



Epidermophyton floccosum as a possible aethiological agent of tinea capitis: a case report

Rudi Chandra^{1*}, Nova Z Lubis²

¹Department of Dermatology & Venereology, Faculty of Medicine, Prima Indonesia University, Medan, Indonesia, ²Department of Dermatology & Venereology, Faculty of Medicine, University of Sumatera Utara/Universitas Sumatera Utara Hospital, Medan, Indonesia

ABSTRACT

Submitted: 2020-09-20
Accepted : 2021-02-01

Tinea capitis is a superficial fungal infection of scalp that affects particularly school-aged children. It is usually caused by dermatophytes, although *Epidermophyton floccosum* can cause the tinea capitis. Tinea capitis caused by the *E. floccosum* is quite interesting because it can confirm the sporadic occurrence of hair parasitism by this dermatophyte. In this article, it was reported tinea capitis caused by *E. floccosum*. A 15-year-old girl, presented with itchy scaly alopecia on scalp since 1 week ago. The patient denied any contact with cat or dog, and there was not any family history. On physical examinations, lymphadenopathies were found at both lateral neck and alopecia with scales and crusts, lenticular to nummular sizes, at scalp. Hair-pull test was positive. The trichoscopy examination found comma hairs, broken hairs, and black dots. The fungal culture examination discovered the growth of *E. floccosum*. The patient was diagnosed with tinea capitis and treated with 500 mg of griseofulvin orally, 10 mg of cetirizine orally, and 2% of ketoconazole shampoo. *Epidermophyton floccosum* is an anthropophilic dermatophyte that frequently causes tinea cruris, tinea pedis, tinea corporis and onychomycosis, but not tinea capitis. Several reports have showed the capability of *E. floccosum* in perforating hairs and causing tinea capitis. In conclusion, *E. floccosum* is a possible aethiological agent of tinea capitis through its ability to perforate hairs.

ABSTRAK

Tinea kapitis merupakan suatu infeksi jamur superfisial pada kulit kepala yang terutama menyerang anak-anak usia sekolah. Tinea kapitis umumnya disebabkan oleh dermatofita, meskipun dapat disebabkan *E. floccosum*. Tinea kapitis yang disebabkan oleh *E. floccosum* cukup menarik karena dapat mengkonfirmasi terjadinya parasitisme rambut secara sporadis oleh dermatofita ini. Dalam makalah ini dilaporkan tinea kapitis yang disebabkan *E. floccosum*. Seorang anak perempuan berusia 15 tahun, datang dengan keluhan kebutakan yang bersisik dan gatal di kulit kepala sejak 1 minggu yang lalu. Pasien menyangkal adanya kontak dengan kucing atau anjing, tidak ada riwayat keluarga. Pada pemeriksa anfisik, ditemukan adanya limfadenopati di leher bagianlateral dan alopesia dengan skuama dan krusta, berukuran lentikular hingga numular, di kulit kepala. Tes tarik rambut positif. Pada pemeriksaan trikoskopi, ditemukan rambut koma, rambut patah, dan bintik hitam. Dari pemeriksaan kultur jamur ditemukan pertumbuhan *E. floccosum*. Pasien didiagnosis dengan tinea kapitis dan diobati dengan griseofulvin 500mg per oral, cetirizine 10mg per oral, dan sampo ketokonazol 2%. *Epidermophyton floccosum* merupakan dermatofita antropofilik yang sering menyebabkan tinea kruris, tinea pedis, tinea korporis dan onikomikosis, tetapi tidak menyebabkan tinea kapitis. Beberapa laporan menunjukkan kemampuan *E. floccosum* dalam memperforasi rambut dan menyebabkan tinea kapitis. Dapat disimpulkan *E. floccosum* mungkin merupakan agen etiologi tinea kapitis melalui kemampuannya untuk memperforasi rambut.

