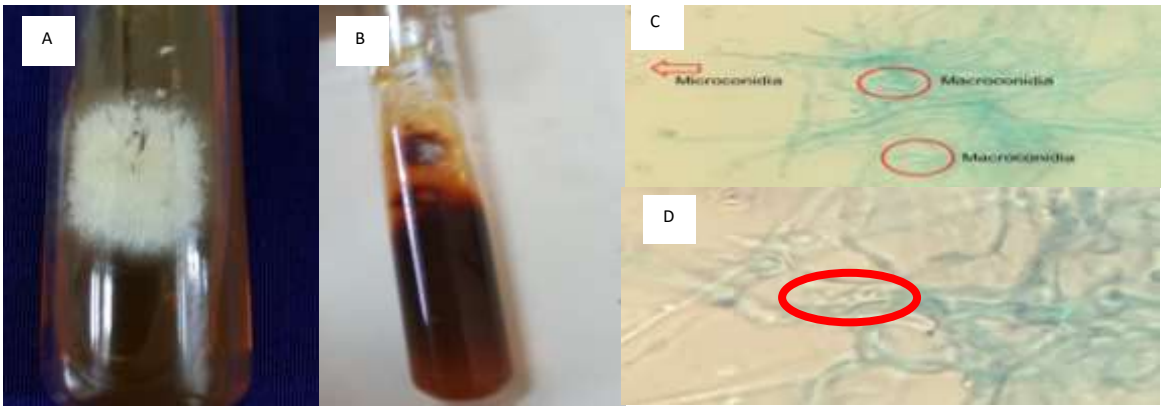

Figure 2. Growth of *M. audoinii* on Sabouroud's Dextrose Agar (SDA) in 10 days. (A) Colonies surface is flat, downy to silky, with a radiating edge, greyish-white in colour. (B) Reverse is light salmon with reddish brown center. (C) Microscopic appearance from fungal culture, magnification 40x. Macroconidia and microconidia. (B) Pectite hyphae.

After 2 weeks of treatment there was no significant progress: itchy sensation and scale still persist, Wood's lamp examination and potassium hydroxide examination were still positive. At follow-up visit on week 4, there was no erythematous plaque, the itchy sensation was decreased and the scale was minimal, but the result of Wood's lamp and potassium hydroxide examination were still positive (Figure 3).

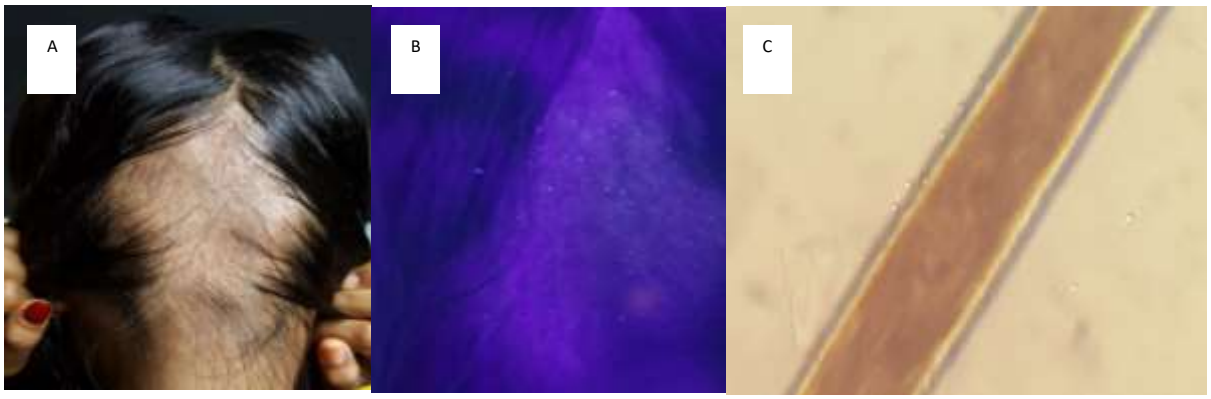

Figure 3. Lesion after 4 weeks of treatment. Wood's lamp examination still showed bright green fluorescence and potassium hydroxide examination showed spores outside the hair shaft, magnification 40x.

