Superficial Fungal Infections

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Fungal infections of the skin and nails are the most common and widespread group of all mycoses; they affect more than 20–25% of the world's population [1]. The incidence of cutaneous mycoses continues to increase, particularly in tropical countries because of the heat and humidity, whereas the prevalence of the causative species of fungi involved has shifted or changed due to migration and changes in socioeconomic status and lifestyle [2]. People with colored skin, especially deeply pigmented or black-skinned populations, show high frequency of superficial mycosis, which may reach up to 41.9% of all dermatoses seen, as has been reported from West Africa [3]; they are frequently registered also in tourists and travelers. The clinical pictures can be subdivided into infections that induce minimal or no inflammatory response, e.g., pityriasis versicolor, tinea nigra, or piedra, and those that induce cutaneous inflammation such as cutaneous candidosis and tinea.

5.1 Pityriasis Versicolor

Pityriasis versicolor (PV) is a superficial fungal infection seen worldwide, with high prevalence observed in hot and humid climates. It affects young adults 20–45 years old, but it is not uncommon also among the children and elderly. There is evidence indicating higher prevalence of PV in males due to increased sebaceous activity [4]. There are conflicting findings on the prevalence of PV in different ethnic populations; in some reports predilection for dark-skinned individuals is registered [5].

PV is caused by different species of genus *Malassezia*, revised into seven, *M. globosa*, *M. restricta*, *M. obtusa*, *M. sympodialis*, *M. furfur*, *M. slooffiae*, and *M. pachydermatis*, as a part of the normal flora of the skin. These are all lipophilic yeasts except *M. pachydermatis*. Although *M. globosa* is considered as the dominant causative agent of PV in many parts of the world [6, 7], several studies revealed that *M. sympodialis* and *M. furfur* are most predominant [8–10].

Clinically, PV is characterized by the presence of fine scaly patches or macules which may be hyper- or hypopigmented; they are usually asymptomatic, and only few patients complain of itching (Fig. 5.1). Most of them are located on the upper part of the trunk, neck, axillae, and arms, although they may be also found on unusual sites such as the lower extremities and face. PV is clinically diagnosed, confirmed through the presence of yeast cells and hyphae in scales taken from lesions after KOH preparation ("spaghetti and meatballs"). Culture is of no need for routine laboratory diagnosis; however, it can be done on media as SDA with olive oil or Dixon's medium with addition of chloramphenicol 0.005 and cycloheximide 0.05, then incubating at 31 °C for 10 days.

Topical azoles (ketoconazole, clotrimazole, or miconazole), ciclopirox, and terbinafine are effective in limited PV lesions. Only in widespread, often recurrent cases systemic therapies with ketoconazole 200 mg/day for 10 days, fluconazole 150 mg/week for 3 weeks, or itraconazole 200 mg/day for 7 days may be warranted. As prophylaxis itraconazole 2× 200 mg once per month is recommended [11, 12].

5.2 Superficial Candidosis

Superficial cutaneous candidosis (candidiasis) includes infections caused by different species of genus *Candida* clinically manifested as *intertrigo*, *diaper dermatitis*,

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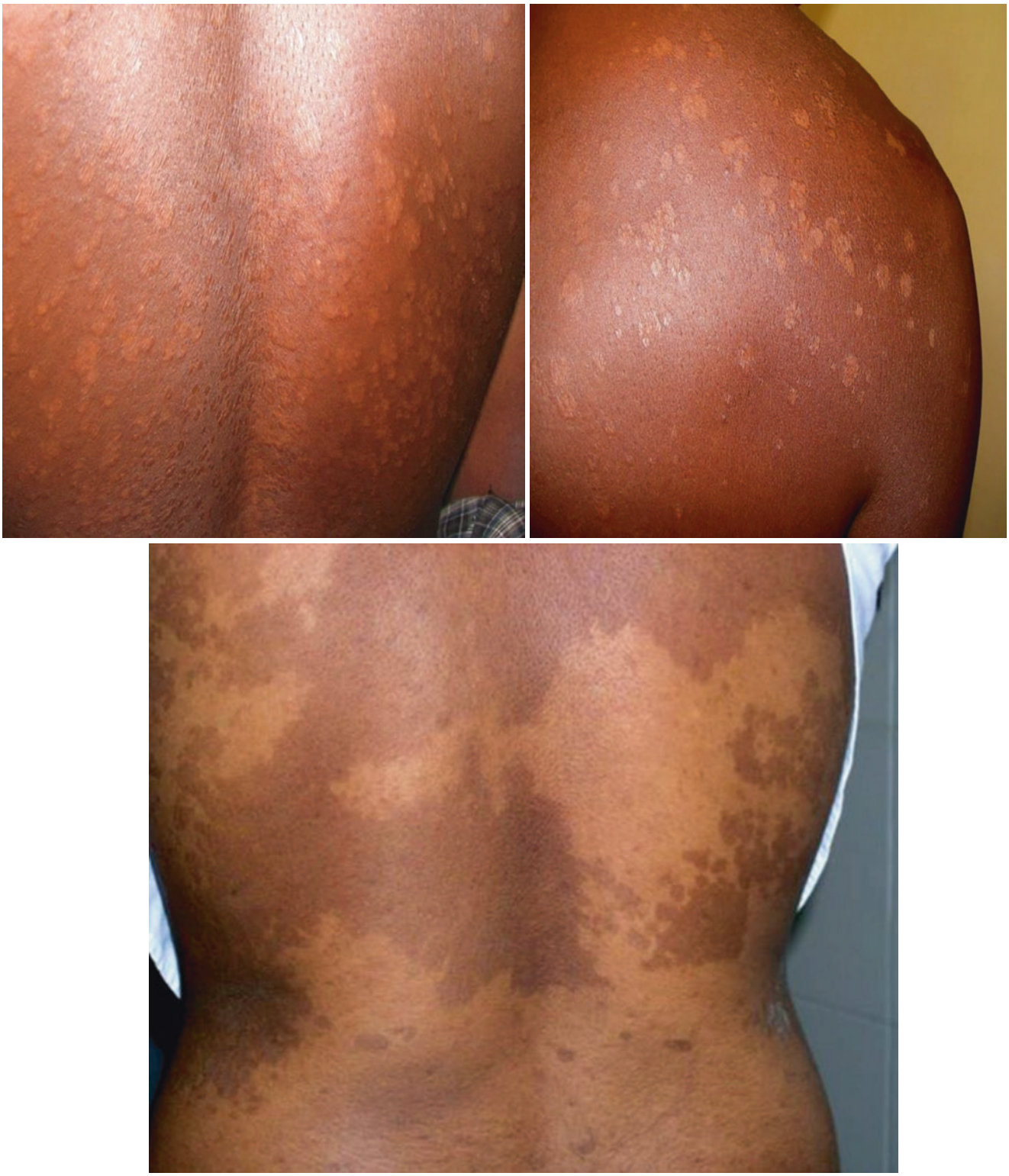

Fig. 5.1 Clinical types of *pityriasis versicolor*: Small hypochromic macules (up) and large hyperchromatic patches on the trunk of young adults

interdigital candidosis, and *candidal paronychia*. The yeast is found on the human skin or mucosa; the infection originates from the patient's own flora, seen in all age groups, often in neonates and the elderly [13]. The most common causative agent is *Candida albicans*; also *C. tropicalis*, *C. parapsilosis*, *and C. glabrata* can be considered in a few instances as a cause. Clinical disease starts when alteration of the host's immune response occurs and the saprophytic dimorphic yeast switches to the pathogenic mycelia form [14]. Mycological diagnosis is done by direct examination after KOH preparations which show blastoconidia and pseudohyphae and through cultures on SDA. *Candida albicans* is characterized by creamy pasty and smooth colonies on SDA, green colonies on chromogenic candida agar (CCA), and the presence of chlamydoconidia on cornmeal agar. Differentiation between *C. albicans* and non-albicans species can be done by growth on CCA, commercial kits such as API and Rap-ID, or by molecular methods, PCR [15] or MALDI-TOF [16].

