Sir,
Tinea incognito represents cutaneous fungal infection whose clinical morphology has been modified by the use of systemic or topical steroids and other immunosuppressive agents (1–3). The clinical manifestation can successfully mimic a large number of other dermatoses, thus leading to misdiagnosis, sometimes with severe consequences.

CASE REPORT
A 68-year-old woman who had had pemphigus foliaceus for 13 years, presented for initiation of more aggressive therapy due to gradual continuous clinical worsening in the last 3 months despite potent topical steroid and long-standing systemic therapy with prednisolone 50 mg/day for one year. At the time of presentation desmoglein-1 antibodies were detectable to a low grade using enzyme-linked immunosorbent assay (ELISA). Clinical examination revealed a bizarre pattern of non-pruritic, brownish circinate plaques, covered with white-greyish greasy scales, which affected excessive areas of the body, with accentuation in the facial, lower torso and buttock areas (Fig. 1). There were no signs of tinea pedis or nail involvement. Histological and immunohistochemical examination revealed no evidence of active pemphigus foliaceus. However, periodic acid-Schiff (PAS) stain, fungal culture and polymerase chain reaction (PCR) analysis showed abundant *Trichophyton rubrum*, leading to the diagnosis tinea incognito. The patient commenced oral treatment with itraconazole 100 mg/day for 4 weeks, in addition to local therapy with ciclopiroxolamine cream once a day with cessation of all topical steroids. In order to decrease the need for systemic corticosteroids and to maintain complete remission of the bullous autoimmune disease with minimum adverse effects, we began maintenance treatment with azathioprine by concurrently tapering the prednisolone daily dosage under 7.5 mg. A follow-up examination 6 months later showed complete resolution of the fungal infection with no recurrence of pemphigus foliaceus.

DISCUSSION
Tinea incognito, first described in 1968 by Ive and Marks (1, 2), is a dermatophytic infection in which topical or systemic steroids, administered as a result of dermatological misdiagnosis or pre-existing pathologies, have modified its clinical appearance. Compared with untreated tinea corporis, tinea incognito usually displays a less raised margin, is less scaly, presents as more pustular, is more extensive and irritable, and can thereby mimic other skin diseases (e.g. pemphigus foliaceus), as described in this case report. In a large retrospective study Romano et al. (4) analysed causative agents, clinical aspects, and sources of infection of 200 cases of tinea incognito. Tinea incognito was found to be due mainly to different *Trichophyton* and *Microsporum* species and clinically presented as lupus erythematosus-, eczema- and rosacea-like on the face and impetigo- and eczema-like on trunk and limbs (4). Furthermore, there have been reports of tinea incognito resembling purpura, seborrhoeic dermatitis, lichen planus, contact dermatitis, psoriasis and erythema migrans (1, 4–6).
Beside these non-systemic infections usually caused by topical application of steroids, systemic immunosuppression can result in the formation of deep subcutaneous abscesses and secondary granuloma formation, known as “Majocchi’s granuloma”, which, for example, can mimic Kaposi’s sarcoma (7–9). Taken together, the great diversity in the clinical picture of tinea incognito can delay proper diagnosis and treatment, as described in this case, where its occurrence was first misinterpreted as a relapse of immunosuppressive-treated pemphigus foliaceus. Once recognized, tinea incognito usually requires systemic treatment with oral antifungal agents. Terbinafine, itraconazole and fluconazole have been shown to be superior to griseofulvin as they accumulate in the skin (10). In our case combined therapy with itraconazole and ciclopiroxolamine for 4 weeks was successful in clearing the fungal infection.

Recent reports and the case presented here demonstrate the need carefully to monitor immunosuppressed patients and to perform frequent skin examinations, as these patients are susceptible to additional clinically atypical superinfections, such as tinea incognito.

REFERENCES