Dermatologic Manifestations of HIV in Africa

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Dermatologic disease is common in HIV-infected individuals, and clinicians caring for patients with HIV infection or AIDS in Africa are routinely confronted with skin problems in their patients. Scarce access to dermatologic specialty care and limited educational resources describing the unique clinical characteristics of HIV-related skin disease can make diagnosing and treating skin diseases a challenge in Africa. This article describes common HIV-related dermatologic conditions in Africa and their differential diagnoses and includes treatment strategies that are likely to be available locally. It is not meant to be comprehensive but rather to serve as a practical resource to aid practitioners by providing images of common conditions and describing distinctive clinical presentations of common conditions.

The growing emphasis on global health and international volunteerism is increasing the demand for clinical education applicable across cultures and borders. The majority of HIV-infected individuals worldwide inhabit developing countries, yet medical research and literature regarding HIV- and AIDS-associated dermatologic disease have focused overwhelmingly on patterns of skin disease observed in white patients in resource-rich environments. Differences in skin pigmentation, climate, hygiene, and other genetic, environmental, demographic, and behavioral variables contribute to unique clinical presentations and epidemiologic patterns of HIV-associated skin disease in Africa compared with North America and Europe. Examples of HIV-related skin diseases with divergent epidemiologic characteristics include the papular pruritic eruption (PPE) associated with HIV, Kaposi sarcoma (KS), cryptococcosis, and photodermatitis. Additionally, mutually common conditions such as seborrhea may have distinctive clinical presentations.

This article may be used as a practical guide to dermatologic disease in HIV-infected individuals in Africa, focusing on common diseases, as well as their differential diagnoses and treatments. It is not meant to be exhaustive, and given the dearth of medical literature regarding HIV dermatology in Africa, the majority of the information is based on the experience and expertise of the authors. Readers are encouraged to consult the literature and experts in the field for clarification or more information. In addition, local health regulations, drug approvals, and availabilities of specific treatments and procedures mentioned below vary considerably by region and are continuously evolving. Practitioners would benefit from consultation with others experienced with the local health care delivery system in resource-limited settings.

Papular Pruritic Eruption

PPE is a very common HIV-related skin disorder in tropical environments, including Africa. The underlying etiology probably reflects a hypersensitivity to insect bites, hence the much higher incidence in tropical than temperate climates. PPE presents as extremely pruritic 0.2- to 1-cm papules that are darker than the patient’s uninvolved skin (Figure 1). They may be excoriated from scratching and thickened or shiny from rubbing. The papules typically are numerous and predominate on the extremities, although the trunk may also be heavily involved. The diagnosis is made clinically and can be confirmed by skin biopsy when available. Immune reconstitution with antiretroviral therapy is the treatment of choice, but improvement typically takes at least 16 weeks; pruritus sometimes improves with use of potent topical steroids or topical capsaicin.

Differential Diagnoses to Consider

Staphylococcal folliculitis. Bacterial folliculitis is a relatively common condition that may resemble PPE. Pruritus is variable, and lesions are typically fewer in number, are follicularly based, and may tend to be more concentrated on the upper trunk, upper arms, upper legs, and buttocks; pustules may be present. Treatment options include topical antiseptics (ie, chlorhexidine) and topical or oral antistaphylococcal antibiotics.

Eosinophilic folliculitis. Less common in Africa, eosinophilic folliculitis is a poorly understood HIV-related der-
matosis that, like PPE, is extremely pruritic. It may be differentiated from PPE based on its distribution, favoring the face, neck, scalp, and upper trunk (and almost never occurring below the nipple line). The papules are typically more urticarial and less shiny and hypopigmented than in PPE (Figure 2). It often mimics acne clinically, but patients complain of severe itching, which is not a feature of acne. It may appear or worsen temporarily during immune reconstitution. Treatment of choice is antiretroviral therapy, but symptoms may be ameliorated with use of potent topical steroids or oral itraconazole 200 mg to 400 mg daily.

Scabies. Scabies infestation is very common and may be mistaken for PPE. Patients present with an intensely pruritic rash (Figure 3). The rash can appear papulonodular like PPE, purpuric, eczematous, or even “crusted” in advanced HIV disease. Crusted, or “Norwegian,” scabies manifests with a thick, powdery, grayish scale that is teeming with mites. Unlike PPE, scabies lesions tend to be clustered, sometimes with visible burrows, in the finger webs, around the waistline, and on the wrists and ankles. Clinicians should always examine the axillae, breasts, umbilicus, and penis in men and boys; involvement of these areas argues for a diagnosis of scabies over PPE or folliculitis. It is not unusual for the scratching to lead to bacterial superinfection. Commonly available treatment options include use of topical benzyl benzoate ester, 6% precipitated sulfur ointment, or oral ivermectin where available.