Keywords:

Epidermophyton Floccosum;
fungal culture;
tinea capitis;
tinea cruris;
onychomycosis;

INTRODUCTION

Tinea capitis is a superficial fungal infection or dermatophytosis of scalp also referred to as ringworm, involving hair shaft and follicles.^{1,2} While it may affect any age group, tinea capitis is particularly common among school-aged children between 3 and 14 years of age. It is well known that tinea capitis is more common in children of African descent compared to Hispanics and Caucasians.³ Tinea capitis represents 4 to 10% of dermatophyte infections. Transmission is increased with decreased personal hygiene, overcrowding and low socioeconomic status. It is more frequent in tropical regions with low socioeconomic conditions and affects almost exclusively children (98%).^{3,4}

The clinical appearance of ringworm of scalp is variable, depending on the type of hair invasion, the level of host resistance and the degree of inflammatory host response. Clinically, tinea capitis is divided into 3 types, non-inflammatory type, "black dot" type, and inflammatory type.⁵ The non-inflammatory type can be found in 90% of affected children and inflammatory type is only found in 10% of prepubescent children. Tinea capitis is caused by dermatophytes that uses keratin as a nutrient source. Scalp erythema, scaling, and crusting are typical signs of this fungal infection.⁴

Many species of dermatophytes are capable of invading hair shafts and causing tinea capitis, especially *Trichophyton sp.* and *Microsporum sp.*, whereas *Epidermophyton floccosum* and *Trichophyton concentricum* do not cause tinea capitis.⁵ Many authors did not correlate the involvement of *E.floccosum* as an aethiological agent in tinea capitis. Otherwise, the

inability of this species to damage hair is often being mentioned. Case of tinea capitis caused by this species is quite interesting because it can confirm the sporadic occurrence of hair parasitism by this dermatophyte.⁶ In this article, it was reported tinea capitis caused by *E. floccosum* and its management.

CASE

A 15-year-old girl was initially presented with scaly alopecia on her scalp since 1 week ago. She experienced itchiness on scalp 3 weeks prior alopecia and found fleas that run on her headscarf. These same symptoms were also experienced by her friends at school. The patient's parents put onions and salts on her scalp, but the complaints did not improve. One week after that appeared bad odor, sticky and slimy hairs, as well as crust on her scalp. One week before the admission, the patient had her hairs trimmed bald, so that the alopecia was seen. The patient denied any contact with cat or dog and had no family history with similar complaint. She had undergone surgery due to ovarian germ cells tumor in the left ovary in 2014 and received six cycles of chemotherapy (consisting of bleomycin, etoposide, and cisplatin).

The physical examinations found lymphadenopathies, Ø 1x1 cm, mobile, pain (-) at the left and right lateral neck. The dermatological examination found alopecia with scales and crusts, lenticular to nummular sizes; some are confluent, at the region of scalp. Thick scales were found on both retroauricular regions (FIGURE 1). Hair pull test was positive. The direct potassium hydroxide examination (10-20%) from skin scraping of the scalp found no hypha or spore. The trichoscopy

examination found comma hairs, broken hairs, black dots, and perifollicular scales (FIGURE 2). The patient weighed 53 kg and was 145 cm tall.

The patient was diagnosed with different diagnosis i.e. tinea capitis, scalp psoriasis, and alopecia areata. Fungal culture was performed

through skin and hair scraping. The patient was treated with once-daily oral administration of 500 mg griseofulvin, once-daily oral administration of 10 mg cetirizine, and administration of 2% ketoconazole shampoo three times a week.



FIGURE 1. Multiple alopecia with scales on scalp; (A) upper, (B) back, (C) left lateral, (D) right lateral views.

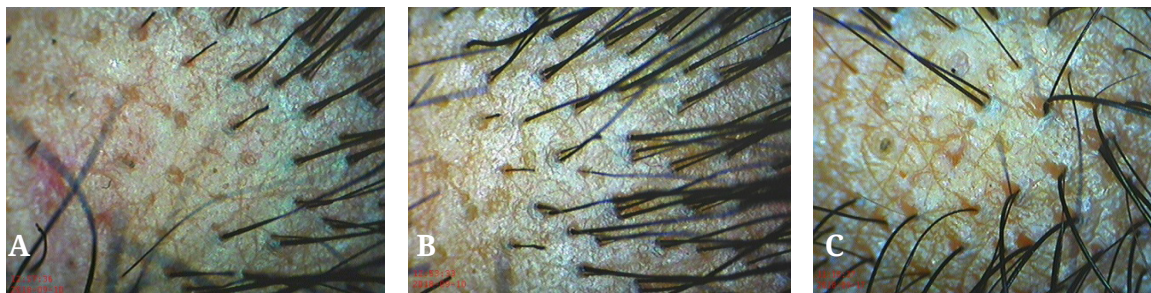


FIGURE 2. Trichoscopy features on several alopecia areas were comma hairs (red arrow), broken hairs (green arrow), black dots (black arrow); (A), (B), and (C), respectively.