Candida intertrigo starts as an erythematous pruritic rash with vesiculopustules, which may rupture and cause maceration and fissuring. The area involved has a scalloped border with white rim consisting of necrotic epidermis surrounding the erythematous macerated lesion. Satellite lesions are often found. The disease develops at moist skin of the submammary folds and the perigenital region, spreading out into the inner thigh area (Fig. 5.2). It occurs most frequently in adipose and diabetic patients and those confined to bed. *Diaper dermatitis* is clinically manifested as erythematous, eczematous patches affecting the gluteal, inguinal, perianal, and genital areas. In severe cases papules, erosions, and rarely ulcers may occur. *Interdigital candidosis*, *candidal paronychia*, and *onychomycosis* are also common. Chronic candidal paronychia shows erythema and swelling of the nail folds and brownish discoloration of the nail plate.

For treatment nystatin, azoles, and ciclopirox are usually used topically. Nystatin, fluconazole, and itraconazole are also administered orally for severe recalcitrant cases. The topical combination of an antimycotic or antiseptic compound with a moderate-strength topical corticosteroid is a good choice in inflammatory intertriginous yeast infections [17].

5.3 Tinea (Dermatophytosis)

Tinea (ringworm) is a superficial fungal infection of the skin, hair, and/or nails caused by dermatophytes, classified in *tinea capitis*, *corporis*, *manum*, *cruris*, *pedis*, and *unguium*. The predominant dermatophytes vary with the geographic area; most species are found worldwide, while some others are geographically restricted such as *T. yaoundi* and *T. soudanense* in Africa and *T. concentricum* in Western Pacific and Malaysia. Today, five or six species account for most dermatophytoses globally [18]. Their distribution is influenced by the changing patterns of migration, growth of tourism, and socioeconomic conditions. Significant changes of the dermatophyte spectrum were observed especially in *tinea capitis*: increased incidence of *T. tonsurans* in Southwest and East of the USA, reaching up to 60–91% due to immigration from Latin America; disappearance of *M. audouinii* and *T. schoenleinii* from Central Europe; as well as strongly increased prevalence of *M. canis* in Germany and West Europe [19, 20]. *M. canis* followed by *T. violaceum* and *T. tonsurans* were found to be predominant in China [21]. Increase of *M. canis* and *T. violaceum* is found in the Middle East [22]. *T. violaceum* is still the commonest cause of tinea capitis in India, Southeast Asia, and some regions of Africa (Ethiopia) [23] and in South America (Brazil). Generally, there is a predominance of anthropophilic dermatophytes with *T. rubrum* on the top [24] with the exception of a few countries.

Tinea capitis may be found as a noninflammatory form presenting round demarcated scaly patches with hair loss showing short stubble of broken hairs, caused by *Microsporum* species (Fig. 5.3). Scattered irregular patches of alopecia with indistinct borders show black dots when the hairs break off at the level of scalp leaving stubs (Fig. 5.4a). This form is caused by *T. tonsurans*, *T. violaceum*, and *T. soudanense.* Advanced infection with *M. canis* and *T. verrucosum* with strong host response showing boggy, purulent plaques with abscess formation has been called *kerion*. Some patients with kerion may develop extensive lymphadenopathy [25]. *Favus* is the most severe, inflammatory form of tinea capitis usually caused by *T. schoenleinii* (Fig. 5.4b). It presents as thick, yellow crusts composed of hyphae and skin debris (scutula) which may cover large areas of scalp. In chronic infections scarring alopecia often develops.

Tinea corporis involves the trunk, legs, and arms characterized by annular scaly lesions; also arcuate, gyrate, or circinate shapes may occur (Fig. 5.5a, b). Scales may be lessened or absent if topical corticosteroids were used (tinea incognito). Lesions can also be vesicular, granulomatous, or verrucous in appearance. Associated symptoms include pruritus and burning. Clinical variants of tinea corporis include *tinea profunda* and *tinea imbricate*; sometimes hypermelanotic lesions are seen in pigmented skin (Figs. 5.6 and 5.7). The first results from an excessive inflammatory response to a dermatophyte, corresponding to a kerion on the scalp, may have granulomatous or verrucous appearance, whereas the second is a rare form caused by *T. concentricum* presenting as large, concentric, scaly lesions resembling erythema gyratum repens [26]. Widespread, generalized tinea is observed in immune-compromised patients.

