Infestations and Parasitoses

16

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Parasitism is a form of symbiosis in which an organism (parasite) lives in or on another organism and benefits at the expense of the host. This host-parasite association may eventuate to the injury of the hosting organism. Parasites may be grouped in *endo*- and *ectoparasites*. Infections with endoparasites can cause systemic diseases with possible dermatological signs, whereas infestations with ectoparasites only cause cutaneous lesions. The burden of parasitoses often rests on communities in countries of hot climate zones, but parasitic infections and infestations are also on the rise in clinical practices in developed countries due to the increase of traveling individuals and migrating populations. The debilitating impact which a persistent itch of certain parasitosis may have must be taken into account when assessing these conditions. Ectoparasites can also be vectors of pathogens, as discussed in other chapters; in humans they are comprised of two major animal groups, parasitic *arachnids* and parasitic *insects* [1], whereby parasitic arachnids include ticks and mites. As for the ectoparasitic insects, they are exemplified by mosquitoes, tsetse flies, fleas, and lice (Table 16.1).

Table 16.1 Cutaneous parasitic infestations

	Parasites	Cutaneous infestations
Ectoparasites	*Sarcoptes scabiei* var. *hominis*	Scabies
	Pediculus humanus (capitis, corporis)	Pediculosis (capitis, corporis)
	Pthirus pubis	Pediculosis pubis
Helminths	*Ancylostoma* spp.	Cutaneous larva migrans
	Strongyloides spp.	Larva currens
	Schistosoma spp.	Cutaneous schistosomiasis
	Enterobius vermicularis	Cutaneous enterobiasis
	Taenia solium	Cutaneous cysticercosis
	Wuchereria bancrofti, *Brugia malayi*, *Brugia timori*	Acute dermatolymph-angioadenitis (lymphatic filariasis)
Protozoa	*Entamoeba histolytica*	Cutaneous amoebiasis

16.1 Scabies

Scabies is one of the most common parasitic conditions, accounting for a substantial proportion of dermatological care in developing countries. Globally, scabies affects more than 130 million people at any time. It is an important communicable endemic disease in resource-poor countries and has been added in 2013 to the list of neglected tropical diseases by the World Health Organization [2, 3]. Rates of scabies occurrence vary in the recent literature from 0.2% in several countries to 71% on the islands of the Pacific Ocean (Oceania) [4]. In countries with Arabic populations, the approximate prevalence of scabies is up to 2.2%, in African countries up to 9.2%. In 2010, it was estimated that the direct effects of scabies infestation on the skin alone led to more than 1.5 million years lived with disability, and the indirect effects of complications on renal and cardiovascular function are far greater. In resource-poor tropical settings, the sheer burden of scabies infestation, as well as their complications, imposes a major cost on health-care systems.

The disease is caused by the human scabies mite *Sarcoptes scabiei* acquired by personal direct skin contact. The mite lays eggs in a previously burrowed tunnel of up to 4 mm into the superficial layer of the epidermis.

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16.1.1 Clinical Pictures

Highly pruritic papules with or without excoriations and S-shaped burrows are the classical skin lesions leading to the clinical diagnosis of scabies. At first infestation, symptoms usually do not appear for up to 2–6 weeks; during that time, however, affected persons can spread the mites even without showing visible clinical features. At reinfestation, lesions appear much sooner, 1–4 days after exposure.

Itching and rash may be limited to common predilection sites, such as the finger web spaces, the flexor surfaces of the wrists and elbows, armpits, genital organs, nipples, along the belt line, lower buttocks, and shoulder, or affect much of the body surface (Fig. 16.1). The posterior auricular folds, head, face, neck, palms, and soles often are involved in infants and young children. In chronic scabies, the lesions may become eczematous and hyperkeratotic, thus masking the disease (Fig. 16.2). In dark skin, scabies may also manifest as granulomatous small nodules [5]. The most common and characteristic symptom of scabies infestation is severe pruritus, especially at night, followed by a non-characteristic papular rash caused by sensitization of the host to the proteins and feces of the parasite. Generalized involvement may remain unrecognized and be treated with topical corticosteroid creams and ointments (Figs. 16.3 and 16.4).

Crusted (Norwegian) scabies is a severe form that often affects the elderly, patients with conditions that prevent from itching or scratching (spinal cord injury, paralysis, loss of sensation, mental debility), and also immunocompromised individuals. Vesicles and thick crusts occur that usually contain large numbers of mites (Figs. 16.5 and 16.6). Notably, itching may be absent due to the reduced general condition of the patient. Patients with crusted scabies are highly contagious.

The intense itching of all types of scabies often leads to super infections with *S. aureus* or β-hemolytic streptococci, with the risk of post-streptococcal glomerulonephritis, particularly in children (Fig. 16.7).

Diagnosis of scabies is suspected when pruritus is out of proportion to the physical findings [6] and is confirmed by finding the mite, ova, or fecal pellets on microscopic examination. Scrapings should be obtained after placing glycerol, mineral oil, or immersion oil over a burrow or papule to prevent dispersion of mites while unroofing the papule with the edge of a scalpel. The obtained material is placed on a slide and covered with a cover slip for examination under a light microscope.

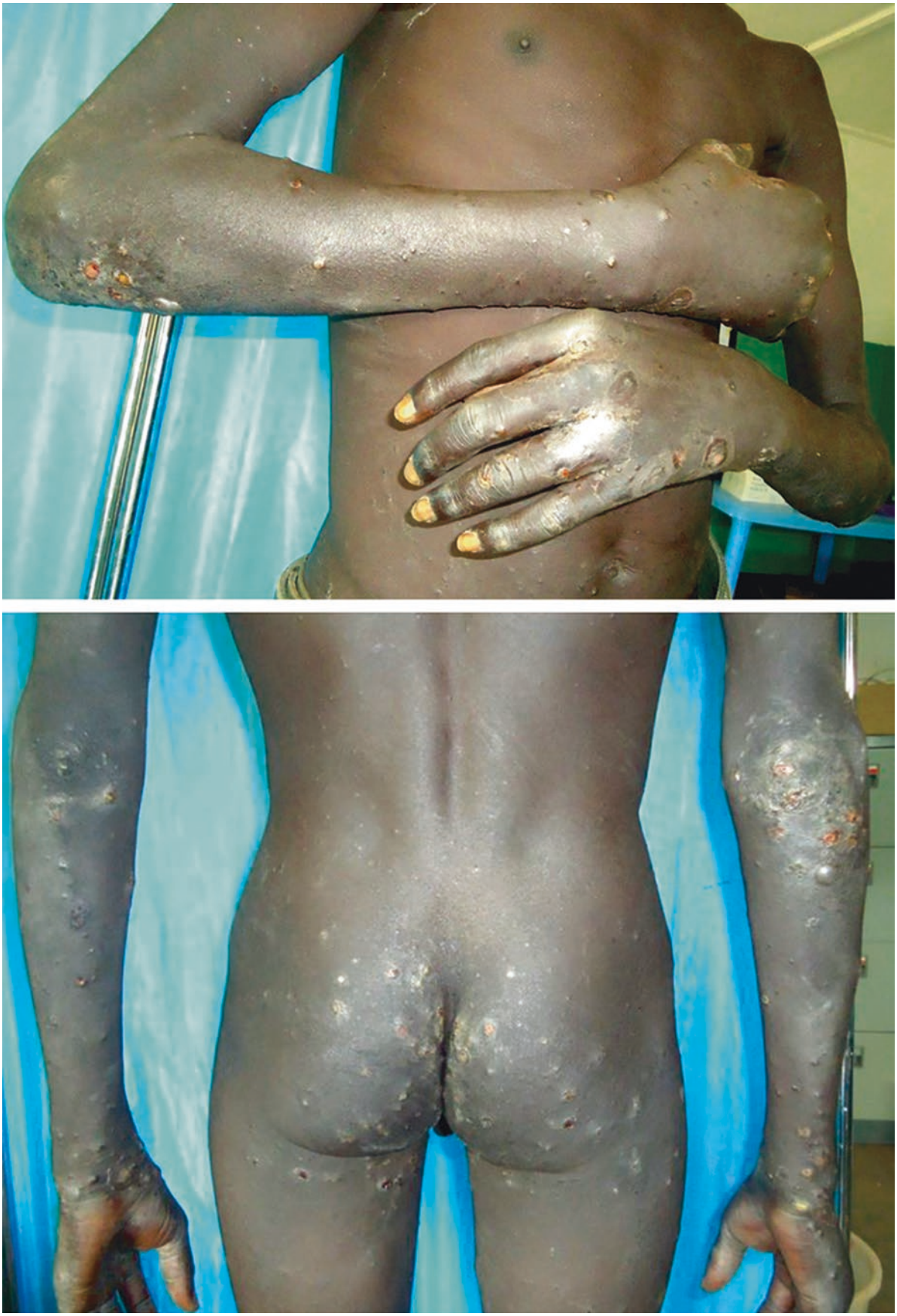

Fig. 16.1 *Scabies.* Disseminated lesions in an immunocompetent adult localized at predilection areas between the fingers, the elbows, and flexor surfaces of the wrists and the buttocks, partly super infected

