Cutaneous Manifestations of HIV Infection

Dr. K. M. Ho
Special Preventive Programme, Department of Health, Hong Kong

ABSTRACT

The cutaneous manifestations of HIV are diverse, encompassing opportunistic and emerging infections, neoplasms and inflammatory disorders caused by dysregulation of immune system. They could present in the usual way or in various novel atypical appearances. These cutaneous manifestations can arouse one to suspect the diagnosis of HIV infection in an otherwise unwary patient or could lead to the diagnosis of serious complications and also AIDS in a patient known of have HIV infection. The conditions can be understood as usual presentation of a common skin condition, typical presentation of an uncommon condition, atypical presentation of a common condition or an uncommon disorders. Many conditions have been reported to be related to HIV infection in the literature in the form of cross-sectional studies or case reports without more stringent scientific evidence. The discussion is not to be all encompassing but only to highlight some of the features.

Keywords: HIV, cutaneous manifestations, clinical approach

EPIDEMIOLOGY

As at the end of April 2000, there had been 1,399 cases of HIV infection reported to the voluntary reporting system of Department of Health since its establishment at 1984. Of them, 455 had developed AIDS already. The skin is affected in virtually all patients with HIV infection at some point during the course of their illness. According to a study on 145 Malaysian Chinese with HIV infection, 104 (71.7%) had mucocutaneous disorders.1 The patients who presented with mucocutaneous diseases also had low CD4 counts and most had AIDS defining illness already. Generalized hyperpigmentation and thrush were particularly high in this group of patients (Table 1).

PATHOGENESIS

Impairment of the skin immune system occurs early in the natural history of HIV infection and is, in part, due to the specific loss of the antigen presenting dendritic cells which could be directly infected by the HIV. This is apparent clinically from the development of anergy on delayed-type hypersensitivity testing. In addition to the loss of CD4 lymphocytes, the subsets of T-helper cells, Th1 and Th2, have been found to play significant roles in HIV infection and AIDS. With declining immunity there is a shift from the normal Th1 dominated immune response, characterized by promotion of cell-mediated immunity and eradication of infection, to a Th2 dominant immune response. Th2 lymphocytes promote humoral immunity, suppress the Th1 response and are associated with anergy.2

The imbalance of Th1 and Th2 response is well documented in common dermatosis like atopic dermatitis without HIV infection. Many of the unusual cutaneous manifestations of HIV infection may be a result of the process developing in a Th2 setting. Further evidence is added by the fact that Th2 cytokines may promote angiogenesis which is important in Kaposi sarcoma (KS) and bacillary angiomatosis associated with HIV.2

CLASSIFICATION

The skin diseases may present in various ways in a patient with HIV infection. These may include: typical clinical presentation of a common skin disease (such as seborrhoeic dermatitis), atypical presentation of a common skin disease (such as giant molluscum in an
adult man), typical presentation of an uncommon disease (such as Kaposi Sarcoma), and atypical presentation of an uncommon disease (such as extrapulmonary pneumocystosis involving the external auditory canal). In addition, the skin diseases may also be related to the treatment given.

Numerous skin conditions have been reported to be associated with HIV infection. These conditions can be categorized into 3 groups. Group 1 includes conditions that, in the appropriate clinical setting, are specific for (or nearly always associated with) HIV infection. Group 2 includes conditions that either occur with an increased prevalence in HIV-infected patients or whose detection in a previously HIV-negative individual should prompt consideration for additional evaluation for HIV infection. Group 3 includes conditions that are co-incidentally occurring in an HIV-positive person but are not related to that person’s HIV infection (Table 2).³

