

SHORT COMMUNICATION

Disseminated Favus Caused by *Microsporium gypseum* in a Patient with Systemic Lupus ErythematosusTutyana Sanusi¹, Jin Gong², Xia Wang¹, Mengjie Zhao¹, Yun Zhao¹, Xiangjie An¹, Chunsen Wang¹, Changzheng Huang^{1*} and Siyuan Chen^{1*}Departments of Dermatology, ¹Union Hospital, Tongji Medical College, Huazhong University of Science and Technology, Wuhan 430022, and ²The First People's Hospital of Jingzhou, Jingzhou 434000, Hubei, China. *E-mail: hcz0501@126.com, siyuanc_cn@126.com

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Fungal infection of the skin caused by *Microsporium gypseum* results in a range of clinical features, including classical ringworm pattern, circumscribed scleroderma-like, eczematoid, and scutula-like lesions. The variety of the lesions may be influenced by the immune status of the individual, but unusual features also can be observed in immunocompetent patients (1, 2). Eight cases of scutula-like lesions associated with *M. gypseum* infection have been reported previously; one case was found in a patient with systemic lupus erythematosus (SLE) (3). We report here the second case of scutula-like disseminated lesions caused by *M. gypseum* in a patient with SLE.

CASE REPORT

A 59-year-old woman presented with a 2-month history of multiple disseminated yellowish-plaques on her forehead, cheek, shoulder, trunk and buttocks. The patient reported that the lesions initially appeared on her forehead with multiple-yellowish, round-oval plaques accompanied by localized hair loss in the affected areas. No “mousy” odour was noted. The lesions gradually increased in number and size and spread to her temporal cheek, chest, shoulder, trunk and buttocks. The lesions were asymptomatic. She had been diagnosed with SLE more than 5 years previously and treated with long-term oral glucocorticoids, with a recent maintenance dose of oral prednisone 15 mg/day. She had also had hypothyroidism for 5 years and was being treated with oral thyroxine. Her medical history included appendectomy and cholecystectomy approximately 20 years previously.

Skin examination revealed well-circumscribed, multiple hyperkeratotic plaques with yellowish or honey-coloured elevated crusts on her forehead, temporal cheek, chest, shoulder, trunk and buttocks. The lesions coalesced into numerous large yellow-brownish cup-shaped crusts (scutula), giving a “honeycomb” appearance. After removal of the crusts, the lesions revealed an erythematous-moist base (Fig. 1). No similar cutaneous lesions were detected in her family members

and no history of contact with dogs or cats was found. The patient worked as a farmer.

A complete blood count and urinalysis examination revealed white blood cell count 7.14 g/l (normal 3.5–9.5 g/l), red blood cell count 2.12 T/l (3.8–5.1 T/l), haemoglobin 70 g/l (115–150 g/l), neutrophils 76.30% (40–75%), haematuria 156.8/μl (<25/μl), ferritin 1,265.9 μg/l (4.6–204 μg/l), IgA 6.75 g/l (0.82–4.53 g/l), C3 0.687 g/l (0.790–1.520 g/l), high-sensitivity C-reactive protein 30.4 mg/l (<8.0 mg/l), erythrocyte sedimentation rate 33 mm/h (<20 mm/h). Hepatitis B surface Antigen (HBsAg), anti-HCV, anti-HIV, and RPR were all negative. Ultrasound of the thyroid revealed multiple calcifications in bilateral lobes. An electrocardiogram was normal.

The histopathological findings of the biopsied lesion demonstrated mild epidermal hyperkeratosis and perivascular infiltration of inflammatory cells. Hyphae and spores were observed in the stratum corneum of the epidermis with haematoxylin-eosin and periodic acid-Schiff staining (Fig. S1a, b¹). Direct microscopic examination of the scales and crusts in 10% KOH preparation showed massive septate hyphae. For culture, specimens were inoculated on Sabouraud dextrose agar medium containing chloramphenicol at 25°C and cultured for approximately 3–5 days. The culture produced white powdery colonies with cinnamon colour and a yellow-brownish reverse. Microscopic examination of the colony demonstrated massive septate hyphae and multiple clusters, thin-walled, fusiform macroconidia, divided into 4–6 cells, typical of *M. gypseum* (Fig. S1c, d¹). The patient was treated with oral itraconazole, 400 mg/day, and topical ketoconazole and naftifine cream for 2 weeks, but unfortunately she did not return for a follow-up visit.

DISCUSSION

Infections are responsible for 30–50% of morbidity and mortality in patients with SLE. Immune system

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Fig. 1. The lesions showed yellow-brownish or honey-coloured crusts and coalesced into numerous large plaques and cup-shaped crusts (scutula) on (a) the forehead, (b) temporal cheek, (c) chest, and (d) trunk.

dysfunction and prolonged systemic therapy with immunosuppressive agents or glucocorticoids are responsible for increased rates of severe fungal infections; *Candida* spp., *Pneumocystis jirovecii* and *Cryptococcus neoformans* are the most frequently reported (4, 5).

Favus is an endothrix invasion characterized by longitudinally arranged hyphae and air space within the hair shaft. The air space is caused by autolysis of the hyphae. Arthroconidia are rarely seen in the infected hair. The infection begins its growth in the perifollicular stratum corneum and spreads into the hair shaft before descending into the follicle to penetrate the cortex. Progressive hyphae invasion distends the follicle, producing a yellow-red follicular papule and then a yellow concave crust. The crusts can gradually coalesce into large adherent plaques mixed with epithelial debris and dense masses of mycelium; the so-called scutula, maybe numerous and clustered in a "range of volcanoes"-like pattern (6, 7).

Tinea favosa or favus is a severe and chronic inflammatory dermatophytosis, which is characterized by the scutula formation. The scalp is the most commonly affected area and is associated with severe alopecia. Favus of the glabrous skin and nails are rarely reported (in only approximately 7% of cases), present with papulovesicular and/or papulosquamous lesions in which typical scutula may be evident (6, 8). Generalized involvement of favus on the scalp and glabrous skin are reported mainly in the rural areas and among individuals living in conditions of poor hygiene (6).

Dermatophytosis caused by *M. gypseum* rarely produce scutula-like lesions. The prevalence of human infections caused by *M. gypseum* is low compared with other dermatophytes; approximately 0.72–5.2% (1, 9). In China, this species was reported in only 1.32% in 2006 (3, 10). Transmission among humans, or from animals to humans, is rarely observed. It is usually passed on through direct contact with the soil containing a great number of spores, and is related to virulent strains, low resistance of the host, or previous skin bruising (3, 11). Krunic et al. (12) speculated that the virulence of the specific dermatophyte may be an important factor in favic invasion.

Feng et al. (3) reported the first case of scutula-like lesions caused by *M. gypseum* in a patient with SLE, localized on the right thigh, and no history of immunosuppression or corticosteroid therapy during diagnosis. The more severe and widespread lesions, involving the glabrous skin, long history of SLE and long-term use of systemic corticosteroids in our case are in contrast to the

previous case. The exact mechanism of scutula formation produced by *M. gypseum* infection in our patient remains unknown. The patient's underlying disease and long-term use of corticosteroids may play an important role in the presentation. As a farmer, the patient may come into direct contact with the soil containing *M. gypseum*, which is a causative agent for this infection.

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