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CASE REPORT

Tinea capitis profunda in an adult case

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ABSTRACT

Tinea capitis is a dermatophyte infection of the scalp that occurs in childhood. Scalp erythema, scaling, pustules, and crusting are typical signs of tinea capitis. Tinea capitis is considered rare in adults. Adult tinea capitis may have polymorphic and atypical clinical presentations. Psoriasis, seborrheic dermatitis, and lichen simplex chronicus should be considered in the differential diagnosis of tinea capitis. Fungal examination should be performed for diagnosis. Early and accurate diagnosis of the disease prevents the formation of scars with the treatment administered. When diagnosing tinea capitis in adults, predisposing factors should be investigated. Here we present a case of tinea capitis profunda in a healthy adult.

Keywords: adult, dermatophyte, tinea capitis

INTRODUCTION

Tinea capitis (TC) is a dermatophyte infection characterized by scaling, alopecia, and pustules on the scalp and hair. It generally affects prepubertal children and is rarely observed in adults. The reason for this may be the fungistatic effect of saturated fatty acids in sebum with puberty. While TC is generally expected in adults with immunosuppression, recent reports have demonstrated that it also occurs in healthy adults. The present study presents a 42-year-old case of tinea capitis profunda without immunosuppression.

CASE REPORT

A 42-year-old female patient presented to our clinic with a three-week history of a painful sore on her scalp, which began as a pustule and spread. Before coming to our department, she was first treated with clobetasol lotion and ampicillin-sulbactam 1 g tablets 2x1 for one week. When there was no response, she received tetracycline capsule 100 mg/day 1x1 and mupirocin ointment treatment from another hospital. Her dermatologic examination revealed widespread pustules with yellowish crusts on the scalp and parietal region, approximately 8x8 cm in diameter, around the hair follicles on an erythematous alopecic area (Figure 1). There was no scutum and no mousy odor. On examination, she had painful lymphadenopathy in the right postauricular region. Wood's light examination did not show any reflection. The patient had a history of taking levothyroxine tablets for hypothyroidism. She was not menopausal. The infection was not present in family history. In the laboratory findings of the patient, erythrocyte sedimentation rate and C-reactive protein were normal. Hemoglobin was 9.5 g/dL and ferritin

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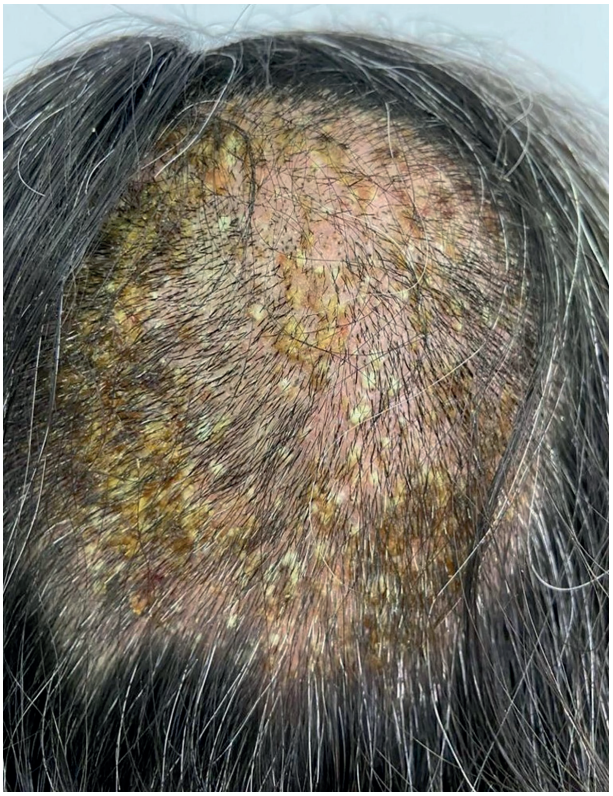

Figure 1. Widespread pustules with yellowish crusts on the scalp and parietal region.

was 3.37 microgram/L (10-204). Laboratory tests indicated no abnormalities except for low hemoglobin and ferritin levels in nonimmunsupressive patient. The sample taken from the patient's pustular lesions was assessed microscopically with potassium hydroxide and septate hyphae and spores was observed (Figure 2). There was no growth in the wound culture and fungal culture taken from the patient. Oral terbinafine 250 mg/day, isocanozole cream and ketocanozole shampoo were prescribed to the patient who was diagnosed with tinea capitis profunda. In the follow-up one month later, the lesions regressed and there was new hair growth (Figure 3).