The patient still treated with antifungal drug and ketokonazole shampoo 3 times weekly. At follow-up visit on week 6, the appearance of his scalp was improved, no erythematous plaque, the itchy sensation and the scale were cured (Figure 4), the result of woodlamp's and potassium hydroxide examination were negative. The patient still continuing antifungal drug and ketoconazole shampoo was applied 3 times weekly. The patient was clinically and mycologically cured after 8 weeks of treatment and alopecia completely resolved in 12 weeks.

Figure 4. Lesion progress. (A) After 6 weeks of treatment. (B) Lesion after 8 weeks of treatment, (C) Lesion after 5 months of treatment.

DISCUSSION

Dermatophytes are keratinophilic fungi that can be pathogenic for humans and animals. There are three genera of dermatophytes: *Tricophyton*, *Microsporum* and *Epidermophyton*, which was then classified into the class of Deuromycetes. Out of 41 species currently recognized, approximately 17 species are the common causes of human infection; 5 *Microsporum* species infects skin, hair and nail; 11 *Tricophyton* species infects skin, hair and nail; 1 *Epidermophyton* species infects skin and rarely infects nail. They have been classified as geophilic, zoophilic and anthropophilic species on the basis of their primary habitat associations. Geophilic dermatophytes are primarily associated with keratinous materials such as hair, feathers, hooves and horns, as a part of their decomposition process. Zoophilic and anthropophilic dermatophytes are adapted to the animal or human host and are the most frequent agents of superficial mycoses in animals and humans infecting the stratum corneum, hair, claws or nails.[14,15]

Tinea capitis is an infection caused by dermatophyte fungi (usually species in the genera *Microsporum* and *Trichophyton*) of scalp hair

follicles and the surrounding skin. It is a common infection among school age children in developing countries, while adult cases are rare. Tinea in adults is often more subtle and its cause is not well understood since it is believed that sebum in adults has protective effects that decrease the risk of infection, but an immune defect may facilitate hair invasion. Physical barriers such as the intact keratinized layer of skin, as well as the effect of UV light, physiochemical factors of temperature, moisture and pH, and fungistatic fatty acids on the skin play a vital role in resisting contact with the organism. In early puberty, the sebaceous glands of the scalp begin to secret sebum containing an increased amount of highly fungistatic and fungicidal fatty acid with a selective activity against fungal. This is thought to explain the rarity of tinea capitis in adults. Dermatophytic colonisation of the scalp disappears at puberty. Colonisation by *Pityrosporum orbiculare* may interfere with dermatophyte contamination, and the thicker calibre of adult hair may protect against dermatophytic invasion. Tinea capitis in adults generally occurs in patients who are immunosuppressed and those infected with HIV. In immunocompetent adults, the clinical features are often atypical. The disease may resemble bacterial folliculitis, folliculitis decalvans, dissecting cellulitis, or the scarring related to lupus erythematosus. It has been postulated that tinea capitis in post-menopausal women may be related to hormonal imbalances that alter the protective effects of sebum. The risk factors of tinea capitis in adult include underlying conditions such as diabetes, anemia, immunosuppression, corticosteroids, hormonal change (e.g. menopause) and degree of exposure to the pathogen (e.g. tinea located elsewhere on the body, contact from infected children or formites).[5,6,7] In nonurban communities, sporadic infections acquired from puppies and kittens are due to *M. canis*.[11,16] This is inconsistent with this case, while the patient often plays with kitten, but the dermatophytes found was *Microsporum audouinii*. There is also no correlation between the patient rheumatic heart disease and tinea capitis, but it is consistent with the risk factor that the patient has not had menarche yet. In normal women, sebum production increases at puberty up to fivefold in men, while in patient with amenorrhea, progesterone level is low, so that the production of sebum, that contain fungistatic and fungicidal fatty acids, also low.