Prurigo nodularis. Relatively common, prurigo nodularis presents with intensely pruritic, thickened, hyperpigmented, excoriated nodules (Figure 4). The nodules are larger (> 1 cm) and typically fewer in number (from 10-100 lesions) than in PPE. Prurigo nodules often start on the extremities and are bilateral and symmetric. With continued pruritus, they can become more widespread and appear on the trunk. Areas where patients cannot scratch, such as the midback, are spared. Prurigo nodularis results from intense scratching and does not have a single underlying etiology but may be secondary to other HIV-related dermatoses (such as photodermatitis, eczema, or PPE), underlying hepatitis C virus infection, renal failure, or lymphoma. Treatment should be directed at the underlying etiology, but symptoms can be treated with occlusion to provide physical protection from scratching, oral antihistamines, potent topical steroids, and topical capsaicin.

Seborrheic Dermatitis

As in the West, seborrhea is a very common skin disorder associated with HIV infection in Africa. Seborrheic dermatitis presents as a mildly itchy to non-itchy, scaly rash (Figure 5). Classically, the scalp, auditory canals, postauricular skin, and hair-bearing areas of the face and body (eyebrows, alar creases, beard, central chest, and axillae) are affected with erythema and “greasy” scale. However, in the authors’ experience, seborrhea has a much more varied clinical presentation in Africa. It may spare the face altogether, affecting the scalp, ears, and skin folds such as the axillae, antecubital fossae, and inner thighs. It may also present as a rash with “powdery” scale and very little underlying erythema, favoring the scalp, ears, neck, shoulders, and back. It can occasionally present as erythroderma (full-body erythema and scale). There may be overlap with inverse psoriasis or eczema. Treatment depends on severity. Typically a combination of topical antifungal drugs directed at *Pityrosporum* yeast and low- to midpotency topical steroids for inflammation will lead to improvement.
Photodermatitis. Counterintuitively, dermatitis caused by sun exposure is more frequently observed in persons with darker skin types and is very common in HIV-infected persons in Africa. It can sometimes be quite difficult to differentiate from seborrhea. Photodermatitis presents as an itchy, scaly rash affecting the sun-exposed regions of the skin (the face, neck, “v” of the chest, dorsal arms, and sometimes lower legs and dorsal feet), sparing skin that is protected from the sun anatomically (eg, under the chin) or by clothing (Figure 6). This distribution is often clinically apparent when the patient’s shirt is removed. HIV infection itself is photosensitizing, and many HIV-infected patients are taking photosensitizing drugs such as sulfonamides. Treatment includes immune restoration, sun-protective clothing such as hats and long-sleeved shirts, and potent topical steroids. Many of these patients earn a living working outside, and sunscreens are not widely available, making avoidance of sun exposure especially difficult. The authors do not recommend stopping sulfonamide prophylaxis because of photodermatitis; rather, these patients should be immune reconstituted to a level at which prophylaxis is no longer indicated.

Eczema. Advanced HIV disease causes dry skin, which can lead to eczema. Eczema is always pruritic and may be acute and weeping, or chronically dry and scaly (Figure 7). A background of xerosis (dry skin) is often present. Typically affected areas in adults include the eyelids, neck, flanks, hands, antecubital and popliteal fossae, and lower legs. Moister areas, such as the axillary skin, are typically spared. Treatment focuses on use of topical steroids and emollients and on avoidance of desiccating agents such as soaps. Emollients (such as petrolatum) should be applied immediately after bathing, while the skin is still moist.

Psoriasis. Psoriasis is relatively common in the HIV-infected population in Africa and may present with typical sharply demarcated, round, thick, scaly papules and plaques favoring the extensor extremities (Figure 8A). Atypical presentations, such as inverse psoriasis affecting the intertriginous areas and erythroderma, are common. There may be substantial overlap with seborrhea, with disease affecting the scalp, axillae, and inner thighs (Figure 8B). Destructive arthritis may be a feature. Most psoriasis will improve with antiretroviral therapy. Additional treatment is usually limited to use of topical steroids and short-contact anthralin therapy. Systemic agents are not routinely available or affordable, though this may vary by region. Psoriasis that has stabilized with antiretroviral therapy but suddenly flares may indicate a concomitant dermatologic condition such as scabies or staphylococcal infection, or it may be a marker of failure of current antiretroviral therapy. When pso-
If psoriasis is suspected, other differential diagnoses to consider include reactive arthritis (Reiter syndrome) and secondary syphilis.