The fungal culture discovered the growth of *E. floccosum*, with flat feathery yellowish colony with yellow to brown reverse pigment. The microscopic examination on the colony found numerous thin and thick walled club-shaped macroconidias (FIGURE 3). At the first follow-up (2 weeks after the treatment), itchiness and scales on the scalp were subsided (FIGURE 4). Lymphadenopathies was disappeared. The trichoscopic examination found increased vellus hairs, no comma hairs,

broken hairs, or black dots (FIGURE 6A & 6B). Therefore, the treatment was continued with once daily administration of 500 mg griseofulvin and 2% ketoconazole shampoo, whereas administration of cetirizine was stopped.

At the 2nd follow-up (4 weeks after treatment), the patient's hairs increased (FIGURE 5). The trichoscopic examination showed increased vellus hairs' growth, no comma hairs, broken hairs, or black dots (FIGURE 6C & 6D). Griseofulvin was continued until 6 weeks.

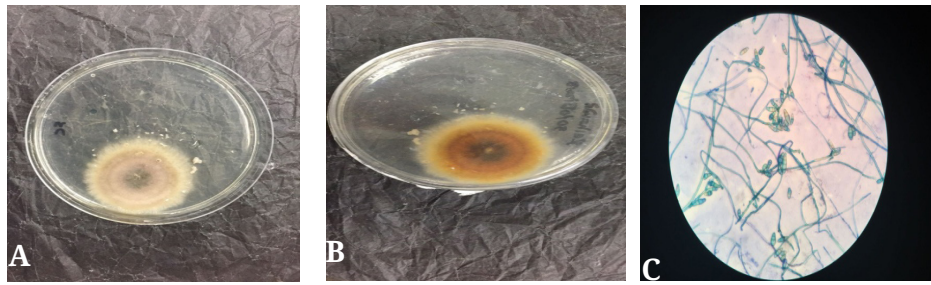


FIGURE 3. Fungal culture in Sabouraud dextrose agar medium; macroscopic (A) upper, (B) bottom, and (C) microscopic view.

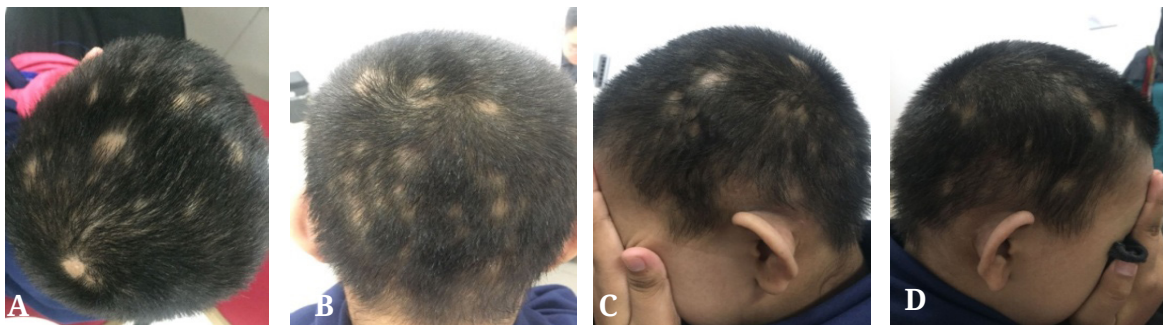


FIGURE 4. At 1st follow-up 2 weeks after treatment; (A) upper, (B) back, (C) left lateral, (D) right lateral views.



FIGURE 5. At 2nd follow-up 4 weeks after treatment; (A) upper, (B) back, (C) left lateral, (D) right lateral views.