DISCUSSION

TC is a superficial fungal infection caused by dermatophytes, and it is commonly observed in prepubertal children. The immune response of the host against dermatophyte infection determines the

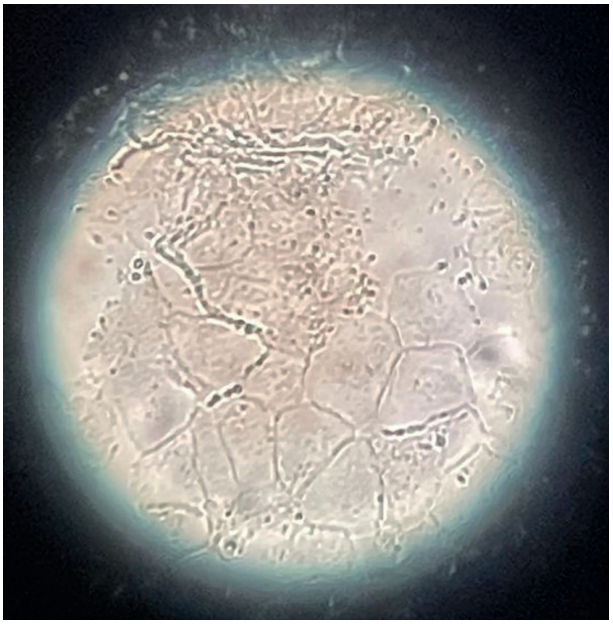

Figure 2. Septate hyphae and spores in the native preparation.

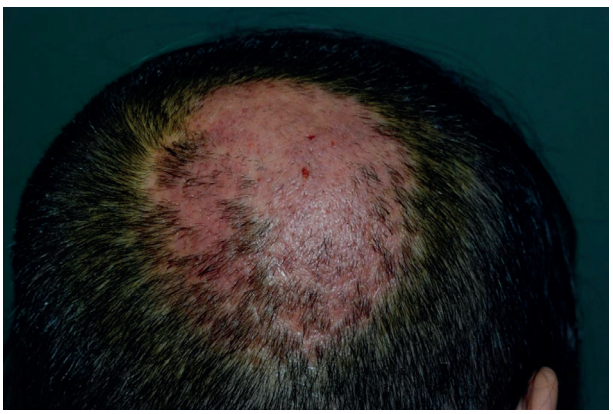

Figure 3. At the end of the first month, the lesions regressed.

clinical presentation and appears in three main clinical forms: tinea capitis superficialis, tinea capitis profunda (kerion celsi), and favus. Tinea capitis profunda is an inflammatory scalp lesion that occurs due to a delayed-type cellular immune response against fungal elements. The follicles that pustulate on the scalp merge over time and become painful inflammatory nodules. TC is rarely observed in adults, which may be attributed to the fungistatic effect of short- and medium-chain fatty acids present in sebum, the maturation of hair follicles,

and the effectiveness of the immune system during the post-pubertal period (1,2).

Risk factors for the development of TC in adults include menopausal status, age over 50 years, diabetes mellitus, and the use of topical steroids on the scalp (2). According to a study conducted in China between 2000 and 2019, only six of the 269 diagnosed TC cases were immunosuppressive. The majority of patients with TC were 45 years of age or older.

The study determined that women were more likely to be diagnosed with the disease than men, and that postmenopausal women were at higher risk of TC (3). In the study by Khosravi et al., 25 out of 121 patients with TC were adults. The disease-causing immunosuppression was observed in 80% of the adult patients and the remaining adult patients were found to be healthy (4). During the examinations of our patient, there were no abnormal clinical and laboratory findings except for iron deficiency.

The clinical presentation of TC may be atypical, which may lead to misdiagnosis and delay in treatment (3,4). It has been reported in the literature that the diagnosis of TC may take anywhere from 20 days to 30 years. A delay in diagnosis and treatment may lead to scarring alopecia on the scalp (3). In our case, it took 3 weeks to get the right diagnosis.

In the differential diagnosis, the possibility of diseases such as folliculitis, folliculitis decalvans, dissecting cellulitis, seborrheic dermatitis, and psoriasis should be taken into account (5). In suspected patients, potassium hydroxide (KOH) examination and fungal culture tests should be performed. In the study by Liang et al., KOH examination was positive in all patients, while the growth rate of fungal culture was detected as 90.7% (3). In our patient's KOH examination, septate hyphae and spores were observed, but no fungal growth was found in the culture.

Some systemic antifungals such as griseofulvin, terbinafine, itraconazole and fluconazole are recommended as first-line treatment alternatives for TC. Topical agents may be added to the treatment to prevent the spread of fungal spores (6).

Treatment must be continued for at least 6 to 8 weeks. Short-term glucocorticoids may be applied to suppress inflammation in the kerion. If secondary infection occurs, appropriate use of antibiotics and epilation of the hair around the lesion may increase the effectiveness of treatment (1,6). We used systemic terbinafine, isoconazole cream, and ketoconazole shampoo in treatment of our patient.

Ethical approval

Written informed consent was obtained from the participants.

Author contribution

Concept: TS; Design: TS; Data Collection or Processing: FNŞ; Analysis or Interpretation: TS, FNŞ; Literature Search: TS, FNŞ; Writing: TS. All authors reviewed the results and approved the final version of the article.

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Conflict of interest

The authors declare that there is no conflict of interest.

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