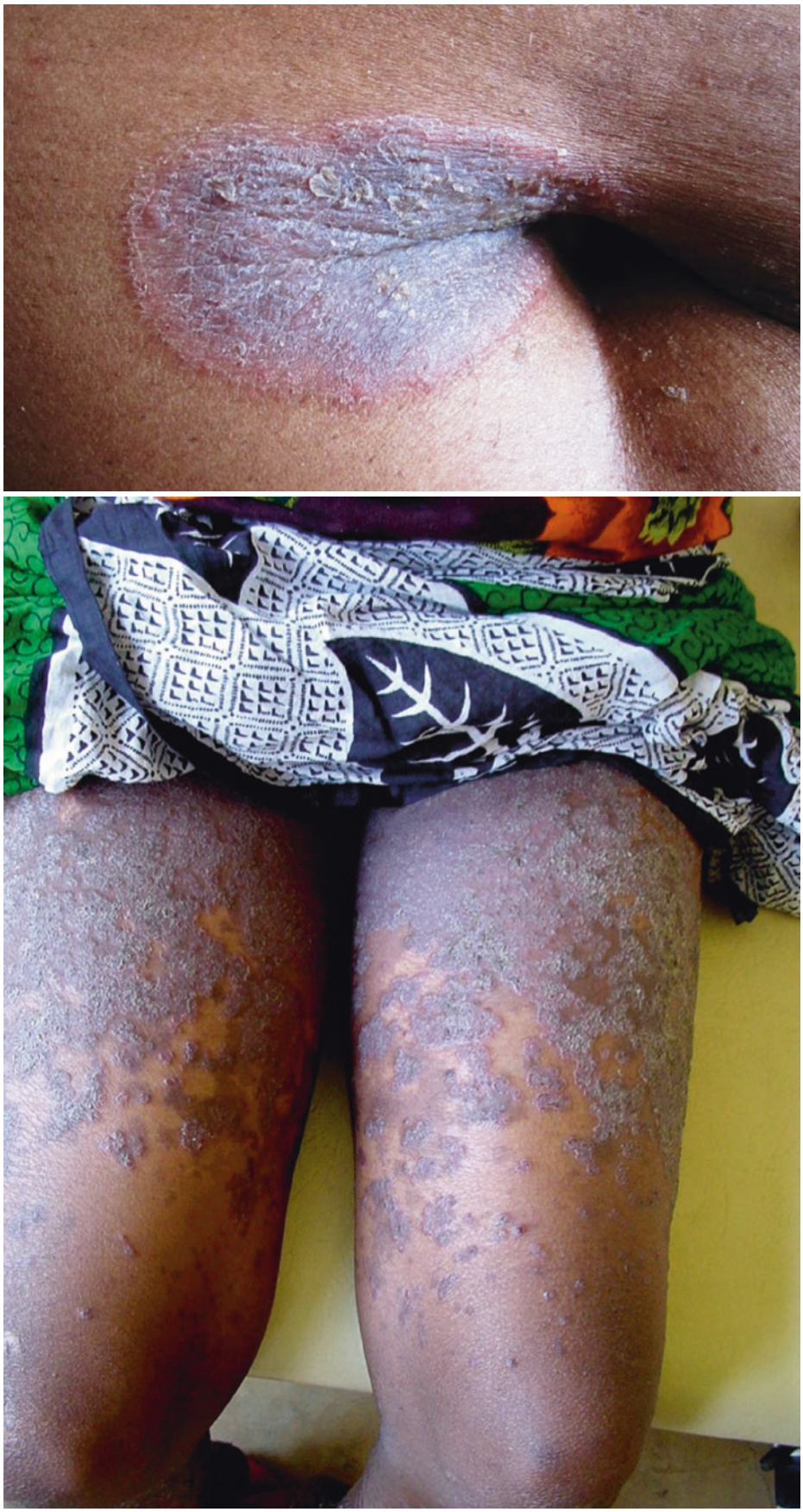

Fig. 5.2 *Candidosis*: Intertrigo type and widespread cutaneous dissemination in a young female (Uganda)

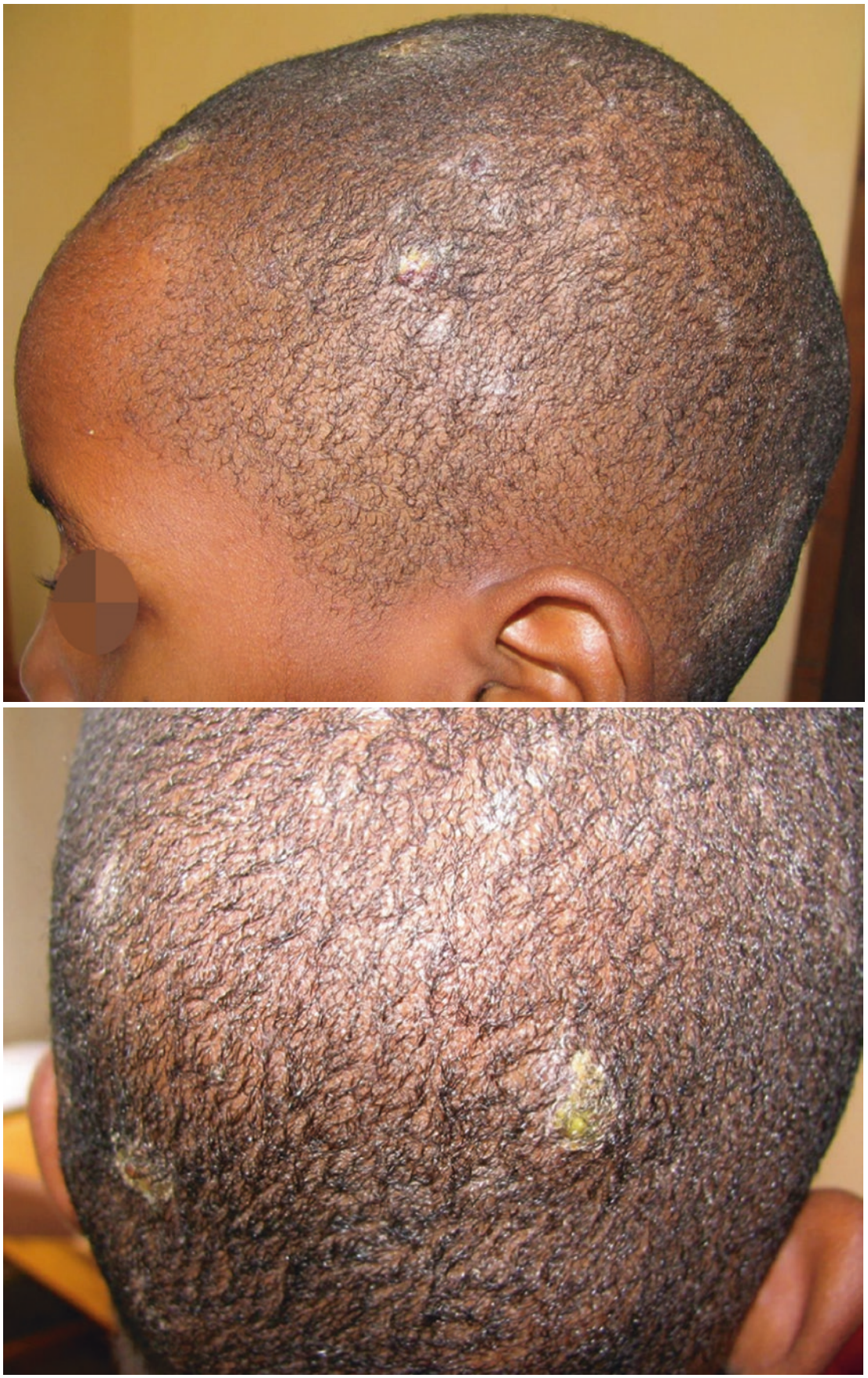

Fig. 5.3 Typical scaly patches of *tinea capitis* (*M. canis*) in young school boys in Tanzania

