

# Evaluation and Treatment of Itching in HIV-Infected Patients

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## Abstract

Itching is a common complaint among patients infected with HIV and may cause significant morbidity and embarrassment. Although idiopathic HIV-pruritus has been described, it is probably less common than was previously thought. In most patients, a careful history and physical examination will show that a dermatosis accounts for their pruritus. Dry skin, seborrheic dermatitis, eczema, psoriasis, pruritic papular eruption, staphylococcal folliculitis and prurigo nodularis are frequently encountered in these patients. These common dermatoses, drug eruptions, several rarer conditions and systemic causes of itching should be excluded before diagnosing idiopathic HIV-pruritus. Treatment should be directed to the underlying skin problem and may be supplemented with sedating antihistamines. Phototherapy is a safe and effective therapeutic modality for many pruritic dermatoses as well as for idiopathic pruritus.

**Key Words:** Itching, pruritus, HIV.

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ITCHING, A COMMON SYMPTOM in HIV-infected patients, can cause embarrassment, discomfort and sleep loss. It may be a presenting symptom of a number of HIV- or non-HIV-associated dermatoses or may occur without any visible eruption (1). Chronic itching and scratching often result in lichenification, excoriation, prurigo nodularis, pigmentary alteration and secondary infection.

Although the pathophysiology of itching is poorly understood, it is thought to result from local skin changes or, in some cases, from a central mechanism possibly related to circulating pruritogens. A number of chemical mediators, including histamine, serotonin, prostaglandins, cytokines, proteases, tachykinins (e.g., substance P), and opioid peptides are thought to be involved on a local level (2). HIV-infected patients frequently display xerosis, i.e., dry skin, which might have some physiochemical effect on "itch fibers." Recently, Schmelz et al. identified histamine-sensitive C-fibers, which have particularly thin axons but excessive terminal branching that may represent the afferent units mediating the itch sensation (3).

In patients infected with HIV, pruritus can develop as a consequence of routine "itchy" der-

matoses, some of which may be exacerbated or may occur more frequently in the setting of HIV infection. These dermatoses include papulosquamous disorders, skin infestations or infections, and drug reactions. Drug eruptions are particularly common because of abnormal immunoreactivity, altered drug metabolism and polypharmacy. Moreover, itching may be associated with systemic diseases, such as renal failure or liver disease, or it may be a manifestation of progressive immunodeficiency and dermatoses peculiar to HIV infection.

Evaluation of the HIV patient with pruritus should include a complete skin and physical exam, and laboratory tests as indicated by the history and clinical presentation. Moreover, the patient's CD4 count and viral level, as measured by polymerase chain reaction (PCR), may also be useful. Optimum anti-viral therapy should be instituted, since this not only slows the progression of HIV infection and increases life expectancy, but also has been reported to reverse certain HIV-related skin disorders (4).

## Xerosis

Generalized asteatosis, often referred to as "xerosis" or "dry skin," is a common finding in HIV-infected patients. It becomes more severe with disease progression as CD4 counts decline. Characteristically, the skin appears rough and lusterless, often with fine scale, best appreciated under tangential lighting. Xerosis is particularly

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prominent on the extremities and is exacerbated in winter months. Alterations in skin hydration and barrier function predispose to irritant dermatitis, pruritus, excoriation and secondary infection. Treatment includes avoidance of harsh soaps and hot water. Emollients should be applied in the first few minutes after bathing, while the skin is still moist. In general the greasier emollients are preferred, but these too may be unacceptable to many patients. Mild, oilated soaps or soap substitutes are recommended.

### Idiopathic Pruritus

Idiopathic pruritus similar to that found in Hodgkin's disease has been described in patients with advanced HIV infection (5, 6). Because such patients often have dry skin, a known cause of itching, some authors think idiopathic pruritus is uncommon. Before attributing itching to idiopathic HIV pruritus, a primary dermatological diagnosis should be sought. Subtle manifestations of scabies, xerosis, atopic dermatitis, and papular eruption of HIV should be eliminated, as should systemic disorders such as renal failure and liver disease, before this diagnosis can be made.