There are three steps of dermatophytes infection: adherence of dermatophytes, penetration to keratinocyte tissue, and immune respon of the host. [17] The possible route of entry for the dermatophytes into the host body is injured skin, scars and burns. The first step is successful adherence of arthroconidia, asexual spores formed by fragmentation of hyphae, to the surface of keratinized tissues. Dermatophytes adhere to the surface of the keratinized tissue to reach the epidermis by germination of athroconidia and then the hypha enters the stratum corneum. Arthroconidia adhesion on keratin tissue reached its maximum within 6 hours, mediated by dermatophytes outer wall fibers that produce keratinase (keratolytic) that can hydrolyze keratin and facilitate the growth of this fungus in the stratum corneum. Dermatophytes proteolytic and lipolytic activity by issuing a serine proteinase (urokinase and tissue plasminogen activator), which causes extracellular protein catabolism in invading the host. This process is influenced by the proximity of the wall of the cell and influence between artrospor sebum and corneocytes facilitated by the process of trauma or lesion on the skin.[1,15,17]

After adherence, arthroconidia (spores) must germinate and penetrate the stratum corneum at a rate faster than desquamation process. The ability of dermatophytes to degrade keratin is considered a major virulence attribute. The penetration process is accomplished by the secretions proteinase, lipase, and musinolitik enzymes, which become nutrients for fungi. During penetration, dermatophytes produce a variety of virulence factors for infection that include both enzymes (such as protease enzym) and non-enzymes (such as xanthomegnin, melanin or melanin-like compounds).[11,18] There are three patterns of hair invasions: ectothrix, endothrix, and favus. Ectothrix infection is defined as fragmentation of the mycelium into conidia around the hair shaft or just beneath the cuticle of the hair with destruction of the cuticle. In endothrix infection arthroconidia formation occurs by fragmentation of hyphae within the hair shaft without destruction of the cuticle. Favus is characterized by production of hyphae, which are parallel to the long axis of the hair shaft. Once the hyphae degenerate, long tunnels are left within the hair shaft and may appear as airspaces within the hair shaft.[1,11,15]

When dermatophytes invade keratinized tissue, an innate immune response in the host tissue is induced by antigens or metabolites from dermatophytes. Each type of antigens may induce different types of responses. The main immune response is production of Th1-type adaptive immune response with the production of proinflammatory cytokines like interleukin (IL)-2 and interferon (IFN)-γ. This response is induced to control the infection. Antigens derived from dermatophytes can induce immediate (type I) or delayed (type IV) hypersensitivity skin test reactions.[18,20,21]

The clinical appearance of tinea capitis depends on the causative species as well as other factors such as the type of organism, the type of hair invasion, level of host resistance and the degree of inflammatory host response. The major clinical types include non inflammatory type, inflammatory type, blackdot and favus. Non inflammatory type or greypatch is seen most commonly with antrophophilic organism. Inflammation, which is the result of a hypersensitivity reaction to the infection, in this setting ranges from follicular pustules to furunculosis or kerion. The "black dot" form of tinea capitis is typically caused by the anthropophilic endothrix organisms *T. tonsurans* and *T. violaceum*. Favus is a chronic fungal infection of the scalp, glabrous skin, and/or nails caused by *T. schoenleinii*. Occasionally *T. violaceum* or *M. gypseum* may cause similar lesions.[1,21] The manifestation found in this case, a well defined 10 cm x 10 cm alopecia based on mild erythematous plaque covered with thin scale located in the parieto-occipitalis area. The hair was greyish in colour, lusterless and easily pulled out of the hair follicle. It is appropriate with the clinical appearance of greypatch type.

Clinical diagnosis of scalp infection can be difficult as presentation are wide ranging and variable, thus as definitive diagnosis can not be made on the clinical appearance alone. The wood's lamp examination, which was traditionally used to diagnose scalp ringworm, can detect infection with *Microsporum* sp. The greenish fluorescence that was seen under the light in this patient was due to an ectothrix infection of hairs, in which fungal spores form a sheath on the outside of the hair. Wood's light examination is of little value nowadays because in western countries most infections are due to *T. tonsurans*, which does not fluorescence.[1,5,22] In this case, by Wood's lamp examination, there was bright green fluorescence.