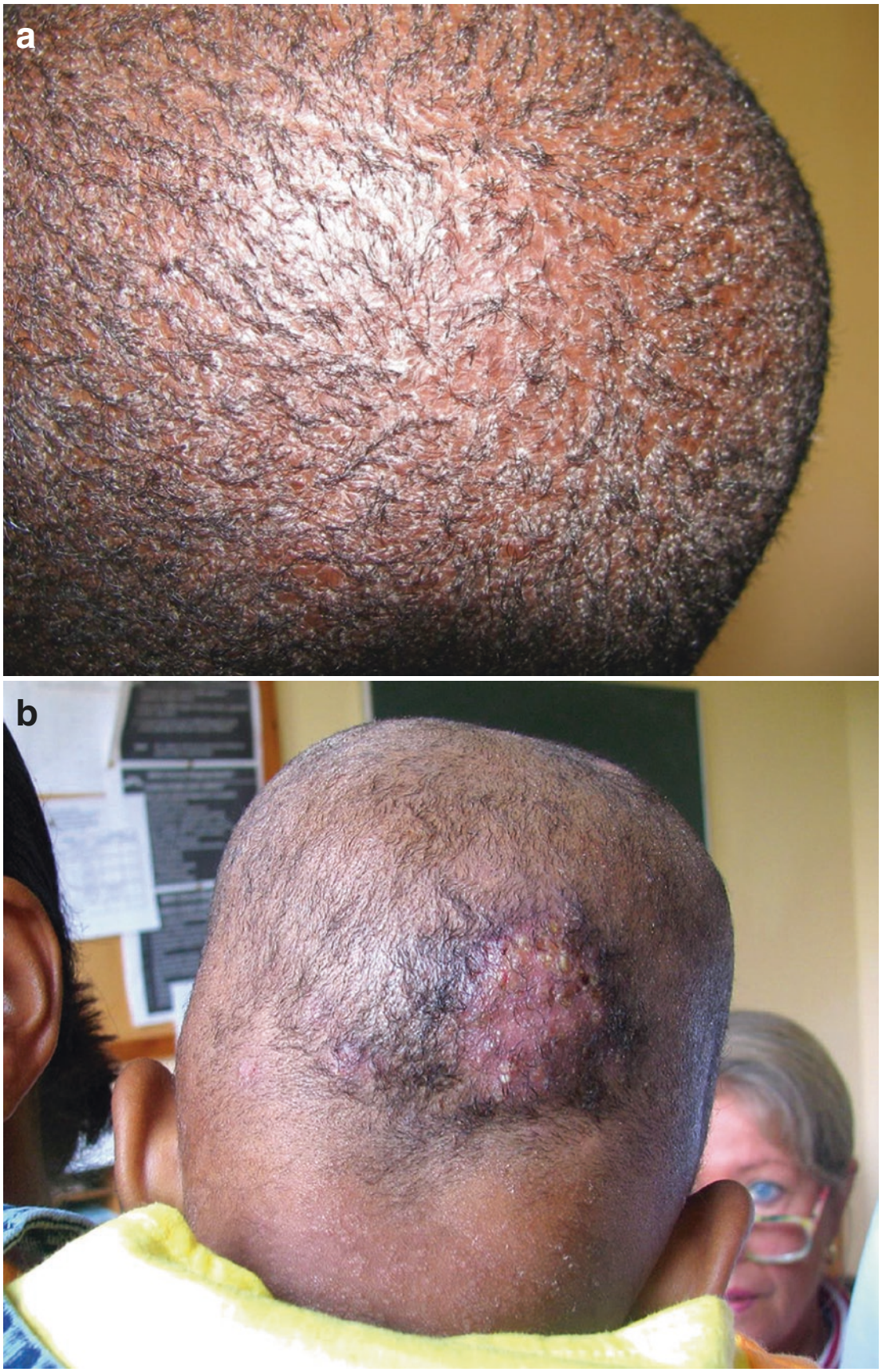

Fig. 5.4 *Tinea capitis.* (**a**). Diffuse fine scaling of the scalp with atrophic scarring alopecia; (**b**). Favus-like inflammatory lesion in a 6-month-old baby (possibly *T. schoenleinii*)

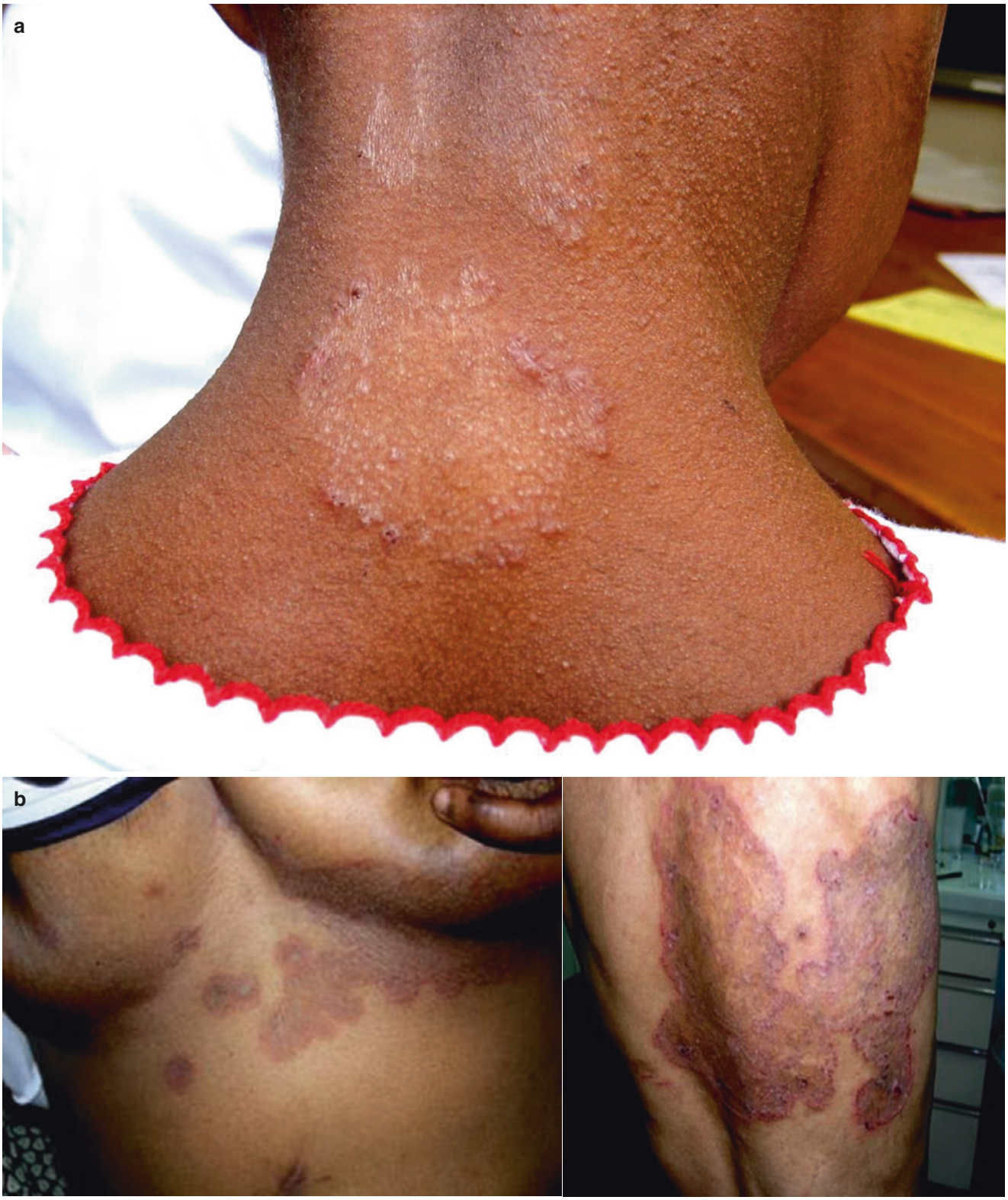

Fig. 5.5 *Tinea corporis:* (**a**). Lesion on the neck in a young school girl, *T. rubrum* (Tanzania); (**b**). Submammary *tinea corporis*, *M. canis* (Egypt); (**c**). *Tinea corporis* on the trunk, *T. rubrum* (Egypt)