Oral antihistamines are of limited benefit. The authors sometimes use sedating antihistamines if the patient has trouble sleeping through the night. It is critical to be aware of drug interactions when using astemizole. Protease inhibitors and anti-fungals such as itraconazole, which are often used to treat HIV-infected patients, inhibit cytochrome P450 enzymes and can lead to fatal arrhythmias if used in conjunction with astemizole or cisapride (7). We have found doxepin, a tricyclic antidepressant with antihistaminic properties, to be particularly useful. It should be used with care in the presence of heart disease, and is contraindicated if there is coexistent narrow-angle glaucoma or prostatic hypertrophy.

Several studies have shown that ultraviolet B (UVB) phototherapy is helpful for the treatment of HIV-associated pruritus. The itching generally diminishes after 4–8 weeks of treatment. Although ultraviolet exposure can theoretically increase HIV replication, we have found no adverse effect of UVB phototherapy on HIV patients' plasma viral levels as measured by quantitative PCR (8). The overall experience of UVB phototherapy in HIV-positive patients suggests that this is a safe modality (9). Caution should be observed with patients presenting cutaneous evidence of Kaposi's sarcoma. Psoralens plus long-

wave ultraviolet light (PUVA) have also been reported to be beneficial in the treatment of idiopathic pruritus (10).

Pentoxifylline and indomethacin have recently been reported to be effective in relieving HIV-related pruritus, although this has not been confirmed in randomized, controlled studies (11, 12).

### Pruritic Papular Eruptions

Several chronically itchy papular and papulopustular eruptions, variously referred to as pruritic papular eruption (PPE) of HIV infection, "itchy red bump disease," eosinophilic pustular folliculitis, eosinophilic folliculitis (EF), papular urticaria, prurigo nodularis and insect bite reactions, have been reported in HIV-infected patients. PPE probably represents a heterogeneous spectrum of papular and papulopustular skin eruptions with similar or overlapping clinical and histological features and response to therapy (13). It has been reported in series from Africa, Haiti, Brazil and the United States (14–16). In the Haitian study (16), PPE was seen in 46% of patients with AIDS and was the presenting manifestation in 79%, often appearing months before the diagnosis of an AIDS-defining condition. PPE, and more specifically EF, is seen in the context of deterioration in the immunity of HIV patients. Patients consistently have CD4 counts below 200 (17, 18). In one study, 81.25% had CD4 counts less than 100, and 75% had CD4 counts below 50 (19). Some investigators have noted a decrease in EF prevalence with the advent of aggressive antiretroviral treatment, although the immune reconstitution accompanying such treatment can sometimes cause a flare-up of EF.

PPE presents as erythematous urticarial papules resembling arthropod bites (Fig. 1). There may be pustules or papules surmounted by pustules, but in our experience the lesions are mostly excoriated. Lesions are typically found on the extremities and trunk, with sparing of the palms, soles and digital web spaces (13). Patients frequently have prurigo nodules if the condition is longstanding. Lichenified patches and plaques on the arms and legs may be seen, probably representing an AIDS-associated, late-onset, atopic dermatitis.

Histopathologic exam in PPE reveals hyperkeratosis, acanthosis, focal dyskeratotic and necrotic cells in the epidermis, and dermal fibrosis with proliferation of factor XIIIa-positive dermal dendrocytes. The dermal infiltrate also includes lymphocytes, plasma cells, eosinophil and mast cells.



**Fig. 1.** Pruritic papular eruption: papule surmounted by pustule.

PPE follows a chronic waxing and waning course, and may have an associated peripheral eosinophilia and elevated levels of IgE. Sanchez et al. evaluated 41 patients with HIV-associated pruritic eruptions (20). They found that 80 percent had CD4 T-cell counts below 100 cell/mm<sup>3</sup>, 77% had elevated IgE levels, and 55% had elevated eosinophil counts. The pruritus is typically not relieved by antihistamines or topical treatment, but UVB phototherapy is usually effective. Ultra-potent topical corticosteroid ointments may be of some benefit.