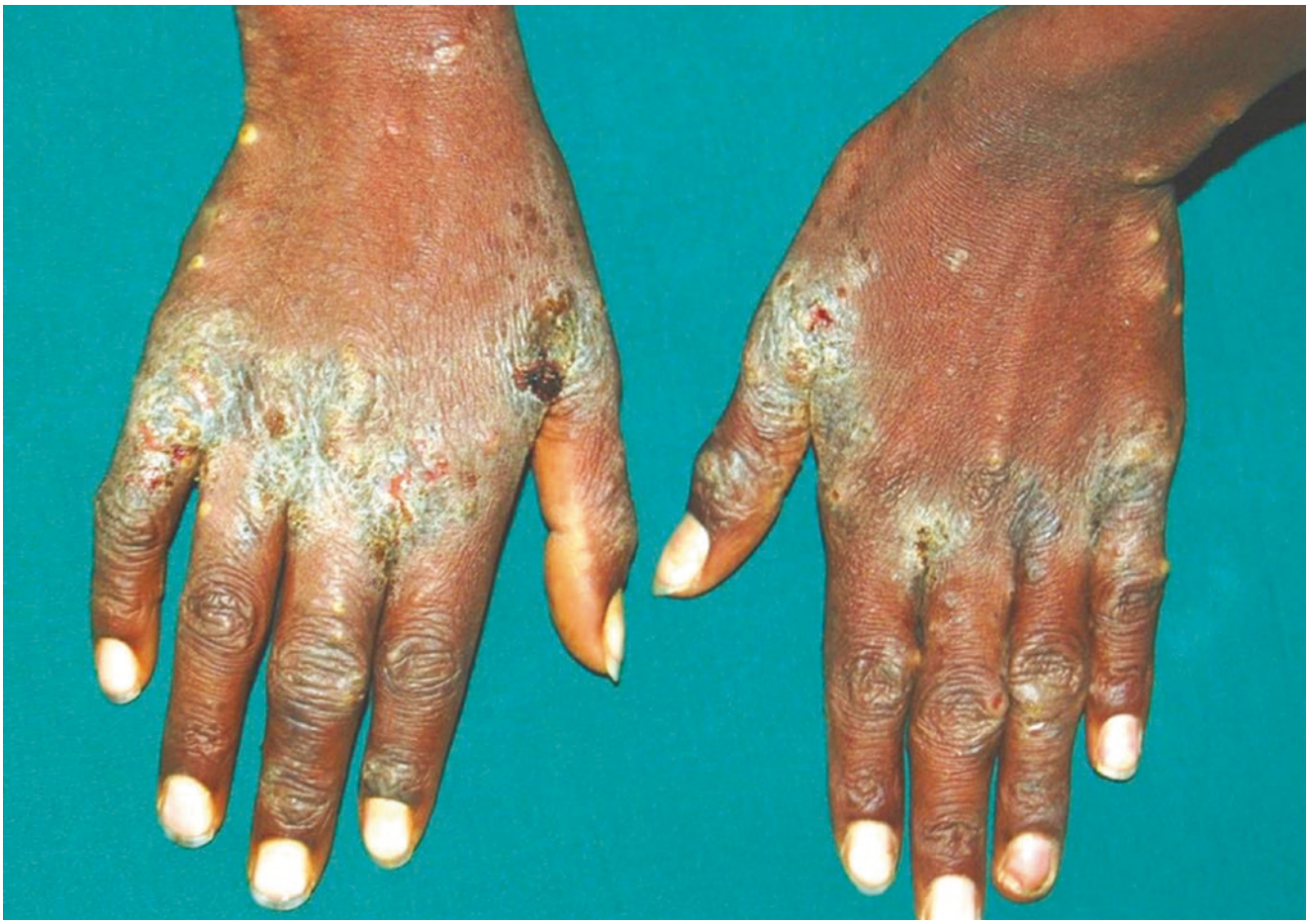

Fig. 16.2 Eczematization of chronic *scabies* with crusts on both hands

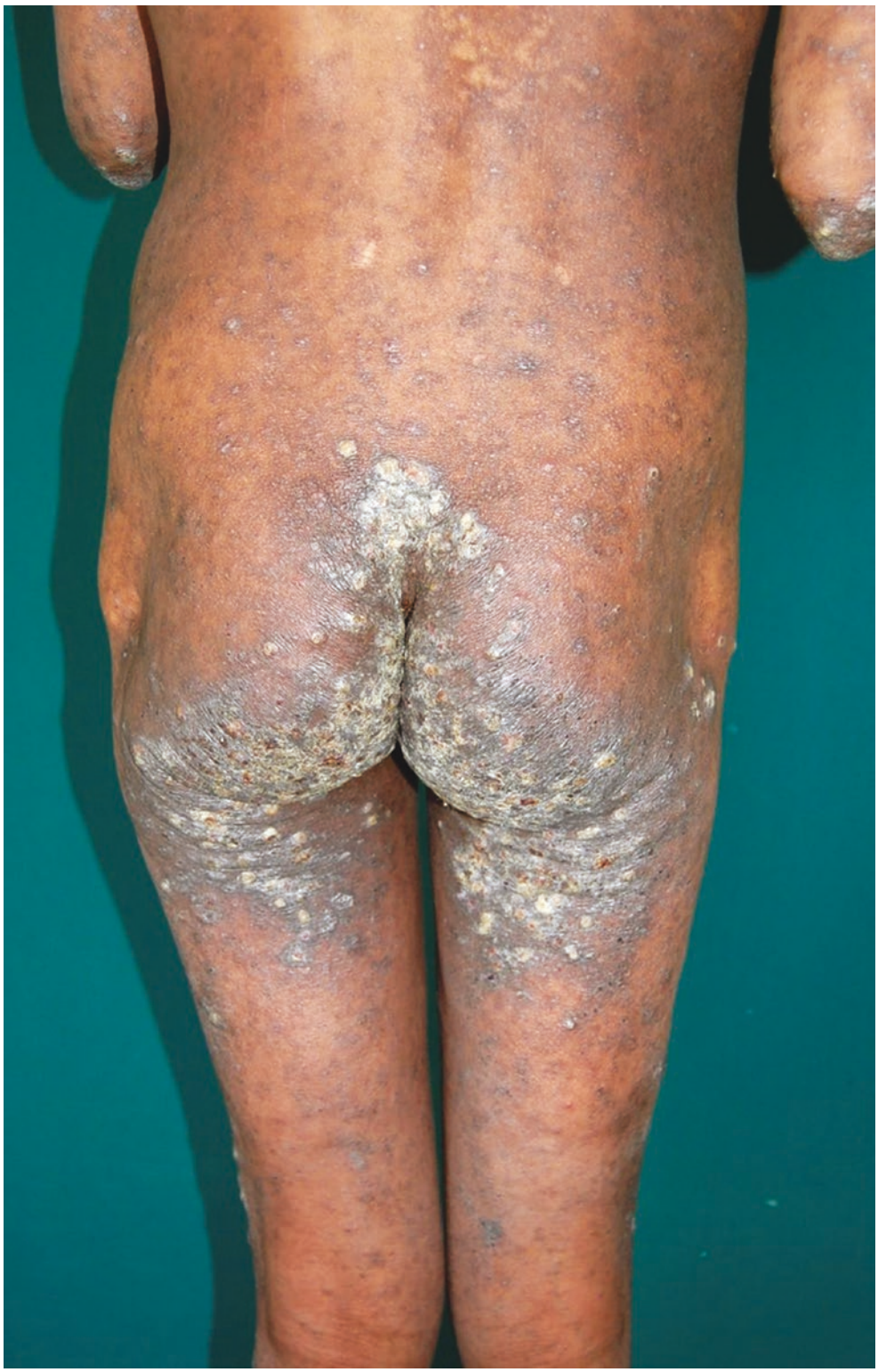

Fig. 16.3 Severe *generalized scabies* in an Ethiopian female immunosuppressed patient, partly eczematized and scaly. Scabies in immunosuppressed individuals is clinically more severe