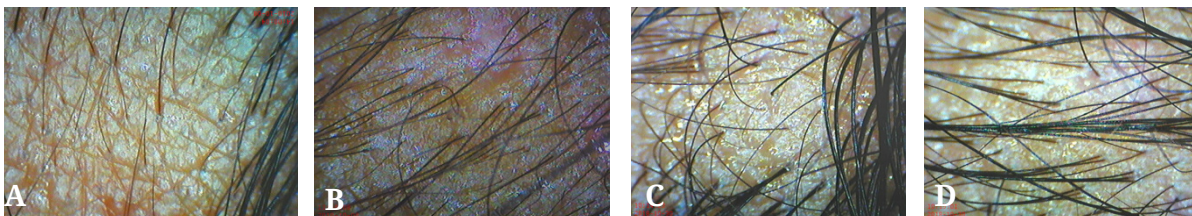


FIGURE 6. Trichoscopy features at the 1st follow-up (A), (B) and 2nd follow-up (C), (D); found increased vellus hairs.

DISCUSSION

The diagnosis of tinea capitis in this case was based on clinical history, physical and dermatological examination, trichoscopy, and culture. The clinical appearance of tinea capitis is highly variable, depending on the causative organism, type of hair invasion and degree of host inflammatory response. Common features consisted of patchy hair loss with varying degrees of scaling and erythema. The presence of regional lymphadenopathy in combination with alopecia and/or scale in a child suspected to have tinea capitis is an important diagnostic clue and should encourage appropriate investigation with fungal culture.⁷ Since these clinical features were found in this case, other examinations were provided to investigate it.

Nowadays, the usefulness of trichoscopy as a supplementary method to examine hair and scalp disorders is well-documented for several conditions and it is a non-invasive, quick and inexpensive procedure.⁸ Trichoscopy features in tinea capitis were black dots, 'comma-shaped' hairs (ecthotrix infection), corkscrew hairs (reported in Afro-Caribbean children), and zigzag shaped hairs.⁹ Few cases reported "comma" and "corkscrew" hair as features in child tinea capitis.^{8,10} In this case, the trichoscopy found comma hairs, broken hairs, and black dots.

The clinical diagnosis of dermatophyte infections could be confirmed by the gold standard examination to identify fungal species through culture. Fungal culture confirmation must be considered while the systemic treatment of oral antifungal drug is given. In present case, *E. floccosum* growth was found in the culture. *Epidermophyton floccosum* is an anthropophilic dermatophyte, where macroscopically in culture as flat feathery colonies with a central fold and yellow

to dull gray-green pigment and yellow to brown reverse pigment. Microscopically, numerous thin and thick-walled club-shaped macroconidia were found.³ *Epidermophyton floccosum* frequently causes tinea cruris, tinea pedis, tinea corporis and onychomycosis, but not tinea capitis.^{5,6} Some researchers rarely associate involvement of *E. floccosum* as the causative agent of tinea capitis. Conversely, the inability of this species to damage hair is often mentioned.⁶ Tinea capitis is typically caused by *Trichophyton* and *Microsporum* species.³ *Trichophyton tonsurans* is the most commonly-found species in the United States and United Kingdom, macroscopically presented as a Suede-like center with feathery periphery, white to yellow or maroon. Reverse pigment is usually dark maroon and microscopically is presented with numerous multiform microconidia and rare cigar-shaped macroconidia.³ *Microsporum canis* is also the most common cause of tinea capitis in Europe, which is macroscopically presented as flat, white to light yellow, coarsely hairy, with closely spaced radial grooves, with yellow to orange reverse pigment, and is microscopically presented with numerous thick walled and echinulate spindle shaped macroconidia with terminal knobs and more than 6 cells.³