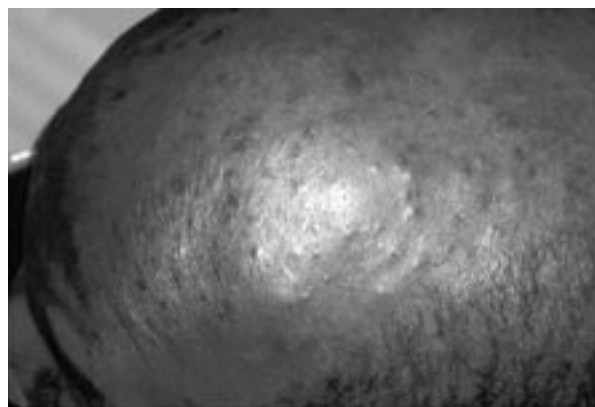
Eosinophilic folliculitis (EF) presents as erythematous urticarial papules, pustules, and/or papules with pinpoint vesicles or pustules on the face, neck, upper chest and back, characteristically above the nipple line (Fig. 2). The upper arms, eyelids, post-auricular areas and scalp are often involved (Figs. 3, 4) (21). More extensive involvement of the forearms, thighs and trunk below the nipples is less common. Pruritus is usually severe and unrelieved by antihistamines, and tends to be chronic with occasional periods



**Fig. 2.** Lesions of eosinophilic folliculitis on upper chest.



**Fig. 3.** Post-auricular lesions of eosinophilic folliculitis.



**Fig. 4.** Eosinophilic folliculitis: scalp involvement.

of remission. Laboratory examination may reveal elevated IgE levels and peripheral eosinophilia.

Histopathologic examination reveals follicular spongiosis and folliculocentric inflammatory infiltrates involving the outer root sheaths of hair follicles. The infiltrate is rich in eosinophils but also contains lymphocytes, histiocytes, neutrophils and mast cells (21, 22). Eosinophilic abscesses and flame figures may be seen. Magro and Crowson found significant coating of perifollicular mononuclear cells with IgE in two HIV-infected patients, a situation analogous to lesional skin of patients with atopic dermatitis (23). A simple method of diagnosing EF which correlates well with biopsy diagnosis is to prepare a smear from an intact pustule if one can be found. Staining with Wright's stain will reveal a predominance of eosinophils (18). A recent study demonstrated that sectioning biopsies horizontally rather than vertically may be helpful in diagnosing EF (24).

Also within the spectrum of PPE are papular eruptions that resemble "insect bite reactions" or

papular urticaria, an entity usually seen in immunocompetent children who have had flea or bed bug bites. Some authors feel that if enough sections are studied, eosinophilic folliculitis will be found. Studying 7 patients with such reactions, Penneys et al. demonstrated increased levels of circulating antibodies to antigens found in salivary glands removed from the salt marsh mosquito, *Aedes taeniorhynchus* (25). These authors ascribed the eruption to chronic recall reactions at previous mosquito bite sites.

There is an overlap not only in the clinical presentations of papular eruptions of HIV but also in different responses to therapy; however, there have been no randomized, controlled trials to date to validate the variety of reported treatments. Eosinophilic folliculitis frequently responds to UVB phototherapy and isotretinoin (8, 26, 27). There are reports that EF may also improve with cetirizine, itraconazole, metronidazole, ivermectin and long-term application of permethrin, the latter two implying a possible role for Demodex mites in the pathogenesis (28–32). In our experience, papular urticaria-like eruptions are often resistant to treatment and although systemic corticosteroids may be efficacious, their long-term use could be problematic in HIV-infected patients. Some investigators use periodic, tapering doses of steroids.

Prurigo nodularis, usually resistant to most therapeutic modalities, sometimes responds to phototherapy and intralesional steroid injections. There is a recent report that thalidomide may be useful, implying a possible immune or neurogenic pathogenesis for this condition (33). Some authors have suggested a mycobacterial causation of prurigo nodularis, but it is likely that the presence of mycobacterial DNA in these lesions, detected by PCR, is a result of secondary invasion of ambient microorganisms caused by scratching (34).