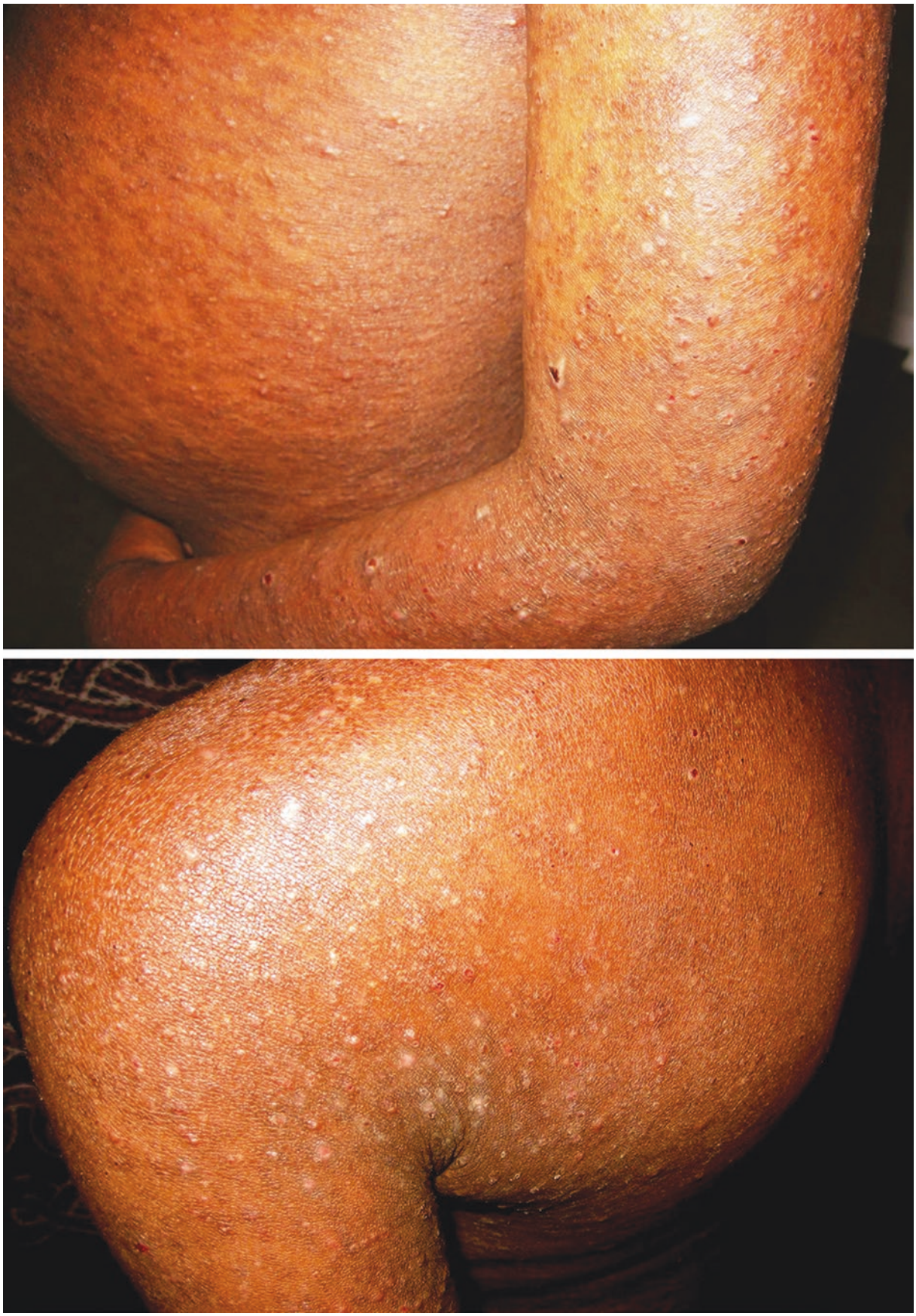

Fig. 16.4 *Generalized scabies*, treated with topical corticosteroids in an East-African young male (Photos: CEO)

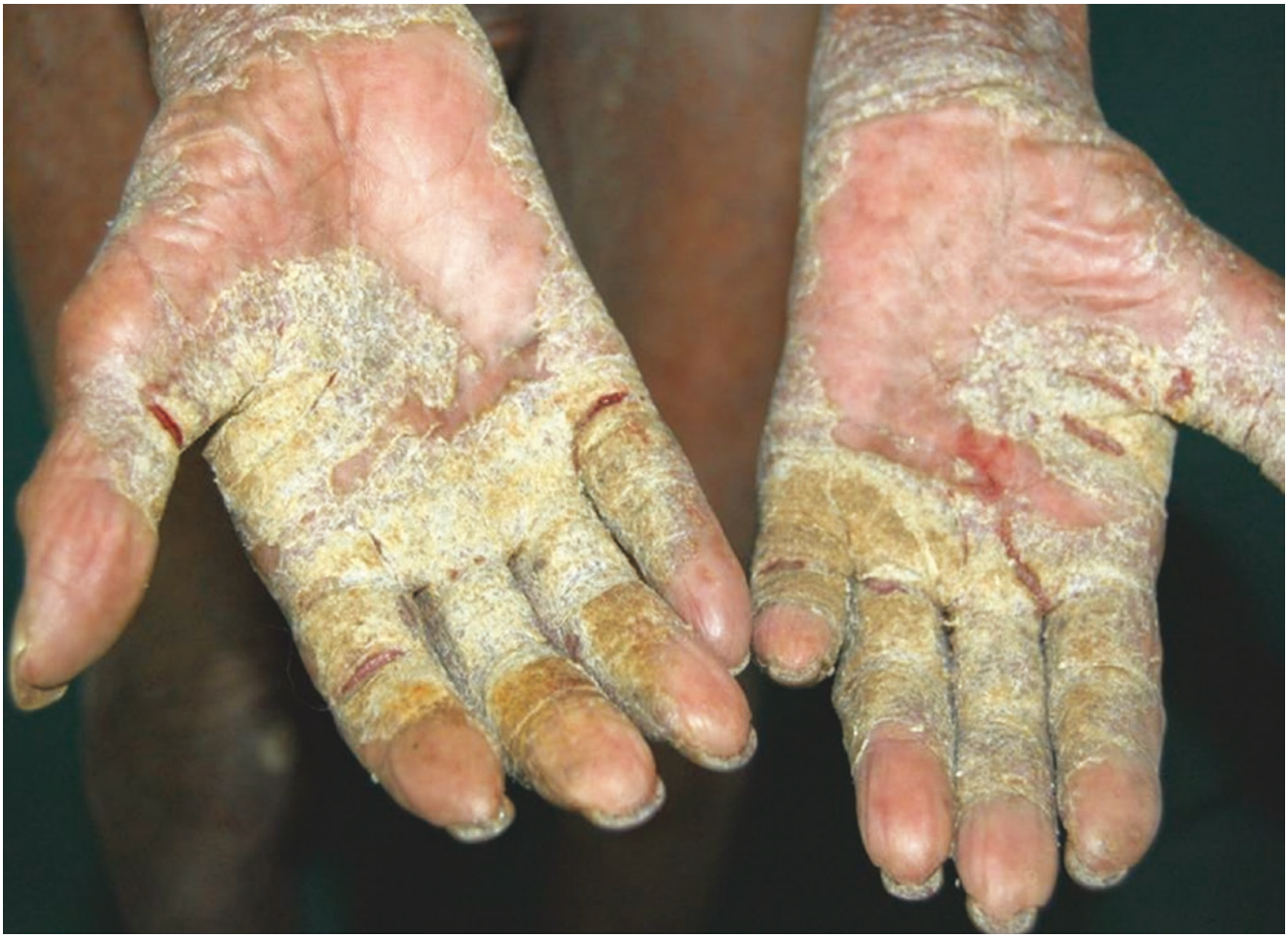

Fig. 16.5 *Norwegian scabies* presenting hyperkeratotic plaques in both hands of an elder immunocompetent patient