Several reports have showed the capability of *E. floccosum* in perforating hairs and causing tinea capitis. Nikpoor *et al.*¹¹ reported a case report of a 9-year-old boy in Southern Iran, presented with multiple erythematous annular lesions involving the whole scalp without hair loss caused by *E. floccosum*.¹¹ Moto *et al.*¹² reported the prevalence of tinea capitis in school-aged children in Kenya in which out of 150 children examined, 122 (81.3%) were found to have fungal lesions caused by *Trichophyton* (61.3%), *Microsporum* (13.3%) and *Epidermophyton* (7.3%). There were 11 (7.3%) children found to be positive for *Epidermophyton* infections within age

group 6–8 years with males being the most affected.¹² Romano also reported four pediatric cases of tinea capitis due to unusual agents. He reported three cases of tinea capitis caused by *T. rubrum* and one case caused by *E. floccosum* in a 9-year-old girl with a 2-year history of autoimmune thrombocytopenia, and therefore she was treated with cyclic systemic cortisone therapy.¹³ Macedo *et al.*⁶ reported the ability of 15 isolates of *E. floccosum* to perforate hairs *in vitro*. Fourteen isolates perforated hair and twelve of them produced perforating organs. All isolates grew at 37°C and produced proteinase, but not phospholipase. There was also a study that reported an extracellular keratinase of some dermatophytes, teleomorphs and related keratinolytic fungi. The study reported that the keratinase of *Microsporum*, *Trichophyton*, and *Epidermophyton* ranged from 50-300 ku/mL, 100-140 ku/mL, and 20 ku/mL, respectively.¹⁴ Therefore, these reports suggested that *E. floccosum* was a possible aethiological agent of tinea capitis due to its ability to produce proteinase and keratinase.

The research found that ovarian germ cell tumor and chemotherapy as the possible risk factors in the patient. Germ-cell tumored of the ovary represent 15-20% of all ovarian tumors. It was a rapidly growing neoplasms arising from primordial germ cells that derived from embrional gonad.¹⁵ These ovarian germ cell tumors were mostly affected children and young adult at the age of 10-20 years.¹⁶ Although there was not any report about the relevance of this tumor with the occurrence of tinea capitis, it cannot be denied that this condition could be a risk factor. Moreover, chemotherapy also implicated patient's immune system. Kang *et al*¹⁷ reported that chemotherapy or chemo- and radiotherapy combination for breast cancers significantly delayed IL-2 recovery. Immune recovery following breast cancer adjuvant therapy was

significantly delayed for an extended time period in numerous immune parameters.¹⁷

Tinea capitis usually needs oral antifungal treatment because dermatophytes penetrate into follicles out of range of topical agents.³ Although topical antifungal is not recommended for tinea capitis, topical antifungal can be used to reduce transmission from spores. Topical antifungal includes 2.5% povidone-iodine, 2% ketoconazole shampoo, 1-2% zinc-pyrithione, and 1-2.5% selenium sulfide.^{3,7} In the present case, 2% ketoconazole shampoo was used to reduce spores transmission.

Based on British Association of Dermatologists guidelines in the treatment of tinea capitis in 2014, the recommended oral antifungal treatments were griseofulvin, terbinafine, itraconazole and fluconazole.⁷ Griseofulvin was the first effective drug used to treat tinea capitis and is still widely used in resource-poor settings as it remains effective.⁵ Griseofulvin and terbinafine have been approved by US Food and Drug Administration as the treatment of tinea capitis.³ Griseofulvin is a fungistatic drug that inhibits nucleic acid synthesis, arrests cell division at metaphase and impairs synthesis of cell wall. The standard licensed treatment protocol was 15-20 mg per kg body weight, daily, in single or divided doses for 6-8 weeks. Taking the drug with fatty food may increase absorption and improve bioavailability.⁷ In this case, the recommendation of oral antifungal treatment was used for tinea capitis where clinically it was improved and confirmed by trichoscopy with increased vellus hairs on the alopecia sites.

CONCLUSION

It is concluded that *E. floccosum* is a possible aethiological agent of tinea capitis through its ability to perforate hairs.

ACKNOWLEDGEMENTS

We would like to thank our colleagues from the Department of Microbiology for discussing with us in determination of the aetiology in this case.