Papular mucinosis, an unusual, sometimes pruritic eruption caused by the deposition of mucin in the dermis, has been reported in patients with HIV infection (35). The eruption, sometimes referred to as lichen myxedematosus, is usually associated with an IgG paraproteinemia. A minority of cases reported in association with HIV infection have been pruritic. We have recently encountered a male patient with lichen myxedematosus with extremely itchy papules on the posterior aspect of the neck and the upper back (Fig. 5). He had no paraproteinemia. In fact, most of the cases reported in association with HIV infection have lacked this feature.

The pathophysiology of papular eruptions is not completely understood, but has been attrib-



**Fig. 5.** Papular mucinosis with cobblestone appearance on upper back.

uted in part to immune dysregulation with abnormal host responses to such infectious agents as staphylococci, saprophytes such as Demodex mites, or Pityrosporum yeasts (36). An immune reaction to antigens in the saliva of biting insects or an autoimmune reaction to a component of sebum has also been postulated (25, 37). There is evidence that a shift toward a Type-2 cytokine profile may be important in eosinophilic folliculitis (38). This is based on recent evidence that Type-2 responses may predominate in late-stage HIV disease, the known loss of delayed-type hypersensitivity (DTH) with AIDS, and the likelihood that IL5 from type-2 cytokine producing cells could attract eosinophils into the lesional skin.

The possibility of immune dysregulation in HIV-related papular eruptions is supported by data that many patients with late-stage disease have elevated eosinophil counts that in most cases correlated with skin diseases such as eosinophilic folliculitis, atopic dermatitis and prurigo nodularis (39). There is a rise in circulating levels of eosinophilic cationic protein (ECP) in both atopic and non-atopic HIV patients (40).

Moreover, there is evidence that pruritic papular eruptions occur in non-HIV-infected patients with immune dysfunction, suggesting that the disturbance of immune function is of paramount importance. PPE has been reported in the syndrome of idiopathic CD4+ lymphocytopenia, and eosinophilic folliculitis has been reported in a patient with Waldenström's macroglobulinemia and in patients immunosuppressed after high-dose chemotherapy and bone marrow engraftment for non-Hodgkin's lymphoma (41, 42). Recently, data was presented supporting a Type-2 cytokine shift in eosinophilic folliculitis and an important role for the chemokine eotaxin (43–46).

### Photodermatitis

Photosensitivity has been described in HIV-infected patients with advanced immunodeficiency, sometimes in the absence of other disease manifestations (47). It is thought to be related to a photosensitizing drug in most cases. Chronic lichenoid photoeruptions (Figs. 6, 7) with severe pruritus and violaceous plaques on the dorsa of the hands, forearms, face and neck were described by Berger and Dhar in patients with severe immune suppression (helper T-cell count under 50 cells per cubic milliliter) (48).

Afro-American patients were disproportionately affected; pigmentary alterations, especially hyperpigmentation, but also hypopigmentation, were prominent. Treatment includes avoidance of sun exposure, use of topical sunblocks that block UVA and UVB, and in some instances, photochemotherapy (psoralen plus UVA or PUVA) or etretinate (49).



**Fig. 6.** Lichenoid photodermatitis: hyperpigmented plaques with central depigmentation on sun-exposed skin of the hands.



**Fig. 7.** Lichenoid photodermatitis: hyperpigmentation on sun-exposed skin of posterior neck and ears associated with several lichenoid plaques.