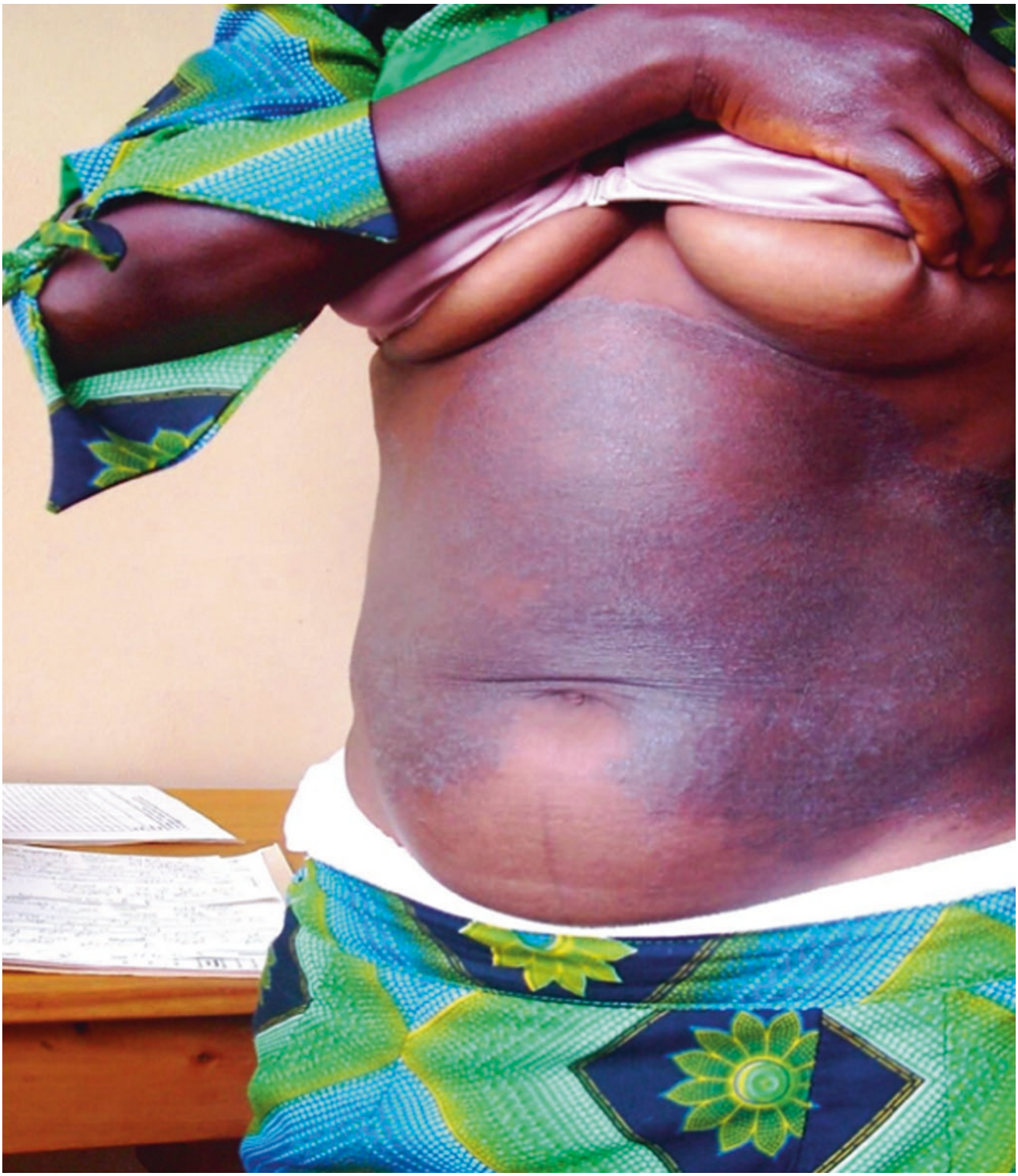

Fig. 5.6 Hypemelanotic *tinea corporis* (most likely *T. violaceum* or *T. soudanense*) in a young female (Uganda)

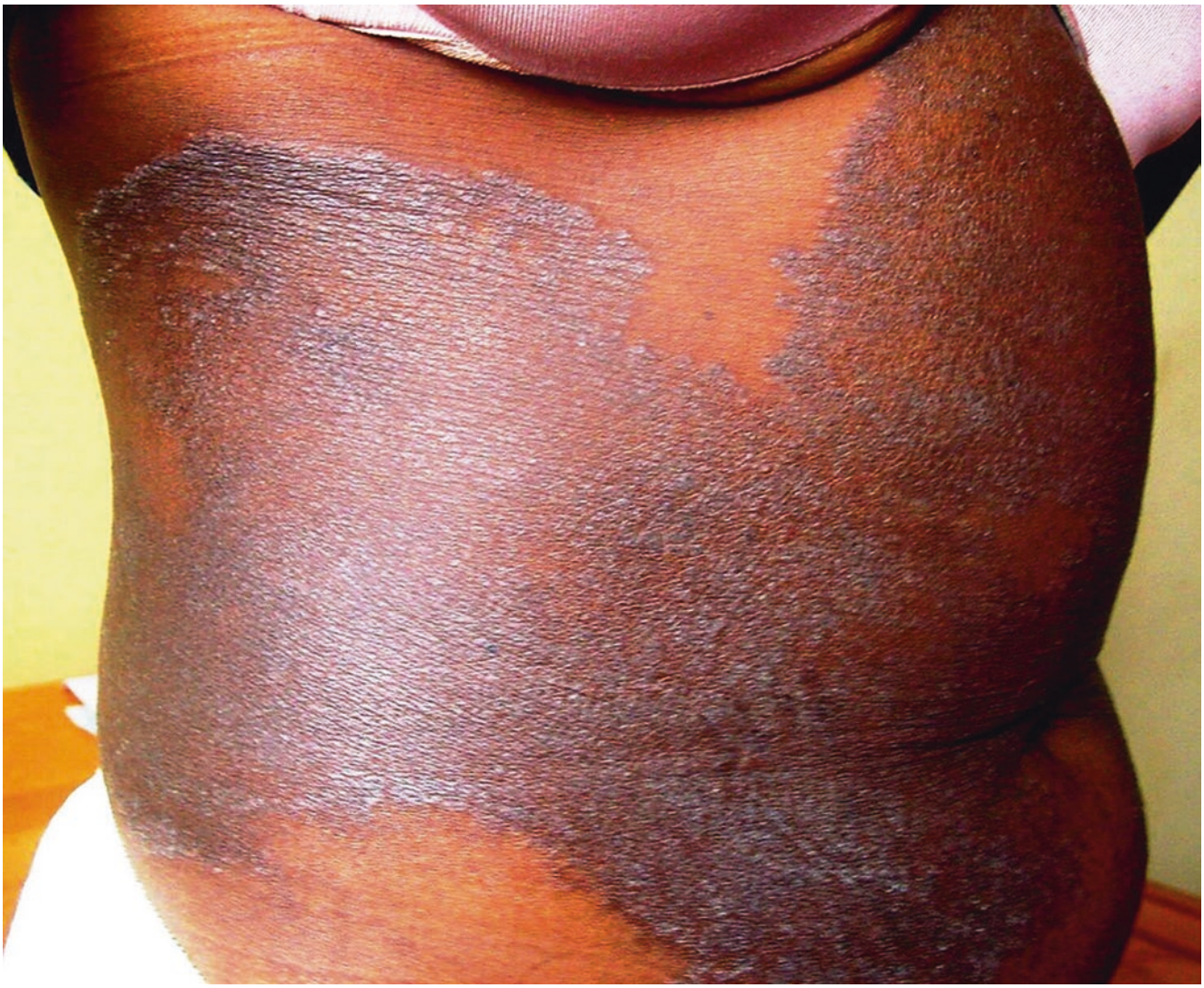

Fig. 5.7 Hypermelanotic *tinea corporis* (unidentified agent)

Tinea manum involves the palms and interdigital spaces caused by *T. rubrum*, *T. mentagrophytes*, and *E. floccosum*. Non-dermatophyte fungi, *Scytalidium dimidiatum* and *S. hyalinum*, can also cause the disease which is usually unilateral, noninflammatory, and showing diffuse hyperkeratosis. The differentiation of the individual agent is well possible in in vitro cultures (Fig. 5.8).