**Table 1. Mucocutaneous manifestations of HIV infection in Malaysian Chinese¹**

<table>
<thead>
<tr>
<th>Skin disorder</th>
<th>N</th>
<th>Skin disease (%)</th>
<th>Whole group (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hyperpigmentation</td>
<td>52</td>
<td>50</td>
<td>35.9</td>
</tr>
<tr>
<td>Nodular prurigo</td>
<td>43</td>
<td>41.3</td>
<td>29.7</td>
</tr>
<tr>
<td>Xerosis</td>
<td>40</td>
<td>38.5</td>
<td>27.6</td>
</tr>
<tr>
<td>Eczema</td>
<td>8</td>
<td>7.7</td>
<td>5.5</td>
</tr>
<tr>
<td>Hair loss</td>
<td>1</td>
<td>1.0</td>
<td>0.7</td>
</tr>
<tr>
<td>Urticaria</td>
<td>3</td>
<td>2.9</td>
<td>2.1</td>
</tr>
<tr>
<td>Psoriasis</td>
<td>12</td>
<td>11.5</td>
<td>8.3</td>
</tr>
<tr>
<td>Seborrheic dermatitis</td>
<td>30</td>
<td>28.8</td>
<td>20.7</td>
</tr>
<tr>
<td>Tinea/onychomycosis</td>
<td>14</td>
<td>13.5</td>
<td>9.7</td>
</tr>
<tr>
<td>Penicillium marneffei</td>
<td>1</td>
<td>1.0</td>
<td>0.7</td>
</tr>
<tr>
<td>Skin histoplasmosis</td>
<td>3</td>
<td>2.9</td>
<td>2.1</td>
</tr>
<tr>
<td>Candidiasis</td>
<td>52</td>
<td>50</td>
<td>35.9</td>
</tr>
<tr>
<td>Hairy leukoplasia</td>
<td>4</td>
<td>3.8</td>
<td>2.8</td>
</tr>
<tr>
<td>Common warts</td>
<td>1</td>
<td>1.0</td>
<td>0.7</td>
</tr>
<tr>
<td>Genital warts</td>
<td>3</td>
<td>2.9</td>
<td>2.1</td>
</tr>
<tr>
<td>Herpes simplex</td>
<td>3</td>
<td>2.9</td>
<td>2.1</td>
</tr>
<tr>
<td>Herpes zoster</td>
<td>5</td>
<td>4.8</td>
<td>3.4</td>
</tr>
<tr>
<td>Molluscum contagiosum</td>
<td>3</td>
<td>2.9</td>
<td>2.1</td>
</tr>
<tr>
<td>Bacterial folliculitis</td>
<td>3</td>
<td>2.9</td>
<td>2.1</td>
</tr>
<tr>
<td>Abscess</td>
<td>5</td>
<td>4.8</td>
<td>3.4</td>
</tr>
<tr>
<td>Drug eruption</td>
<td>3</td>
<td>2.9</td>
<td>2.1</td>
</tr>
<tr>
<td>Scabies</td>
<td>2</td>
<td>1.9</td>
<td>1.4</td>
</tr>
<tr>
<td>Kaposi’s sarcoma</td>
<td>1</td>
<td>1.0</td>
<td>0.7</td>
</tr>
</tbody>
</table>

(n=104/145)

The significance of skin diseases in HIV infected individual can be many folds. Apart from alerting an individual or the attending physician the underlying diagnosis of HIV infection, it can hamper the quality of life of an individual. It can also be the first clinical manifestation of an underlying important complication of HIV infection such as opportunistic infections. There may also be complicated interaction between the treatment for skin conditions and HIV infection itself and/or antiretroviral treatment. Notorious examples are PUVA, methotrexate, cyclosporin, which should be used with caution for treating patients with HIV and psoriasis.

Skin diseases in HIV infected can be classified under the following categories (Table 3).⁴

**Viral infection**

*Acute exanthema of HIV disease⁵*

Typically it presents as a fine morbilliform eruption involving the trunk, chest, and upper arms simulating measles/rubella. It usually develops after 2-4 weeks of infection and is accompanied by systemic ‘flu’ like illness.
Table 2. Classification of mucocutaneous conditions in patients with HIV infection

**Group 1: Conditions that are specific for HIV infection**
- Bacillary angiomatosis
- Diffuse interstitial lymphocytosis syndrome
- Herpetic geometric glossitis
- Kaposi sarcoma
- Oral hairy leukoplakia
- Proximal white subungual onychomycosis

**Group 2: Conditions with an increased prevalence in patients with HIV infection**
- Acquired ichthyosis
- Alopecia areata and alopecia universalis
- Candidiasis (esophageal, oral, and vulvovaginal)
- Dermatofibromas (multiples)
- Dermatophyte infection - nails and skin
- Drug reactions
- Eosinophilic folliculitis
- Epithelial neoplasms (basal cell carcinoma and mucosal tumors)
- Eythema elevatum diutinum
- Eyelash trichomegaly - acquired
- Granuloma annulare
- Herpes simplex virus infection
- Human papillomavirus infection
- Lymphomas - especially non-Hodgkins B-cell
- Molluscum contagiosum
- Photo-induced and photoaggravated conditions
- Porphyria cutanea tarda
- Prurigo nodularis
- Psoriasis vulgaris
- Scabies
- Seborrheic dermatitis
- Staphylococcus aureus infection
- Syphilis
- Systemic fungal infection - especially with cutaneous and oral fungal - related lesions
- Cryptococcosis
- Histoplasmosis
- Varicella-zoster virus infection - especially disseminated or more than 1 episode
- Vitiligo