### Papulosquamous Disorders Exacerbated by HIV

#### Psoriasis

Psoriasis occurs with approximately the same frequency (about 1%) in HIV-infected patients as in patients who are not infected (50). It is frequently itchy for both groups of patients. The condition may be exacerbated by underlying HIV infection, is more difficult to treat than non-HIV-related psoriasis, and may become worse as patients progress to AIDS (51, 52). A significant proportion of patients with HIV-associated psoriasis have a pattern of acral involvement, often with pustules and sometimes with severe, destructive nail changes (Fig. 8) (50). Psoriasis in HIV-infected patients may respond to the usual measures, such as topical corticosteroids, calcipotriol or tazarotene for localized disease and PUVA or UVB phototherapy for more extensive involvement. In addition, antiretroviral therapy is often associated with an improvement in psoriasis (52, 53). A recent survey found that phototherapy is widely used for HIV-infected patients: 80% received UVB, 9% received PUVA and the remaining 11% received a variety of combination and other therapies (54). A case has been made that PUVA may be preferable to UVB therapy because of its increased efficacy, especially with thick plaques and palmoplantar involvement, as is frequently encountered in the setting of HIV-associated psoriasis (55). As with UVB, PUVA may also theoretically activate HIV replication; however, pilot studies have found no evidence of increased HIV activity in HIV patients treated with PUVA (56, 57). Nevertheless, PUVA may not be preferable in HIV patients, due to the gastrointestinal side effects of psoralens and the



**Fig. 8.** Acral psoriasis, psoriatic arthritis and destructive nail changes.

potential for carcinogenesis, which may be exacerbated by the immunosuppressed status of these patients. Systemic retinoids such as etretinate or acitretin have been particularly useful for treatment of generalized psoriasis. Methotrexate has been reported to cause profound leukopenia and death in some psoriatic patients with HIV infection and is avoided by many practitioners (49). However, in a recent report, there was no clear deterioration with methotrexate use for AIDS-associated psoriatic arthritis (58). Because of the known interaction with sulfamethoxazole-trimethoprim, methotrexate should not be used in patients receiving this drug for prophylaxis against *Pneumocystis carinii*.

### Eczema/Atopic Dermatitis

Although there are no prospective studies of the effect of HIV infection on atopic dermatitis, eczematous dermatitis has been described in both pediatric and adult patients with HIV infection as well as HIV-infected hemophiliacs (59). A recent survey of atopic manifestations in HIV-infected patients found chronic pruritic rashes or eczema in 29% of patients (60). Family history of asthma or hay fever was significantly associated with chronic pruritic rashes and eczema. There is a report of a 40-year-old male AIDS patient who developed atopic dermatitis after treatment with AL721, a mixture of egg yolk lipids used in the past in the treatment of AIDS (61).

### Seborrheic Dermatitis

Seborrheic dermatitis may be a marker for early HIV disease (CD4 counts > 500). HIV-infected patients often have extensive involvement, with redness and scaling of the central face and scalp (Fig. 9). The pathophysiology is unclear but is thought possibly to be related to an inflammatory response to the yeast *Pityrosporum ovale*. Because tinea infections such as tinea faciei (Fig. 10) may occur in this patient population and mimic seborrheic dermatitis, a potassium hydroxide examination for hyphae should be performed if the diagnosis is in doubt (62). Treatment includes shampoos with coal tar, selenium sulfide, ketoconazole or pyrithione zinc and creams containing mild corticosteroids or ketoconazole.

### Skin Infections/Infestations

#### Fungal (Tinea, Candida)

Cutaneous fungal infection with dermatophytes (tinea infection) is common in patients



Fig. 9. HIV-associated seborrheic dermatitis with extensive scaling.



Fig. 10. Annular lesions of tinea faciei.

infected with HIV. One study found a 37.3% colonization rate among HIV-infected homosexual males compared to a 31.8% rate among non-HIV-infected homosexual male controls (63). As with psoriasis, the prevalence of tinea infection may not be greater in HIV-infected individuals; however, the infection may be exacerbated by immunodeficiency. We have encountered a number of HIV-infected patients with extensive tinea infections, including tinea capitis (Fig. 11A), faciei, corporis (Fig. 11B), cruris, pedis and manuum. In addition, tinea infections in this patient population may be more resistant to treatment. Treat-