Tinea barbae is typically caused by the zoophilic *T. mentagrophytes* var. *mentagrophytes*, *T. verrucosum*, and *M. canis*. Due to the zoophilic organisms and the large number of terminal hair follicles in the affected area, the clinical presentation tends to be severe, with intense inflammation and multiple follicular pustules. Abscesses, sinus tracts, bacterial superinfection, and even a kerion-like lesion may develop. Noninflammatory forms caused by *T. violaceum* and *T. rubrum* resembling tinea corporis are also known.

Tinea cruris is an infection of the inguinal region, in particular the inner aspects of the upper thighs and crural folds, with extension on the abdomen and buttocks in more severe cases. While occurring worldwide in men and women, it is more common in tropical environment, caused

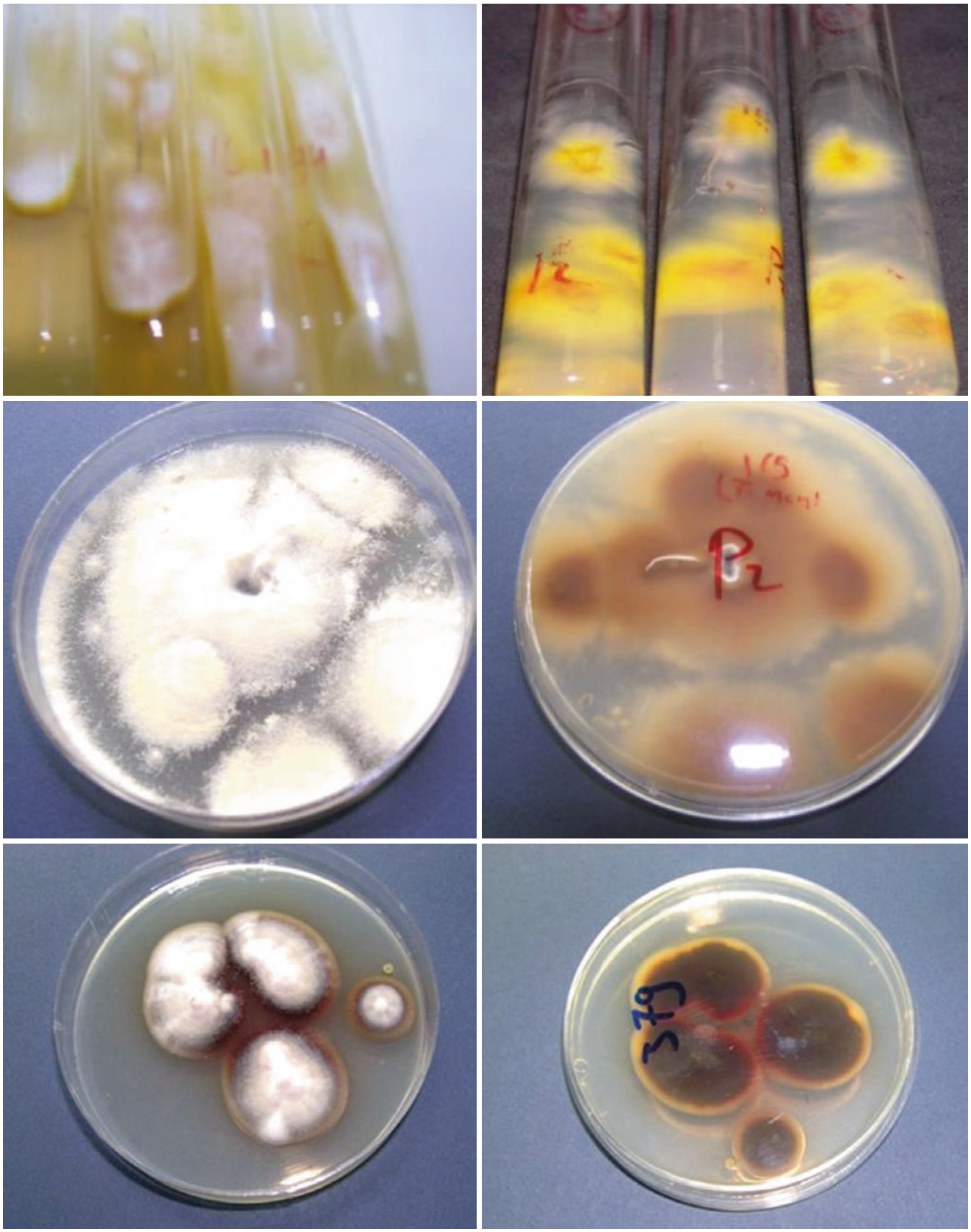

Fig. 5.8 Characteristics of cultures of common dermatophytes. Up: Culture of *M. canis* on SDA with yellow color in reverse. Middle: culture of *T. mentagrophytes* var. *mentagrophytes* with brownish color in reverse. Down: culture of *T. rubrum*, front and reverse

by *T. rubrum*, *T. mentagrophytes*, and *E. floccosum*. Characteristic lesions are sharply demarcated with a raised erythematous scaly advancing borders, often with containing vesicles or pustules. Beginning as circinate in shape, the lesion may become serpiginous, remain unilateral, or become bilateral and symmetric *tinea pedis*. The feet are a common location for dermatophyte infection in adults, affecting both sexes. The lack of sebaceous glands and the moist environment created by wearing occlusive shoes are contributing factors. It may involve the entire plantar surface covered with fine white scaling, sometimes vesicles and pustules, or be limited in the interdigital spaces, characterized by oozing, maceration, and fissuring (Fig. 5.9a) (Table 5.1).

Treatment of dermatophyte infections depends on the clinical type and the age of the patient. Topical antifungal preparations such as Whitfield ointment, sulfur in creams, and Castellani solution are still used in some developing countries for their low cost, but azoles, allylamine, terbinafine, and ciclopirox are being gradually introduced. Systemic management is needed in tinea capitis, chronic tinea corporis, tinea pedis, and others which do not respond to topical treatment. Particular care is to be taken for management in children [27, 28].