**Group 3: Conditions that coincidentally occur in patients with HIV infection**
- Autoimmune bullous disorders
- Calciphylaxis
- Granuloma inguinale
- Kawasaki disease - adult onset
- Lichen amyloidosis
- Pityriasis rubra pilaris
- Pyoderma gangrenosum
- Reactive perforating dermatosis
- Transient acantholytic dermatosis

Table 3. Clinical Classification of Skin Conditions in Patients with HIV Infection

<table>
<thead>
<tr>
<th>Bacterial infections</th>
<th>Fungal infections</th>
<th>Viral infections</th>
<th>Inflammatory dis.</th>
<th>Miscellaneous</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bacterial folliculitis</td>
<td>Superficial</td>
<td>Herpes zoster</td>
<td>Papulosquamous</td>
<td>Neoplastic</td>
</tr>
<tr>
<td>Boil</td>
<td>Dermatophytosis</td>
<td>Herpes simplex</td>
<td>Seborrheic</td>
<td>Kaposi's sarcoma (KS)</td>
</tr>
<tr>
<td>Impetigo</td>
<td>Pityrosporum yeast</td>
<td>Molluscum</td>
<td>Dermatitis</td>
<td>Non Hodgkin's lymphoma (NHL)</td>
</tr>
<tr>
<td>Skin abscess</td>
<td>Systemic</td>
<td>Contagiosum</td>
<td>Psoriasis</td>
<td>Arthropod</td>
</tr>
<tr>
<td>Syphilis (1st &amp; 2nd)</td>
<td>Cryptococcosis</td>
<td>Human papilloma wart (HPV)</td>
<td>Atopic dermatitis</td>
<td>Scabies</td>
</tr>
<tr>
<td>Tuberculosis</td>
<td>Penicilliosis</td>
<td>Pruritic papular</td>
<td>Eosinophilic</td>
<td>Miscellaneous</td>
</tr>
<tr>
<td>Atypical</td>
<td></td>
<td>Folliculitis</td>
<td>Folliculitis</td>
<td>Nail and hair</td>
</tr>
<tr>
<td>Mycobacterium</td>
<td></td>
<td>Drug eruption</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bacillary</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Angiomatosis</td>
<td></td>
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</tr>
</tbody>
</table>
**Herpes simplex virus**

Typically the primary skin lesion is vesicles arranged in crops involving orofacial or genital area. Atypical presentations include persistent non-healing ulcers, warty molluscum like nodular lesion, and disseminated and generalized involvement of the body.

The diagnosis is by clinical examination confirmed by viral culture, direct immunofluorescence study or histopathology. The treatment is with acyclovir.

**Varicella zoster virus**

Typically the primary skin lesion is vesicles arranged in crops distribute along one or more than one dermatomes. Atypical presentations include persistent non-healing ulcers, warty molluscum like nodular lesion, and disseminated and generalized involvement of the body. Recurrent zoster is not uncommon.5

The diagnosis is made clinically, but viral culture, direct immunofluorescence study or histopathology may sometimes be required for confirmation. The treatment is with systemic acyclovir.

**Molluscum contagiosum**

Typically the primary skin lesion is white or red papules or small nodules with or without umbilication. Atypical presentations include: large and multiple lesions on the face in adults. The diagnosis is mainly by clinical, but histopathology may be required for confirmation in atypical cases. Curettage and iodinization has been used as treatment. Spontaneous resolution after HAART has been reported.

**Human papilloma virus**

Typically the primary skin lesion is warty papules or small nodules. Subclinical wart virus infection is very common as well. Atypical presentations include multiple large lesions that are refractory to conventional treatment.

The diagnosis is mainly clinical, but may also be confirmed by histopathology. Type 6 and 11 are usually responsible for genital wart while 16, 18, 31 and 33 are responsible for cloacogenic carcinoma.

Treatments commonly used in our locality include topical podophyllin/podophyllotoxin, cryotherapy, electrocauterization, topical imiquimod. Treatment for wart in person with HIV infection may be prolonged and relapses are common.