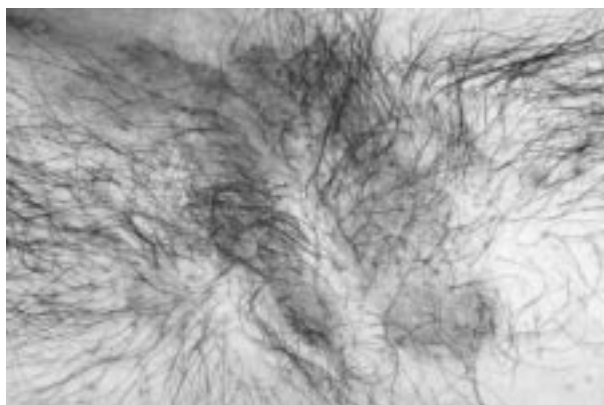
**Fig. 11. A.** Tinea capitis: “black dot ringworm” in HIV-infected adult patient. **B.** Tinea corporis with extensive scaling of the trunk. (A and B represent the same patient.)



ment includes topical antifungal agents, and in more severe cases systemic antifungal agents such as fluconazole, itraconazole or terbinafine. The absorption of itraconazole requires an acid pH in the stomach and may be impaired in some patients with HIV infection. Certain drugs such as didanosine (DDI) can interfere with the absorption of itraconazole (64).

### Bacterial Folliculitis

*Staphylococcus aureus* folliculitis is a common cutaneous infection in HIV-infected patients. Duvic described a pruritic intertriginous or axillary eruption in HIV-infected patients, with confluent red lesions and satellite pustulosis resembling cutaneous candidiasis (Fig. 12) (5). The high prevalence of staphylococcal skin infections in HIV-positive patients may be due in part to high staphylococcal nasal carriage rates in these patients, i.e., twice that of controls (65). Although carriage rates were



**Fig. 12.** Staphylococcal-infected dermatitis of the axilla.

increased over controls in hospitalized HIV-infected patients in one study, another study found no independent association between HIV status and staphylococcal nasal colonization in outpatient drug users (66). This study did find inhalational drug use to be independently associated with nasal carriage of staphylococci. Mupirocin ointment applied intranasally eradicated staphylococci for several weeks in HIV-infected patients but the effect waned over time, with persistently negative cultures of 63%, 45% and 29%, at 2, 6, and 10 weeks respectively (67).

A chronic recalcitrant “toxic-shock-like” disorder has been described in HIV-infected patients (68). Staphylococci may also superinfect molluscum contagiosum lesions and lead to symptoms of pruritus. Treatment of staphylococcal infections includes oral antibiotics such as dicloxacillin, chlorhexidine gluconate (Hibiclens) washes and topical mupirocin.

HIV-infected patients occasionally suffer infestations with scabies and pediculosis. Atypical presentations including crusted scabies (Fig. 13) have been reported in this population. Patients with crusted (Norwegian) sca-