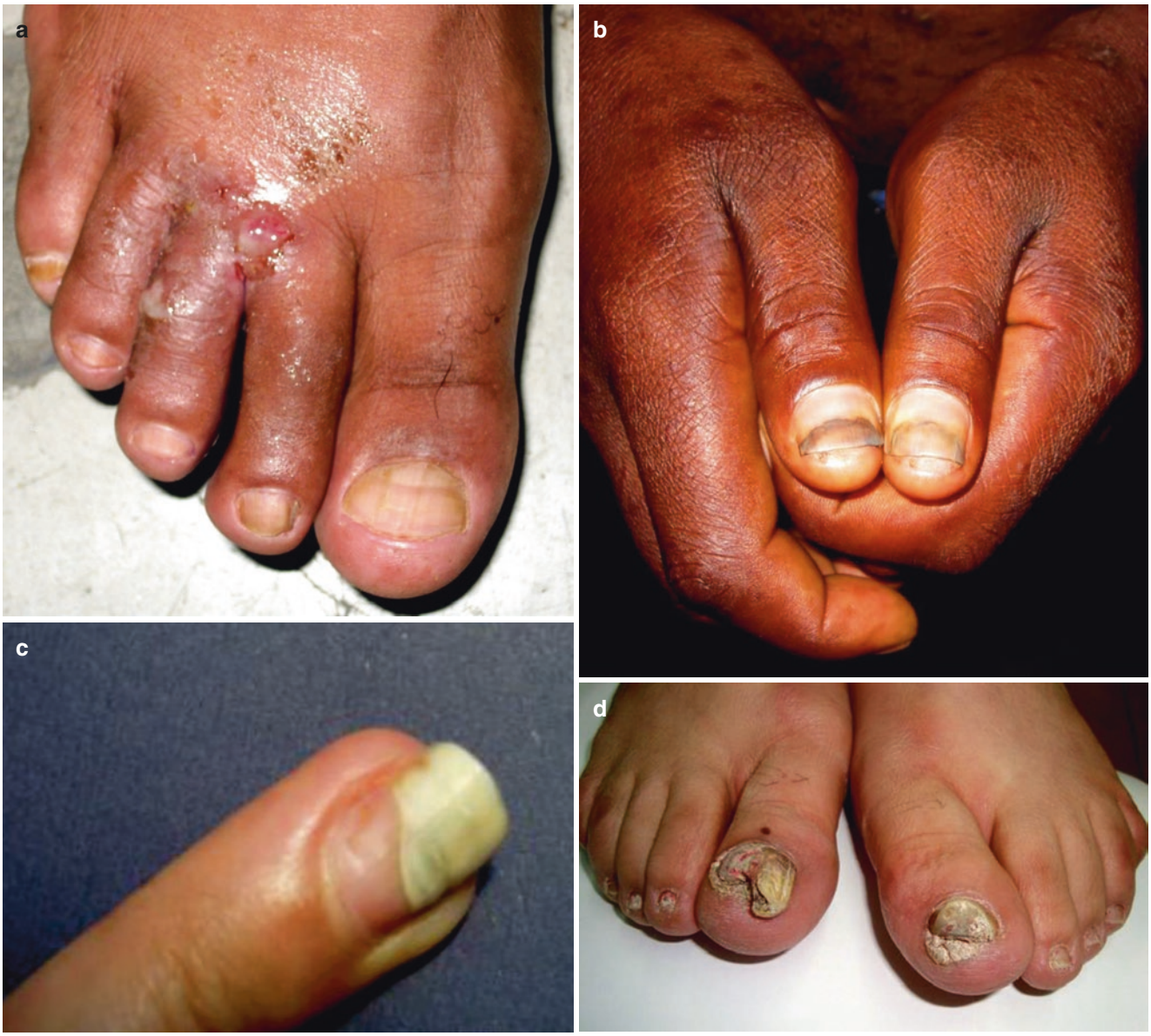

Fig. 5.9 (**a**). *Tinea interdigitalis*, superinfected; (**b**). *Proximal subungual onychomycosis* (PSO); (**c**). *Distal lateral subungual onychomycosis* (DLSO); (**d**). *Total dystrophic onychomycosis* (TDO)

Table 5.1 Treatment of tinea

Clinical type	Topical	Systemic
Tinea capitis	Shampoo as adjunct management	Griseofulvin (ultra-micronized suspension), 10 mg/kg/day for 6–12 weeks Terbinafine suspension, 7 mg/kg/day, for 6 weeks Fluconazole, 8 mg/kg once a week, for 8 weeks Itraconazole, 5 mg/kg/day, for 6 weeks
Tinea corporis/ tinea cruris	Azoles 2% 1×/day for 1–2 weeks Terbinafine 1–2×/day for1–4 weeks Ciclopirox 2×/day for 4 weeks	Terbinafine, 250 mg/day, for 2–4 weeks Fluconazole, 150 mg once/week, for 2–4 weeks Itraconazole, 200 mg/day, for 1 week
Tinea pedis/ tinea manum	Azoles 2% 1×/day for 6 weeks Terbinafine 1% 1×/day for 6 weeks Ciclopirox 0.77% 2×/day for 1 week	Terbinafine, 250 mg/day, for 2 weeks Fluconazole, 150 mg once a week, for 2–4 weeks Itraconazole, 200 mg 2×/day, for 1 week

5.4 Tinea Nigra

Tinea nigra is an uncommon superficial fungal infection of the stratum corneum caused by *Hortaea werneckii*, in rare instances by *Stenella araguata*, having a predilection for tropical and subtropical regions. Most reports originate from Latin America [29, 30] and Asia [31]. The disease may present as an imported condition from endemic regions to temperate climate zones, usually affecting children and young adults [32, 33]. *H. werneckii* belongs to the order *Capnodiales* in *Ascomycota* and is a halotolerant and halophilic fungus with its natural habitat in hot climate zones, in areas containing high concentration of salt as beach soil, salt pans, and seawater [34]. *Hortaea* has the ability to survive in high salinity and low pH; it thrives on the skin by production of polysaccharides and feeds by assimilation of decomposed lipids. The black color of the lesions results from the accumulation of melanin-like granules in the fungus. Clinically, the lesions are characterized by light brown to black macules which gradually transform into non-scaly patches. The affected areas lack erythema or induration; they usually occur on the palm or soles and may reach 1–5 cm in diameter. Most cases are unilateral. Using dermatoscope, wispy brown strands or pigmented spicules are recognizable and differentiate it from melanoma. KOH preparations of skin scrapings reveal dark pigmented branching hyphae. Culture of skin scrapings on SDA with chloramphenicol and cycloheximide at 25–28 °C reveals slow-growing dematiaceous isolates characterized by moist and shiny black yeast-like that turns to moldy phase with greenish black color. Microscopically, brown septated hyphae and conidia with transverse septa are seen. Dermoscopy may be useful for diagnosis [35].

Treatment is usually done using keratolytic agents such as urea, salicylic acid, and Whitfield ointment, but topical azoles such as miconazole and ketoconazole, terbinafine, or ciclopirox are preferred over several weeks [36]. Most oral antifungal drugs are not effective.

5.5 Onychomycosis

Onychomycosis represents 30% of superficial mycosis and 50% of all nail disorders, with increasing incidence in the elderly. It is caused by dermatophytes such as *T. rubrum* and *T. mentagrophytes*, by yeasts especially *C. albicans* and *C. parapsilosis*, and rarely by non-dermatophyte molds [37–39]. While dermatophytes are the most common agents in temperate countries, non-dermatophytes predominate in hot climate zones. The site and pattern of invasion lead to different clinical types: one may clinically classify [40] a *distal lateral subungual onychomycosis* (DLSO), a *superficial white onychomycosis* (SWO), a *proximal subungual onychomycosis* (PSO), the *endonyx* type, and a *total dystrophic onychomycosis* (TDO; see Fig. 5.9b–d). DLSO is the most common type, characterized by yellowish brown discoloration of the nail plate with onycholysis and subungual hyperkeratosis; PSO is rare and appears as onycholysis showing white to creamy or yellow crumply patches on the proximal part of the nail surface, while TDO affects the nail plate and often also the surrounding periungual tissue. Candidal onychomycosis mostly presents the DLSO type. Mixed types of onychomycosis may occur. Rarely, endonyx infections are caused by *T. violaceum* and *T. soudanense.* Diagnosis is done by conventional KOH preparations, histological examination of nail clippings after PAS staining, and cultures.

Onychomycosis may represent an independent and important predictor for development of diabetic foot syndrome and foot ulcer [40] and is difficult to cure due to the high rate of recurrences [41]. Topical treatment is sufficient for infected nail plates, while oral management is recommended for adequate cure when the nail matrix is affected. Other factors, such as the number of the affected toenails/fingernails, their size, multi-morbidity of the patient, drug interactions, as well as the pathogen agent involved, are also considered. The selection of the antifungal drug depends on the fungus isolated, especially in infections caused by non-dermatophytes (see Table 5.2) [42, 43].