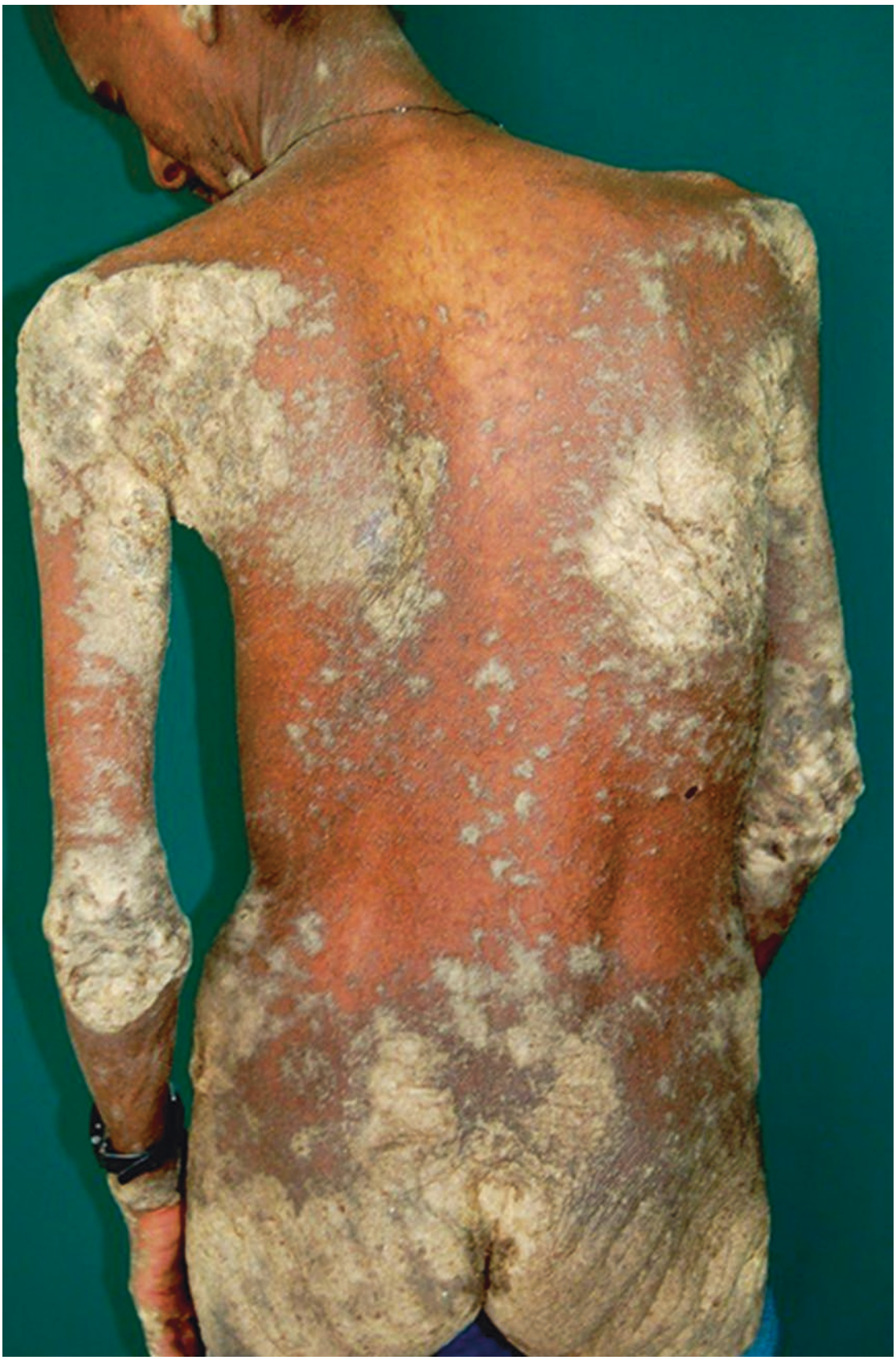

Fig. 16.6 Severe generalized *scabies of the Norwegian type* in an immunocompromised patient

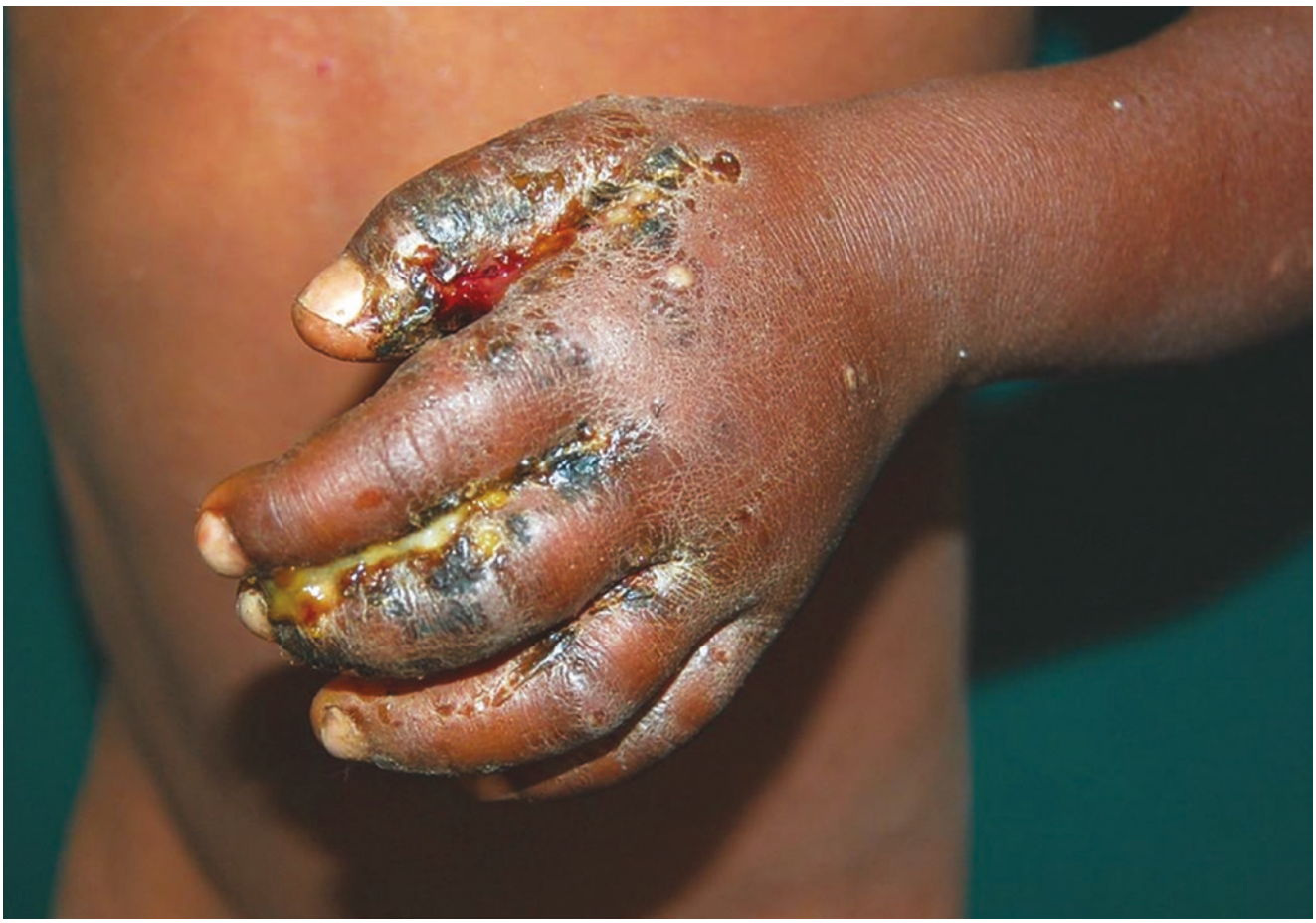

Fig. 16.7 *Scabies*. Severe super infection of the hand in a child. The intense itch often leads to super infections with S. aureus or β-hemolytic streptococci, with the risk of post-streptococcal glomerulonephritis, particularly in children