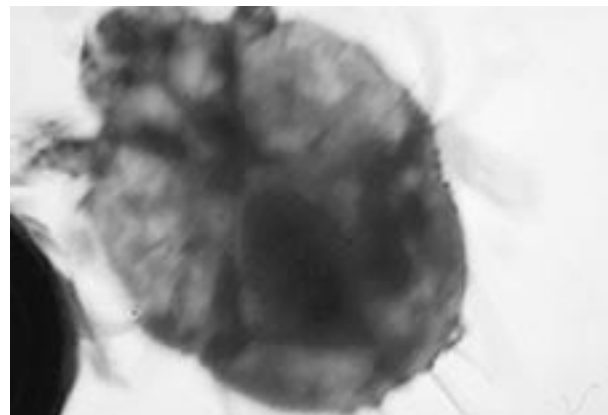


**Fig. 13.** Hyperkeratotic lesions of crusted scabies.



**Fig. 15.** Flaking on exam table from patient with crusted scabies and psoriasis.

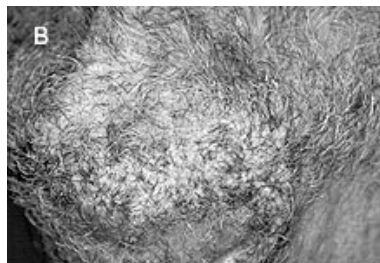
bies often present with a generalized papular, crusted and scaly eruption. The scale is typically a “dirty” gray or brown. There may be involvement of the neck, scalp and beard area (Figs. 14A, 14B). We have recently seen a patient with coexistent psoriasis and crusted scabies (Fig. 14C). After two weeks of topical treatment he returned and was still shedding scales (Fig. 15), which demonstrated scabies mites (Fig. 16). This underscores the contagiousness of this condition and the need for isolation of such patients until they are adequately treated and deemed noninfectious. Scabies may also cause papular urticaria with erythematous papules noted on the extremities. Treatment of crusted scabies consists of keratolytics to remove scales and the application of scabicides such as permethrin, lindane or malathion lotion. Recently ivermectin, a drug used for onchocerciasis, has been found to be effective in the treatment of crusted scabies in HIV-infected patients. The dose is 200 g/kg given once followed by a second dose one week later. Family members should be treated, as should fomites. Pediculosis pubis may also be seen in HIV-infected patients. Infestation may also occur on the eyelids or on the trunk in hairy individuals (Figs. 17A, 17B).



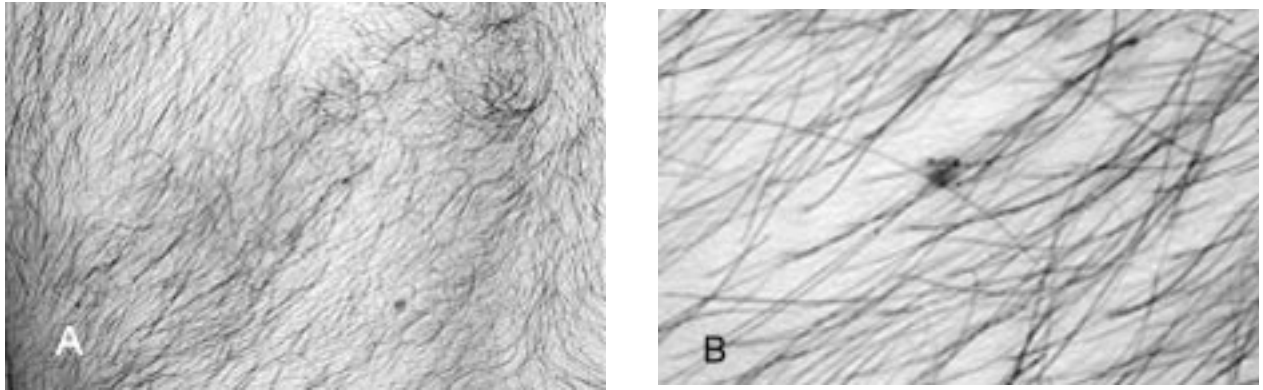
**Fig. 16.** Scabies mite from exfoliated scale.

**Systemic Disease**

Systemic causes of pruritus in HIV-infected patients (other than HIV infection itself) are relatively uncommon (69). They include chronic renal failure as a result of HIV nephropathy, hepatic failure as a result of co-infection with hepatitis B or C, and systemic lymphoma. Drug eruptions are common in the setting of HIV infection and may cause severe pruritus. Moreover, dermatographism may be a cause of itching in



**Fig. 14.** **A.** Crusted scabies on posterior neck and scalp. **B.** Crusted scabies of the beard area. **C.** Crusted scabies in patient with extensive psoriasis. (A, B and C represent the same patient.)



**Fig. 17.** A. Erythematous area on the back of HIV-infected patient with pruritus. B. Closer examination reveals crab louse.

some patients (Fig. 18). Work-up of systemic causes of pruritus includes a history and physical examination, complete blood count, liver and kidney function tests, hepatitis serologies, and chest X-ray. The presence of jaundice, lymphadenopathy, hepatosplenomegaly, or subtle signs of hyper- or hypothyroidism should direct further testing.

### Conclusion

Pruritus is an important cause of discomfort and morbidity in patients infected with the human immunodeficiency virus. Work-up should include a careful skin examination to rule out a primary dermatologic etiology before attributing itching to idiopathic HIV pruritus. If no dermatologic cause is found, a systemic cause or medication-related etiology should be sought.

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**Fig. 18.** Dermatographism is not uncommonly found to be cause of itching.

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