REFERENCES

- Gupta AK, Mays RR, Versteeg SG, Piraccini BM, Shear NH, Piguat V, et al. Tinea capitis in children: a systematic review of management. *J Eur Acad Dermatol Venereol* 2018; 32(12):2264-74. <https://doi.org/10.1111/jdv.15088>.
- Meadows-Oliver M. Tinea capitis: diagnostic criteria and treatment options. *Dermatol Nurs* 2009; 21(5):281-6.
- Schieke SM, Garg A. Superficial Fungal Infection. In: Goldsmith LA, Katz SI, Gilchrist BA, Paller AS, Leffell DJ, Wolff K. (Eds.): *Fitzpatrick's Dermatology In General Medicine*. 8th edition. New York: McGraw-Hill Companies 2012. 2277-97.
- Salazar E, Asz-Sigall D, Vega D, Arenas R. Tinea capitis: unusual chronic presentation in an elderly woman. *J Infect Dis Epidemiol* 2018; 4(1):048. <https://doi.org/10.23937/2474-3658/1510048>
- Hay RJ. Tinea capitis: current status. *Mycopathologia* 2017; 182(1-2):87-93. <https://doi.org/10.1007/s11046-016-0058-8>
- Macêdo DPC, Neves RP, Magalhães OMC, de Souza-Motta CM, de Queiroz LA. Pathogenic aspects of epidermophytonfloccosum langeron et milochevitvhas a possible aetiological agent of tinea capitis. *Braz J Microbiol* 2005; 36(1):36-7. <https://doi.org/10.1590/S1517-83822005000100007>
- Fuller LC, Barton RC, MohdMustapa MF, Proudfoot LE, Punjabi SP, Higgins EM. British association of dermatologists' guidelines for the management of tinea capitis 2014. *Br J Dermatol* 2014; 171(3):454-63. <https://doi.org/10.1111/bjd.13196>
- Schechtman RC, Silva NDV, Quaresma MV, Filho FB, Buçard AM, Sodré CT. Dermatoscopic findings as a complementary tool in the differential diagnosis of the etiological agent of tinea capitis. *An Bras Dermatol* 2015; 90(3 Suppl 1):13-5. <https://doi.org/10.1590/abd1806-4841.20153787>
- El-Taweel AE, El-Esawy F, Abdel-Salam O. Different trichoscopic features of tinea capitis and alopecia areata in pediatric patients. *Dermatol Res Pract* 2014; 2014:848763. <https://doi.org/10.1155/2014/848763>
- Pinheiro AMC, Lobato LA, Varella TCN. Dermoscopy findings in tinea capitis. case report and literature review. *An Bras Dermatol* 2012; 87(2):313-4. <https://doi.org/10.1590/s0365-05962012000200022>
- Nikpoor N, Leppard BJ. Ringworm on the scalp due to *Epidermophyton floccosum*. *Clin Exp Dermatol* 1977; 2(1):69-71.
- Moto JN, Maingi JM, Nyamache AK. Prevalence of Tinea capitis in school going children from Mathare, informal settlement in Nairobi, Kenya. *BMC Res Notes* 2015; 8:274. <https://doi.org/10.1186/s13104-015-1240-7>
- Romano C. Case Reports. Four paediatric cases of tinea capitis due to unusual agents. *Mycoses* 1999; 42(5-6):421-5. <https://doi.org/10.1046/j.1439-0507.1999.00494.x>
- Singh I. Extracellular keratinase of some dermatophytes, their teleomorphs and related keratinolytic fungi. *Euro J Exp Bio* 2014; 4(4):57-60.

15. Maniar KP, Vang R. Germ Cell Tumors of the Ovary. In: Kurman R, Hedrick Ellenson L, Ronnett B. (eds) Blaustein's Pathology of the Female Genital Tract. Springer, Cham. 2019. pp 1047-1124.
https://doi.org/10.1007/978-3-319-46334-6_16
16. Fonseca A, Frazier AL, Shaikh F. Germ cell tumors in adolescents and young adults. *J Oncol Pract* 2019; 15(8):433-41.
<https://doi.org/10.1200/JOP.19.00190>
17. Kang DH, Weaver MT, Park NJ, Smith B, McArdle T, Carpenter J. Significant impairment in immune recovery after cancer treatment. *Nurs Res* 2009; 58(2):105-14.
<https://doi.org/10.1097/NNR.0b013e31818fcec>