16.1.2 Treatment

Primary treatment is the administration of topical or oral scabicides [7]. Permethrin, a synthetic pyrethroid, is the first-line topical drug; older children and adults should apply 5% cream (60 g) to the entire body from the neck down, wash it off after 8–14 h, and repeat the procedure after 7 days. Periorbital and perioral regions should be avoided; special attention should be given to intertriginous areas and the umbilical area, fingers, and toes. Alternative medications are emulsions of 10–25% benzyl benzoate or crotamiton (5% gel, 10% lotion, cream, or ointment) to be applied on 3–5 consecutive days. Topical application of precipitated sulfur 6–10% in petrolatum over 24 h for 3 consecutive days is safe and effective for infants <2 months of age.

Ivermectin in a single oral dose 200 μg/kg bw (to be repeated after 7–10 days) is indicated for patients who do not respond or are unable to adhere to topical regimens and in those with widespread crusted scabies. The drug has been successfully used in epidemics in nursing homes, etc. Close contacts should be treated and personal items such as towels, clothing, bedding, etc. should be washed in hot water and dried in a hot drier or isolated in a closed plastic bag for at least 3 days.

Pruritus can be managed with topical corticosteroids and/or oral antihistamines (e.g., hydroxyzine up to 25 mg orally 4×/day). Caution is required when given to elderly patients with hepatic, renal, or cardiac disorders, not recommended for pregnant or lactating women and children <5 years old or with <15 kg body weight.

Secondary infection should be considered in patients with weeping, yellow-crusted lesions and treated with systemic or topical antibiotics against *Staphylococcus aureus* and *Streptococcus pyogenes*. Symptoms and lesions take up to 3 weeks to resolve despite killing of the mites; treatment failures due to resistance, poor penetration, reinfection, or nodular scabies are difficult to recognize. Skin scrapings can be done periodically to check for persistent mites.

16.2 Pediculosis

Pediculosis is worldwide a most common ectoparasitic infestation affecting hundreds of millions of people [8], mainly school children. In the USA, 6–12 million children are afflicted annually [9]. Independent of the country and culture, girls are usually more frequently infested than boys [10]. Although there is no clear association between head lice and poor hygiene or low socioeconomic status [11], crowded urban slums, migrants, refugees, and similar communities are preferred. In a study in Israel, 65% of all Ethiopian migrants were infested with head lice [12]. During the civil wars in Burundi, Rwanda, and Zaire in the 1990s, the prevalence of lice infestations reached 90–100% [13]. The prevalence of pediculosis capitis ranges from 1 to 20% in European countries, occurring in small epidemics mostly in play groups, kindergartens, and schools [10]. To some degree, there has been an increase in the incidence of pediculosis during the last decades, also due to treatment-resistant lice and treatment failures. Pediculosis often results in substantial personal embarrassment and social distress.

The scalp, body, pubis, and eyelashes are infested by three species, the head louse (*Pediculus humanus capitis*), the body louse (*Pediculus humanus corporis*), and pubic louse (*Pthirus pubis*) [10], all of them causing inflammation, itching, and skin discoloration. Head and body lice are transmitted by close contacts mostly under cramped, crowded conditions and pubic lice via sexual contact. The three species differ substantially in morphology and clinical features. Head and pubic lice live directly on the host while body lice in garments. The body louse may also be vector of infectious agents and cause relapsing fever and typhus [14]. Pediculosis corporis differs from scabies by the absence of skin burrows, and the hands and feet are not involved.

16.2.1 Clinical Picture

Louse infestation typically affects the hair of the scalp although it may involve any other hair-bearing areas, causing severe discomfort and pruritus (Fig. 16.8). Physical examination often reveals mild excoriations. Secondary bacterial infection

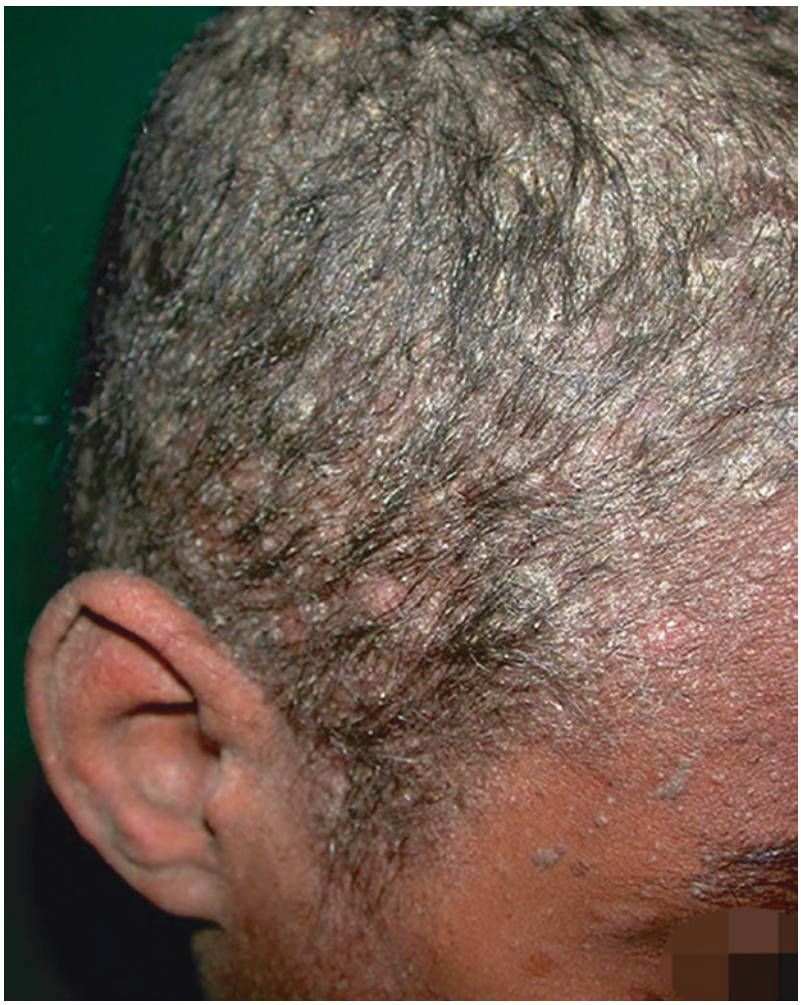

Fig. 16.8 *Pediculosis capitis* with scaly excoriations in an immunocompromised child

may cause folliculitis and regional lymphadenopathy; impetigo and furunculosis are occasionally detected and constitutional symptoms may occur, especially in infants and young children. Infestation of the eyelashes manifests as eye itching, burning, and local irritation. Lice are found usually at the back of the head or behind the ears by combing the wet scalp hair with a fine-tooth comb; in a case, usually ≤20 lice are present; nits are commonly fixed on the basis of the hair shafts. Each adult female louse lays three to five eggs/day; nits typically vastly outnumber the lice. Diagnosis of body lice is by demonstration of lice and nits in clothing, especially at the seams.

16.2.2 Treatment

Secondary bacterial infections should be treated first before eliminating the lice. Wet-crusted lesions are treated by wet compresses with potassium permanganate 1:9000 2×/day, followed by topical mupirocin or other antibacterial preparation.