**Bacterial infections**

**Boils, folliculitis, impetigo or related conditions**

Typically the primary skin lesion is pustule for boil and folliculitis. The primary skin lesion in impetigo is weeping macule or small patch with peripheral fine scale. They are commonly cause by staphylococcus or streptococcus. Pseudomonas may be the cause in some cases. Atypical presentations include botryomycosis that presents with papules or plaques surrounded by pustules on the trunk, neck or extremities related to staphylococcus.

The diagnosis is made clinically with swab for culture. The treatment is as for person without HIV infection by antibiotics, antiseptics with or without surgical drainage.

**Tuberculosis**

Both Mycobacteria tuberculosis and non-tuberculosis mycobacteria can cause skin lesions. The skin can be involved directly by the mycobacteria, as part of disseminated disease or reaction to an underlying infection (tuberculids). Therefore, mycobacterial skin lesions may assume various different appearances. Small papules and pustules that resemble folliculitis, eruption that resemble atopic dermatitis, localized cutaneous abscess, suppurative lymphadenitis, non-specific ulcerations, palmar and plantar hyperkeratoses, and sporotrichoid nodules all have been reported.5

The diagnosis is by culture or by histopathology supplemented with molecular diagnostic technique such as PCR. The treatment is similar to those with systemic involvement according to the type of mycobacteria isolated and the immune status of the individual.

**Syphilis**

The typical primary skin lesion in primary syphilis is chancriform ulcer usually over the genital area. Skin lesions in secondary syphilis can be protean in their manifestation. Classically, it is papulosquamous lesions with involvement of the palms and soles, and mucous membrane lesions like snail track ulcer and condylomata lata.

Atypical unusual presentations that may defy diagnosis include rapid progression from the primary chancre through to secondary stages, lues maligna, sclerodermoid lesions, papular verrucous plaques, extensive oral ulcerations, keratoderma, deep cutaneous nodules, rubeoliform eruptions.5
The diagnosis is by demonstration of the typical spirochaette in the mucocutaneous lesions or histopathological sections, or by serology. More aggressive treatment than non-HIV infected patient is usually adopted but not with firm scientific support. Aggressive course and progression to late systemic syphilis, and relapse after apparently adequate treatment is well reported. Close monitoring is recommended.

**Bacillary angiomatosis**

Typically the primary lesions are vascular papules or nodules (pyogenic granuloma like) distribute over the face or upper trunk. It is an important differential diagnosis for Kaposi Sarcoma (KS). The diagnosis is by histopathology with special stain (e.g. Warthin Starry stain). Treatments include macrolide (or azalides) antibiotics, and doxycycline. Ciprofloxacin, rifampicin and septrin have also activity against the causative organism (Bartonella sp.).

**Fungal infection**

**Candida sp**

Candida infection in HIV infected person typically presents with oral thrush (classic thrush, angular cheilitis, diffuse glossitis or median rhomboid glossitis), vaginal thrush (candida balanitis in male), other presentation like intertrigo, paronychia with or without onychomycosis and disseminated infection are well known. The beefy red colour, satellite lesions with peripheral scallop scale are other useful signs in arousing the suspicion of the diagnosis.

The diagnosis is usually by clinical supplemented by culture technique. The treatment: is by oral imidazole or systemic triazoles. Amphotericin B is occasionally required for severe infection.

**Superficial dermatophytosis**

Dermatophytes can affect the body (tinea corporis), sole (tinea pedis), groin (tinea cruris), face (tinea faciale), scalp (tinea capitis) and nail (tinea unguium; onychomycosis). Typically the presentation is classic annular lesion with active margin and central clearing in tinea corporis, faciale and cruris, and diffuse hyperkeratosis or vesiculation in tinea pedis. Atypical presentation for tinea faciale mimicking erythema multiforme or seborrhoeic dermatitis, and tinea pedis presenting as keratoderma blennorrhagica like lesion has been reported.

The diagnosis is by microscopic examination or culture of scale/nail/hair sample obtained. Treatments commonly used are topical imidazoles, Whitfield ointment, oral griseofulvin, itraconazole, or terbinafine.

**Pityrosporum yeast**

It can cause pityriasis versicolor, pityrosporum folliculitis and is also related to seborrhoeic dermatitis. The typical presentation is pityriasis versicolor with hypopigmented macules with superficial fine powdery scale over upper trunk. Pityrosporum folliculitis presents with itchy monomorphic folliculitis or follicular papules over upper back, chest or face.

The diagnosis is by clinical or microscopic examination of scale obtained or by histopathology. The treatment used is topical or oral imidazoles (or triazole).