**Drug Eruptions**

With the roll-out of antiretroviral therapy in Africa, associated drug eruptions occur commonly. Two types of drug eruptions that deserve special mention are Stevens-Johnson syndrome/toxic epidermal necrolysis (SJ/S/TEN) and single or multiple fixed-drug eruptions (Figure 9).

In the case of simple drug eruptions that are not life-threatening or incapacitating to the patient, clinicians may elect to “treat through” the symptoms with topical steroids and antihistamines. In this situation, patients should be monitored closely for the development of blisters, mucous membrane involvement, or systemic symptoms.

The overwhelming majority of serious drug eruptions in East Africa are caused by sulfonamides or nevirapine (Figure 10). Women starting nevirapine treatment at CD4+ counts above 250 cells/µL are particularly at risk of severe hypersensitivity reactions.\(^3\) SJS/TEN is often easily recognized, as most patients present for care late in the course of the disease, with erosion of mucous membranes (particularly the lower lip) and skin. Systemic steroid use is controversial; the authors do not recommend treatment with oral steroids unless used within the first 24 hours of symptom onset. Treatment is discontinuation of the offending drug as well as all other nonessential oral medications, and supportive care.

Fixed-drug eruptions typically present as strikingly round and sharply demarcated, intensely hyperpigmented patches (Figure 11). They may be single or multiple and located anywhere on the body, but the lips and genitals are frequent sites of involvement. The eruption may occur in the same place with each exposure or affect a new area of skin. In the authors’ experience, the overwhelmingly common culprit is an antibiotic, most often a sulfonamide, although a host of other drugs may cause this eruption. In regions where antibiotics are available over the counter, fixed-drug eruptions occur frequently.

**Kaposi Sarcoma**

Because of the endemic nature of human herpesvirus 8 in the region, KS is very common in HIV-infected persons in Africa. The demographics of KS in Africa are very different from those in the West, with men, women, and children all commonly affected (as opposed to western countries, where HIV-related KS is primarily a disease of men who have sex with men). KS classically presents as asymptomatic red-
dish-purple to brown papules, plaques, or tumors favoring the head and neck (particularly the nasal tip, the palate, and around the neck), upper chest, genitals, inner thighs, lower legs, and soles of the feet (Figure 12).

Unfortunately, KS often presents in advanced stages in Africa, with accompanying lymphedema, aerodigestive tract involvement, or both. The cornerstone of treatment for KS currently remains early detection and antiretroviral therapy initiation, as liposomal doxorubicin/danorubicin chemotherapy is rarely available in Africa, and medical practitioners with experience and infrastructure to safely administer chemotherapy are few. The prognosis for individuals presenting with advanced disease is poor. Skin biopsy is necessary to differentiate KS from clinically similar HIV-related vascular proliferations such as bacillary angiomatosis (BA). Immune reconstitution may initially cause flaring of KS lesions, which can be life-threatening. Patients should be monitored closely during the initial phase of immune restoration.

**Differential Diagnoses to Consider**

**Bacillary angiomatosis.** Although only sparsely reported in Africa, BA is probably more common than the literature reflects because of a lack of widely available skin biopsy and histopathology services. Like KS, it most often presents as solitary or multiple asymptomatic red-purple bumps (Figure 13). BA may also affect bone and viscera and can be fatal if untreated. Diagnosis is confirmed by a silver stain of a skin biopsy sample that reflects the presence of the causative organism, *Bartonella henselae* or *B. quintana*. Treatment consists of at least 6 weeks of oral erythromycin or doxycycline. For visceral involvement, 3 months of antibiotic treatment should be considered.

**Lymphoma.** Although non-Hodgkin lymphoma presents in the skin relatively rarely, cutaneous metastases can present as red to purple papules or plaques in the skin, often with a more translucent or “jellylike” appearance than KS (Figure 14). The diagnosis can be made by skin biopsy.

**Others.** Other disorders that can mimic KS in dark-skinned patients include pyogenic granuloma, warts, scars, postinflammatory hyperpigmentation, lichen planus (Figure 15), and inflammatory tinea faciei or tinea corporis.

This underscores the importance of skin biopsy in confirming the diagnosis of KS, especially before administering chemotherapy.