Management of pediculosis often requires rotation of pediculicides to avoid resistance. Gamma-benzene hexachloride is rubbed to the scalp at night and then shampooed next night, with permethrin 1% creams/ointments or malathion 0.5% solution as alternatives, following their particular instructions. A second course may be required after 7–14 days, if lice are still seen. After applying the topical treatment, nits have to be removed by using a fine-tooth comb on wet hair, in order to prevent reinfestation; shaving of the area may facilitate the efficacy. Most pediculicides also kill nits. Dead nits remain after successful treatment; however, they do not signify active infestation and do not have to be removed; they grow away from the scalp with time. Absence of nits less than 7 mm from the scalp rules out current active infection. An alternate schedule on days 0, 7, and 14 has been proposed for nonovicidal pediculicides. New topical compounds such as benzyl alcohol 5% (nonovicidal) and spinosad 0.9% (ovicidal) were recently introduced and were found effective in cases with resistant lice [15].

A single oral dose of ivermectin 200 μg/kg, repeated in 10 days, has been shown to be effective against head pediculosis; however, ivermectin should not be used for young children with <15 kg body weight. The drug is also available as 0.5–1% topical preparations.

Hot air has been shown to kill >88% of the nits but is variably effective in killing hatched lice. Thirty minutes of hot air, slightly cooler than a blow drier, may be an effective adjunctive measure to treat head lice.

Controversy surrounds the need to clean the personal items of the afflicted persons and the need to exclude children with head lice or nits from school; there are no conclusive data supporting either approach. However, some experts recommend replacement of personal items or thorough cleaning, followed by drying at 55 °C for 30 min. Items that cannot be washed may be placed in airtight plastic bags for 2 weeks to kill the lice, which live only about 10 days. Primary treatment of body lice is thorough cleaning (e.g., cleaning, followed by drying at 65 °C) or replacement of clothing and bedding. Oral antihistamines may be required to alleviate itching.

16.3 Cutaneous Larva Migrans

Cutaneous larva migrans, often called *creeping eruption*, is one of the most common diseases transmitted to travelers, accounting for about 10% of all skin diseases seen when returning from hot climate zones [16]. The disease occurs worldwide, most often in tropical environments. It is usually acquired on sandy soils in Asia, Africa, South or Central America, and the Caribbean [17], caused by the animal hookworm *Ancylostoma* spp.; however, other species such as *Gnathostoma* spp. or even non-hookworm species such as *Dirofilaria conjunctivae*, *Capillaria*, and *Strongyloides* may also be the causative agent [5]. The eggs of the worms in dog or cat feces develop into infective larvae when left in warm moist ground or sand and penetrate into the unprotected skin, when it directly contacts contaminated soil or sand. As a rule, the feet, legs, buttocks, or the back are affected, while the larvae remain confined to the upper dermis.

16.3.1 Clinical Picture

Cutaneous larva migrans causes intense pruritus; classical signs are papules at the site of entry, followed by a growing linear or serpiginous, subcutaneous trail of inflammation that can reach 15–20 cm in length, justifying the designation of a creeping eruption. Most common localization is the dorsal side of the feet (Fig. 16.9), followed by the hands, arms, buttocks, and genitalia (Figs. 16.10 and 16.11). Patients may also develop papules and vesicles resembling folliculitis ("hookworm folliculitis"). The lesions are found occasionally in various other sites such as the scalp, oral mucosa, and the trunk [5]. As a rare complication, patchy pulmonary infiltrates with peripheral blood eosinophilia occur (self-limiting *Löffler syndrome*).

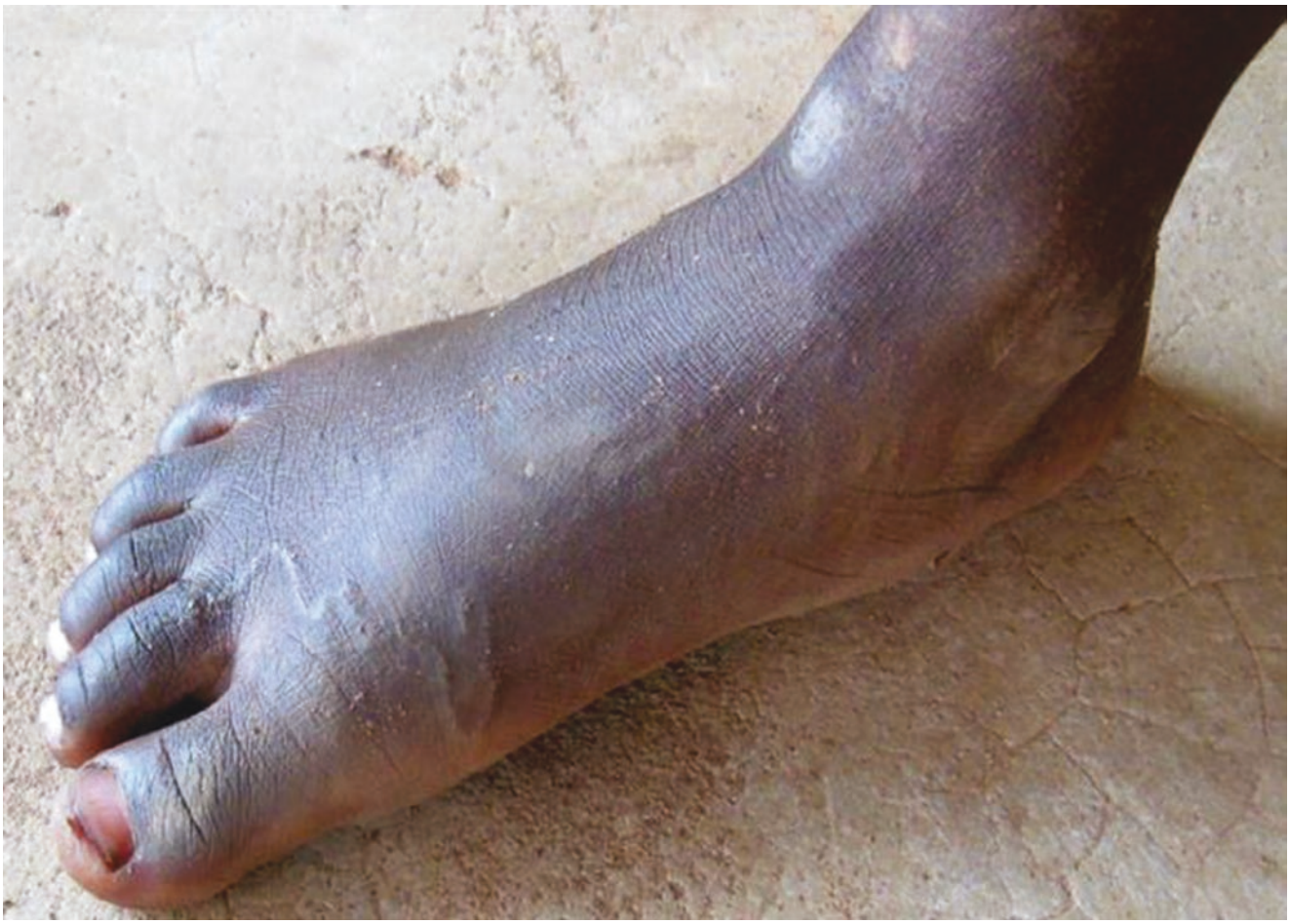

Fig. 16.9 *Cutaneous larva migrans*. Classical serpiginous, subcutaneous trail at the dorsal side of the foot