Microscopy examination using 10-30% solution of potassium hydroxide provides the most rapid means of diagnosis. Examination of properly mounted specimen will demonstrate the type of hair invasion involved. Although the specificity of potassium hydroxide examination is high, the rate of false negative interpretation may exceed 40% even for experienced observers. The microscopic appearance for *M. audouinii* is found pectinate (comb-like) hyphae.[1,5] In this case, arthrospore were seen outside the hair shaft which demonstrated echthotrix infection and also there was found macroconidia and microconidia, pectinate (comb like) hyphae, and racquet hyphae.

Definitive diagnosis and exact identification of the causative organism of tinea capitis could be determined by culture. The sample should be inoculated in fungal culture medium such as SDA. Growth of colonies is observed in 1 to 6 weeks.[1,5] The strain isolated in this case lesions was characterized by microscopic examination and culture characteristics, showing colonies surface is flat, downy to silky, with a radiating edge, greyish-white in colour and that was compatible with *M. audoinii.*

There are topical and oral therapy for tinea capitis. Although a small percentage of patients may clear with topical agents, topical therapy alone is not recommended for the management of tinea capitis, because topical agents are unable to penetrate in the hair follicle sufficiently to clear the infection. A small randomized trial found that topical treatment increases the rate of eradication which may reduce the transmissibility of the organisms by reducing the shedding of fungal spores at the beginning of systemic treatment and may shorten the cure rate with oral antifungal. However, topical agents are used to reduce transmission of spores, such as povidone-iodine, ketokonazole 2% and selenium sulfide 1% shampoo. 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 mycology cured.[1,19,23]

Griseofulvin is a fungistatic drug that inhibits nucleic acid synthesis, arrests cell division at metaphase and impairs synthesis of the cell wall. There is over 50 years of experience in the use of the drug, and it remains the only licensed product for use in the treatment of tinea capitis in children in the U.K. Current dosage recommendation of griseofulvin is 15–25 mg/kg/day of the microsize form, and 15 mg/kg/day in divided doses of the ultramicrosize form for 8 weeks. A recent meta-analysis of randomized controlled trials (RCTs) suggests that 8 weeks of griseofulvin treatment is significantly more effective than 4 weeks of terbinafine in confirmed *Microsporum* infection.[1,19,23]

Although in the UK griseofulvin remains the only licensed treatment for tinea capitis in children, cumulative evidence now demonstrates that newer antifungal agents have higher response rates, and are safe and more cost-effective. The newest oral antifungal agents including oral terbinafine, itraconazole, fluconazole, and ketoconazole are effective in tinea capitis.[1,19,23]

In this case, after the diagnosis of tinea capitis greypatch type was made, the patient was treated with combination oral griseofulvin 125 mg microsize 2x3 tablets per day and ketoconazole 2% shampoo

everyday. We use griseovulvin, because is still the treatment of choice for cases caused by *Microsporum* species. Its efficacy is superior to that terbinafine, and altough its efficacy and treatment duration is matched by that of fluconazole and itraconazole, griseovulfin is cheaper.

The definitive end point for adequate treatment must be mycological cure, rather than clinical response. Therefore, follow-up with repeat mycology sampling is recommended at the end of the standard treatment period and then monthly until mycological clearance is documented. [19] In this case, the patient was clinically and mycologically cured after 8 weeks of treatment and alopecia completely resolved in 12 weeks. Tinea capitis is a superficial fungal infection predominantly caused by *Microsporum* and *Tricophyton* species. The wood's lamp can used to detect *Microsporum* sp infection with greenish fluorescence as the result. Definitive diagnosis and exact identification of the causative organism of tinea capitis could be determined by culture. The goal of treatment is to eliminate the causal organisms in the form of clinical and mycologic healing as quickly and safely as possible and also to prevent scarring and reduction of transmission to others.

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