**Molluscum Contagiosum**

Molluscum contagiosum (MC) is very common in HIV-infected persons, par-

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**Figure 13.** Two examples of bacillary angiomatosis. Vascular papules and nodules are difficult to differentiate clinically from Kaposi sarcoma.

**Figure 14.** B-cell lymphoma. This individual exhibits typical lesions of nodular Kaposi sarcoma on the medial thigh, with a more translucent red-purple plaque overlying an enlarged inguinal lymph node representing lymphoma cutis.

**Figure 15.** Lichen planus. Purple papules with overlying fine white scale. Note the linear arrangement of papules where the skin was scratched; this is known as the Koebner phenomenon and does not occur in Kaposi sarcoma.

**Figure 16.** A single molluscum and hundreds of flat warts in an HIV-infected child. Note the umbilicated center of molluscum. Flat warts may also exhibit the Koebner phenomenon, appearing in strikingly linear arrangements.
particularly pediatric patients, in Africa (Figure 16). MC is caused by a pox-virus. It presents with dome-shaped, umbilicated papules, typically 3 mm to 8 mm, although giant lesions can occur in advanced HIV disease. Antiretroviral therapy is the mainstay of treatment, but curettage or silver nitrate treatment may also be used.

**Differential Diagnosis to Consider**

**Cryptococcosis.** Disseminated cryptococcal disease can present in the skin, often preceding or simultaneous with the onset of meningitis symptoms. Clinically, the lesions resemble MC, although the central umbilication often has a hemorrhagic crust. The skin lesions of cryptococcosis are often eruptive, as opposed to the slower, insidious onset of MC (Figure 17). Clinicians should maintain a high level of suspicion for cutaneous cryptococcosis in Africa, where cryptococcal disease is very prevalent, particularly if the lesions appear over a short time course.

**Warts**

Viral warts, both genital and nongenital, are very common in Africa (Figure 16). Particularly in pediatric patients, hundreds of flat warts are a frequent, and stigmatizing, finding that may or may not improve with antiretroviral therapy. Cryotherapy, the standard wart treatment in developed countries, is generally not available. Large genital lesions may be treated with 25% podophyllin, which is painted on the warts and washed off in 4 hours to 6 hours, and nongenital warts may be treated with salicylic acid preparations. Genital warts should be monitored for rapidly growing firm nodules or ulcers that may indicate human papillomavirus-induced squamous cell carcinoma.

**Herpes Simplex Virus and Varicella-Zoster Virus Infections**

Infections with herpes simplex virus and varicella-zoster virus are quite common in Africa and most often present in the typical fashion. Large, chronic ulcerations of the face, genitals, or buttocks due to herpes simplex virus infection are frequent in patients with advanced HIV disease (Figure 18). A “scalloped” border to a chronic ulceration can be a clue to the diagnosis. Oral acyclovir treatment is generally available, whereas intravenous acyclovir can be difficult to obtain, and alternative antiviral drugs are not widely available.

**Tinea**

Tinea is a universally common skin problem in HIV-infected patients. Typical tinea has an inflammatory, scaly border; in darkly pigmented patients, the area of central “clearing” is usually hyperpigmented (Figure 20). Common locations include the hands, feet, and lower back or buttocks. Special considerations in Africa include tinea incognita.
to, a noninflammatory presentation of tinea caused by use of widely available over-the-counter skin products that contain potent steroids. Topical antifungal drugs can be used for localized disease, whereas oral antifungal drugs such as griseofulvin or ketoconazole may be necessary for widespread infection or involvement of hair follicles (majocchi granuloma), indicated by follicular papules or pustules. Tinea capitis requires treatment with oral antifungal drugs for a minimum of 6 weeks.

Other Considerations

In addition to familiarity with the specific entities described above, the clinician with less experience with dermatologic disease in darkly pigmented persons will benefit from an understanding of a few general principles. First, erythema may be difficult to appreciate and may appear gray, violet, or simply hyperpigmented. Additionally, darkly pigmented individuals commonly experience a phenomenon called postinflammatory pigment alteration (PIPA) in response to underlying inflammation, regardless of the cause. Hyperpigmentation, hypopigmentation, or both may occur. There is no treatment for PIPA, aside from treating the underlying condition and allowing the pigmentedary changes to resolve. Also, bacterial superinfection with Staphylococcus or Streptococcus species, known as “impetiginization,” is extraordinarily common with all pruritic skin diseases in Africa. This presents as golden or honey-colored crusting and often superficial erosion of skin. Although primary impetigo can occur, patients should be examined for an underlying pruritic disease such as eczema, scabies, or insect bites.

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References