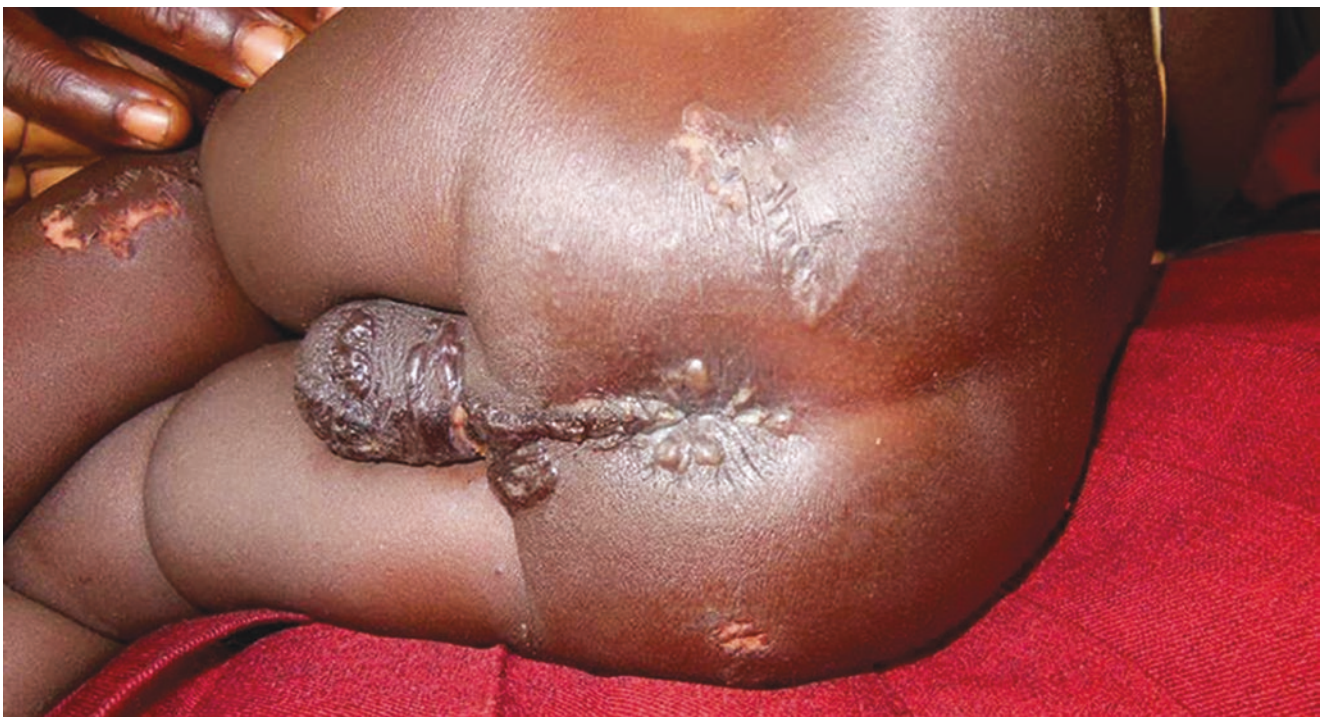

Fig. 16.10 *Cutaneous larva migrans*. Creeping eruption at the buttocks and the genitalia of a child

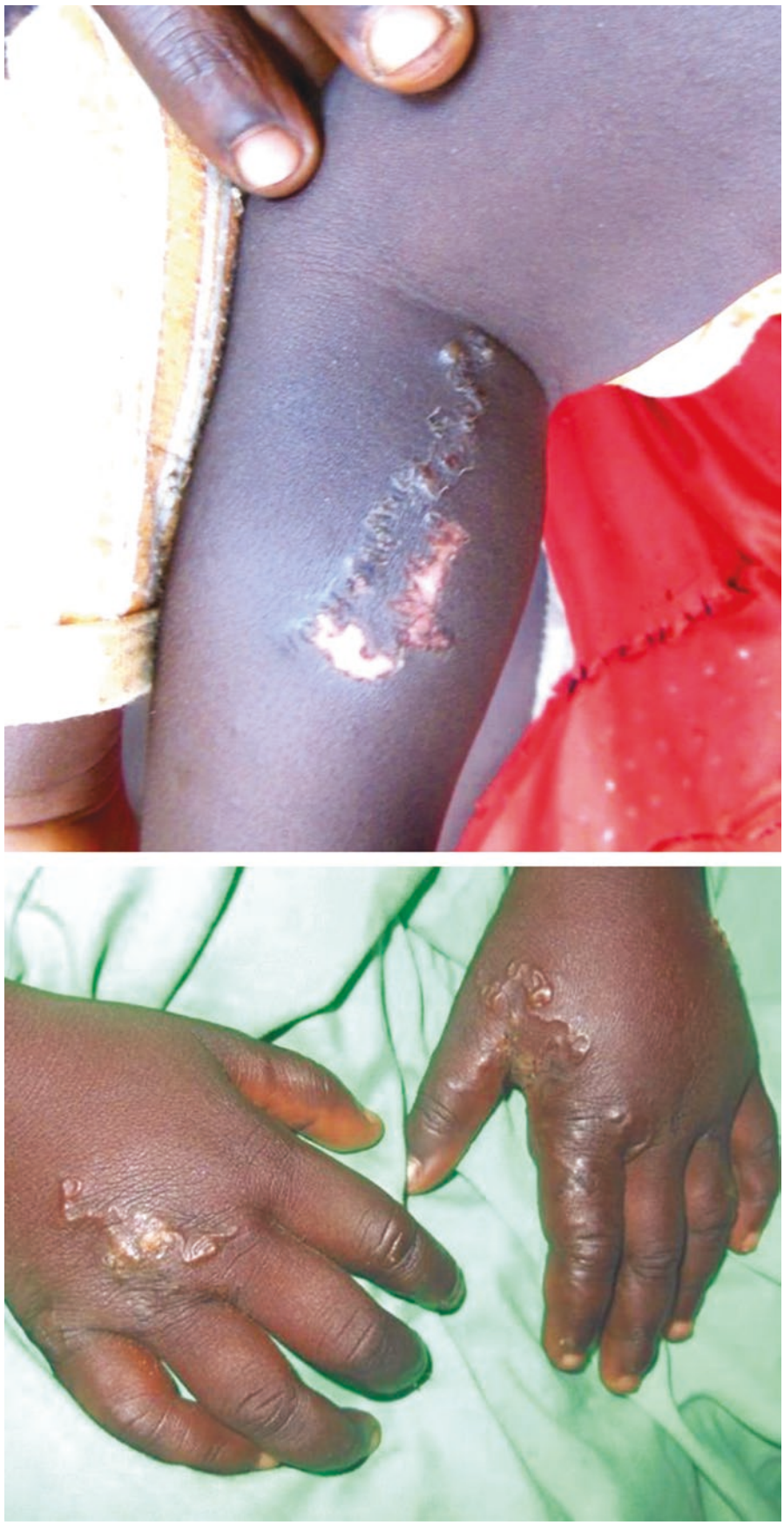

Fig. 16.11 *Cutaneous larva migrans*. Chronified, partly bullous multiple lesions in children

16.3.2 Treatment

Although the infestation resolves spontaneously after a few weeks, discomfort and the risk of secondary bacterial infection require treatment. Topical thiabendazole 15% liquid or cream (compounded) 2–3×/day for 5 days is effective. Oral thiabendazole is rarely used since it is not well tolerated. Oral albendazole (400 mg 1×/day for 3–7 days) and ivermectin 200 μg/kg bw 1×/day for 1–2 days are well tolerated and cure the infestation.

16.4 Larva Currens

Larva currens is a recurrent serpiginous maculopapular or urticarial rash on the buttocks, perineum, and thighs caused by *Strongyloides* spp. [5]. The condition is most often due to *Strongyloides stercoralis*, rarely to *Strongyloides fulleborni* [18]. *Strongyloidiasis* is known to occur in all continents but is most common in the tropics, subtropical regions, and hot climate zones. There are limited epidemiological data available on larva currens, but it is estimated that 30–100 million people are affected by strongyloidiasis worldwide [19].

16.4.1 Clinical Picture

The larvae in strongyloidiasis migrate more rapidly than in cutaneous larva migrans, reaching up to 10 cm/h. Unusual clinical manifestations of larva currens may include presentation as linear urticaria, as well as extensive purpura and skin invasion due to migrating larvae [5]. It may also appear as generalized petechial papules concentrated on the trunk or as progressive purpuric petechial eruptions with reticulated pattern concentrated over the abdomen.

The infestation may remain latent and manifest disseminated in immunocompromised individuals several years after exposure. The diagnosis is based on the clinical features and travel history. Filariform larvae may also be demonstrated in biopsies taken from the site of the rash and/or in dermal granulomas.

Serial stool examination remains as the gold standard for the diagnosis of strongyloidiasis. The *Baermann* concentration and *Harada-Mori* methods are also used as alternative diagnostic tools due to the low sensitivity of other techniques.