The combination of topical and/or systemic antimycotic treatment with laser sessions (0.65 ms pulsed Nd:Yag laser, two to three sessions every 3 weeks) is a frequently used regimen, despite the absence of valid supporting study data.

5.6 Piedra

Piedra is a nonaggressive fungal infection characterized by nodular lesions formed around the hair shaft [44]. *White piedra* occurs more frequently in regions of tropical and mild climates such as in South America, the Middle East, India, Southeast Asia, Africa, parts of Japan, Europe, and Southeast USA. Causative agents are species of *Trichosporon*, a yeast widely distributed in nature, possibly also a part of normal flora of the human skin and nail. White piedra nodules can be seen by the naked eye usually localized in the distal part of the hair shaft and be easily detached. Between the nodules is the hair shaft, normal; no broken hairs are seen. The nodules can affect scalp hair, beard, moustache, and also pubic hairs [45]. In children the disease is more prevalent in the scalp hair [46]. Examination of infected hairs with 10% KOH shows the fungal elements as rounded arthrospores. Culture can be done by inoculating affected hair on Sabouraud's dextrose agar (SDA) and incubating for 7 days at 30 °C. The yeast appears as creamy rough and heaped with wrinkled surface colonies (Fig. 5.10). Individuals with black skin were

Table 5.2 Topical and systemic treatment of onychomycosis

Management	Fingernails	Toenails
Topical: Ciclopirox 8%	Once daily for 24 weeks	Once daily for 48 weeks
Topical: Amorolfine 5%	Once or twice weekly for 24 weeks	Once or twice for 36–48 weeks
Systemic: Fluconazole	150 mg/week for more than 6 months	150 mg weekly for more than 6 months
Systemic: Itraconazole	Pulse management: 200 mg twice/day for 1 week /month (2–3 pulses)	Pulse management, 200 mg/day for 12 weeks or 4–6 pulses
Systemic: Terbinafine	250 mg/day for 6 weeks	250 mg/day for 12 weeks

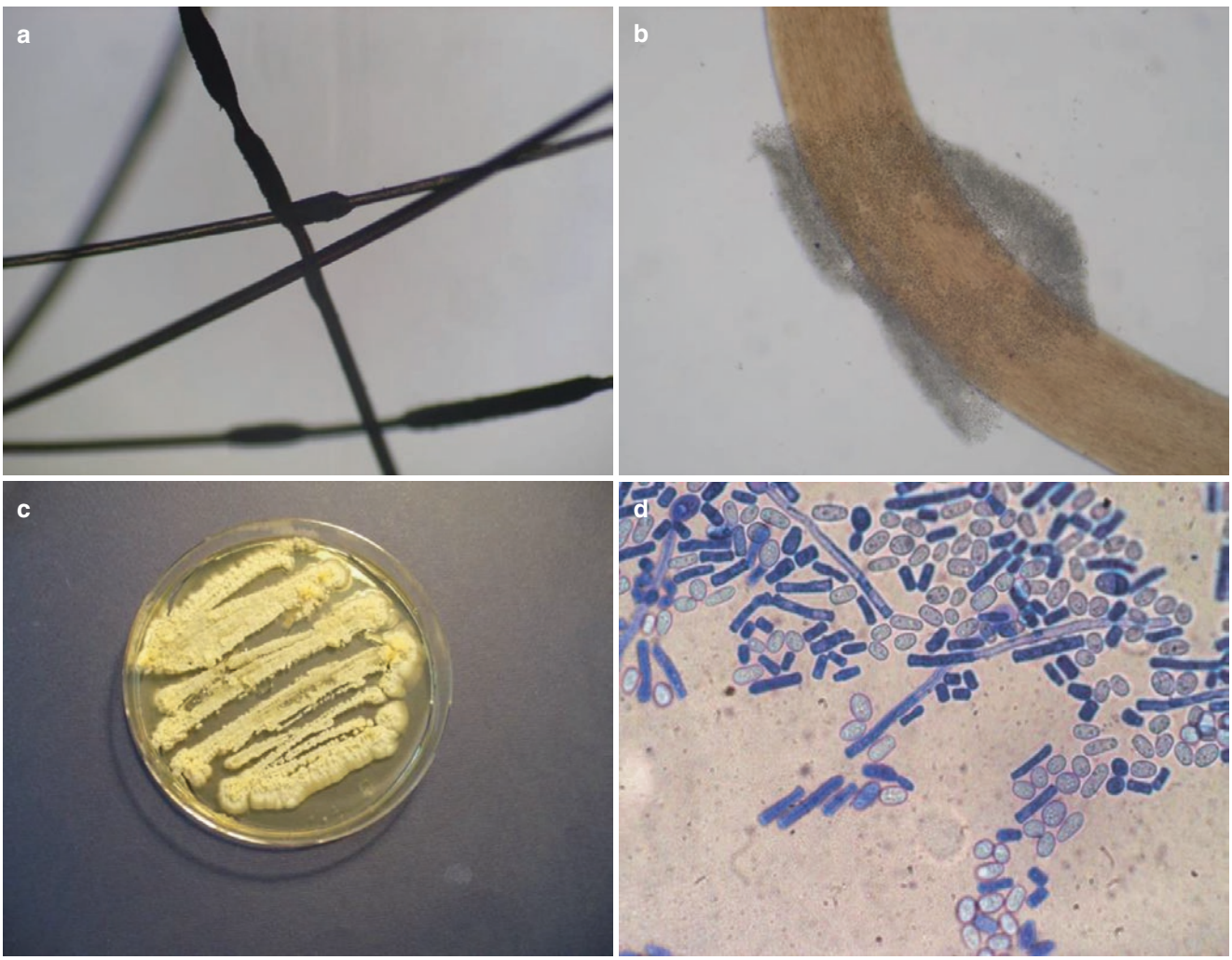

Fig. 5.10 *White piedra*: (**a**) nodules with different lengths; (**b**) nodules surrounding the hair shaft; (**c**) *Trichosporon* spp. colonies on SDA; (**d**) microscopic characters of *Trichosporon* spp. isolated from white piedra

found to be more frequently affected, preferably males 18–35 years old [46]. Best treatment is to shave the hair along with washing with 2% ketoconazole shampoo once a week for 3 months. A combination of an oral azole antifungal for 1 month together with topical treatment for 2–3 months may be also effective.

Black piedra is caused by an ascomycetous dematiaceous fungus, *Piedra hortae* [44]. It is prevalent in tropical countries, especially in South America and Southeast Asia. Due to world travel, it may occur sporadically in other regions. Adults tend to be significantly more often affected than children, without preference of gender. The fungus is keratolytic and forms its ascostroma within the nodules. KOH preparations of nodules show colored hyphae with arthroconidia, while mature nodules show banana-shaped asci with elongated ascospores. Culture of nodules on Sabouraud's medium containing cycloheximide reveals slow growth of compact brown-black colonies with brown thick-walled septated hyphae, chlamydoconidia, asci, and ascospores. On clinical examination the affected hairs generally show four to eight small dark brown and hard nodules strongly cemented to the hair shaft. Scalp hair is the most often affected, although beard, moustache, and pubic hairs may also be involved [44, 47]. Mixed black and white piedra in the same patient has been observed [48]. Shaving or cutting the affected hair and washing by azole shampoos proved to be ineffective; oral terbinafine 250 mg/day for 3–6 weeks was found successful [49].

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