16.4.2 Treatment

Strongyloidiasis is treated with a single dose or two daily doses of ivermectin 200 μg/kg bw. As an alternative, oral albendazole may also be given 400 mg 2×/day for 7 days. If not treated, larva currens has been reported to persist for as long as 65 years due to autoinfection [5].

16.5 Cutaneous Enterobiasis (Oxyuriasis)

Cutaneous enterobiasis is common among children and institutionalized persons. It is estimated that more than 200 million people are infected worldwide [5].The disease is caused by *Enterobius vermicularis* transmitted through ingestion or inhalation of embryonated eggs and autoinfection through hand-to-mouth spread. The primary cutaneous manifestation of enterobiasis is nocturnal anal and perianal pruritus that begins 4–6 weeks after infection and may last for months. This is caused by the migration of female worms depositing eggs in the perianal area.

16.5.1 Clinical Picture

Scratching may cause skin irritation, and in more serious cases eczematous dermatitis, hemorrhage, or secondary bacterial infections occur. *Enterobius vermicularis*, if found in ectopic locations, can elicit severe granulomatous inflammation. Ectopic migration often results in infestation of the genital tract in women. This may cause granuloma of the uterus, ovary, fallopian tubes, and pelvic peritoneum [5]. In some cases, this may result in a polypoid mass in the anal region and pruritus vulvae with vaginal discharge. Involvement of the vulva in enterobiasis may be mistaken for carcinoma because of the presence of differentiated cell proliferation.

The disease is diagnosed using a Scotch test from the perianal area or cellulose-tape slide test, performed in the morning before defecation [19]; the presence of *Enterobius* can also be diagnosed by demonstrating eggs of the helminths in the feces (Fig. 16.12), perianal scrapings, or swabs from under the fingernails or by clinically detecting adult worms around the anus, usually at night.

16.5.2 Treatment

Albendazole is the treatment of choice, although mebendazole and pyrantel pamoate are also effective. Considering its familial distribution, the entire family should be treated to prevent reinfection [5].

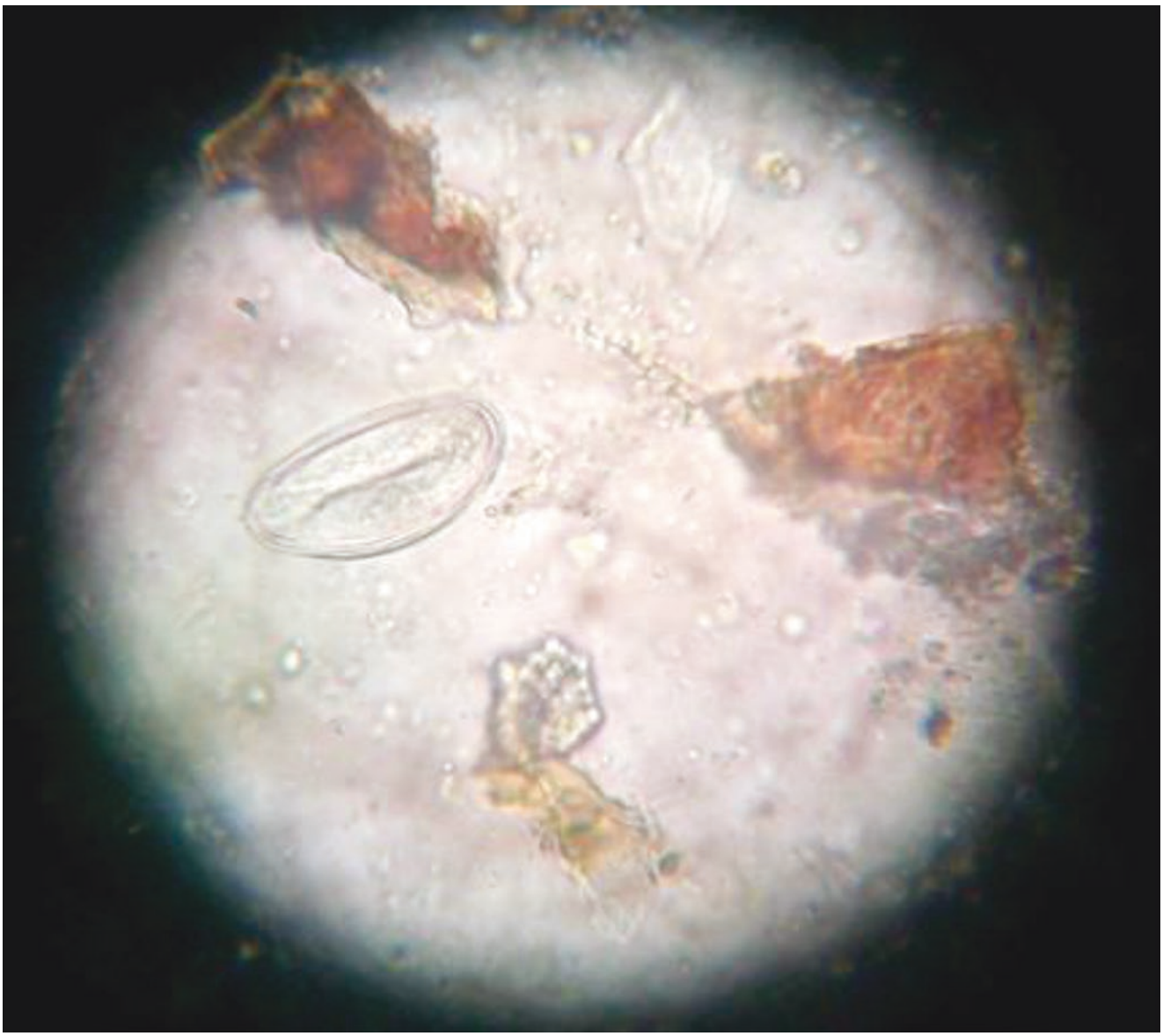

Fig. 16.12 *Enterobiasis*. Microscopical detection of *Enterobius vermicularis* eggs in the feces of a patient

16.6 Cutaneous Cysticercosis

Cases with cutaneous involvement have been reported in India, Africa, Mexico, and South America [20]. Cysticercosis, caused by the larvae of *Taenia solium*, occurs when a person ingests the parasite's eggs. The larvae lodge in the tissues, including the muscle and brain, and form spherical milky white cysts or cysticerci [19]. Approximately half of all cases present cutaneous manifestations, detectable as well-defined anechoic or hypoechoic lesions with or without calcification. The number of lesions may vary and may be too many to count. Cysticercosis also has the uncommon etiology of subcutaneous swellings in children [21]. Although less common, cutaneous cysticercosis may also result in neurocysticercosis. Diagnosis of the disease requires imaging and serological examinations. The history of travel and food intake should be also considered.

Treatment of cutaneous cysticercosis includes the use of anthelminthics such as albendazole or praziquantel together with surgery. There is, however, no generally accepted treatment regimen for the condition [21].

16.7 Cutaneous Amoebiasis

Cutaneous amoebiasis is caused by infection of wounds with *Entamoeba histolytica* trophozoites, a protozoan parasite found worldwide. It is transmitted via direct or indirect fecal-oral route [22]. Cutaneous amoebiasis is a rare skin manifestation of the infestation; amoebiasis can destroy various

human tissues, such as the intestinal mucosa, the liver, and to a lesser extent the brain and skin.

The disease initially presents as a deep-seated, well-defined, indurated, painful, progressively enlarging plaque with overlying ulcers and sinuses discharging pus and blood. Amoebiasis may also manifest as skin abscess, which may start as erythematous nodules and erythema nodosum [23].

Clinical suspicion and a simple wet drop examination may be used to diagnose the lesion [24]. Sensitivity is increased when tissues or smears are prepared from the edges of the ulcer. Erythrophagocytosis is a sign of the pathogenicity of the disease. For treatment oral metronidazole is the drug of choice [19].

Conclusions

A series of parasitic infections and infestations are in rising, both in developing and developed countries. The causative agents are transferred across the continents with increased globalization and traveling. Any success to control these entities, first aiming a possible elimination of scabies, will depend on developing clinical and public large-scale health programs in resource-poor countries.

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