**Penicilliosis**

Typical lesions are umbilicated papules simulating molluscum contagiosum. Atypical presentation may include eczema, folliculitis, subcutaneous nodule and morbilliform rash. The diagnosis: is by histopathology and/or culture of biopsy specimen. The treatment used can be amphotericin B or itraconazole.

**Cryptococcus**

Typically it presents as molluscum contagiosum like lesions. Atypical presentation mimic HSV, cellulitis, HS or hypertrophic lesions like rhinophyma. The diagnosis is by histopathology and/or appropriate culture. Treatments used are amphotericin B or fluconazole/itraconazole.

**Other fungi**

Histoplasma, blastomycosis and coccidiomycosis can also present with skin lesions (usually mimicking molluscum or KS/pyogenic granuloma). However, they are seldom reported in this part of the world.

**Parasite and protozoa**

**Scabies**

The typical presentation is itchy papulovesicular lesions affecting finger web, palm, volar wrist, axilla, waist, groin and genital area with positive family history. Norwegian scabies is well reported. Clinically, it presents with crusted (hyperkeratotic) plaques on erythematos base distribute all over the body including face. Itchiness may be absent.
The diagnosis is made clinically supplemented by microscopic examination of skin scraping or biopsy. The treatment is same as for non-HIV cases. Newer treatments like topical permethrin or oral ivermectin is not yet available in our locality.

**Pneumocystosis**
Extrapulmonary involvement is well reported. The typical skin lesions are vascular papulonodules on the external auditory canal or nare of patients who have received inhalation pentamidine prophylaxis. The diagnosis is by histopathology. The treatment is by septrin or systemic pentamidine.

**Neoplastic diseases**

**Kaposi sarcoma**
The typical presentation in KS are asymptomatic bluish/reddish macules, papulonodules or plaque on nearly any sites on the body. The diagnosis is by clinical and confirmed by histopathology. It is thought to be related to human herpes virus 8 (renamed as KSHV). The treatment may be expectant, local destructive, local chemotherapeutic or radiotherapy or systemic chemotherapy dependent on the symptom and organ involvement. HAART may sometimes induce remission of KS.

**Non-Hodgkin’s lymphoma (NHL)**
The B-cell types of NHL may present as fleshy skin papulonodules/plaques. The T-cell types may present as bizarre shaped patches, plaques or nodules with superficial scaling and inter/intra-lesional variation.

The diagnosis is by histopathology supplemented by immunohistochemical/molecular techniques. The treatment of B-cell type NHL is mainly by systemic chemotherapy. The treatment for T-cell type NHL are chemophototherapy, radiotherapy (including total body electron beam), interferon, and systemic chemotherapy.

**Cloacogenic carcinoma (or related dystrophic conditions)**
It is related to HPV infection and the pathogenesis is probably quite similar as that for carcinoma of the cervix in female. The disease incidence is increased in HIV infected population. Typically these lesions present as wart like papules ( Bowenoid papulosis/vulval dystrophy) or squamous carcinoma like exophytic growth involving anogenital area. The diagnosis is by histopathology. The treatment is as for non-HIV infected person with similar conditions.

**Inflammatory dermatosis**

**Papulosquamous disorders**
This group of skin conditions encompasses psoriasis, dermatitis, lichen planus, etc.

**Psoriasis**
Psoriasis develops in 5% of patients with HIV infection. Patients with HIV-associated psoriasis may develop arthritis in up to 10% of cases, an incidence that is significantly higher than non-HIV infected patients with psoriasis.

Psoriasis typically presents as erythematous well demarcated plaque with silvery scale distribute more on the extensor surface of the limbs and body. HIV-related psoriasis may develop in patients with mild pre-existing psoriasis that suddenly undergoes severe exacerbation in patients once AIDS develops, or it may develop spontaneously at some point after HIV seroconversion in an individual who has never before had clinical disease. The diagnosis is usually by clinical and occasionally by histopathology.

The treatment for psoriasis composes of topical tar, salicylic acid, steroid, dithranol, calcipotriol, phototherapy (PUVA can theoretically depress the body immunological function), and systemic agents such as retinoid, methotrexate (that should also be used with caution in HIV patient).

**Seborrhoeic dermatitis**
This is probably one of the commonest skin condition encountered in patient with HIV infection. It is seen in 85% of all HIV-infected individuals at some point during the course of their disease.

Typical presentation is poorly marginated scaly erythematous patches affecting glabella, nasolabial fold, external auditory canal, scalp, presternal area and occasionally the groin as well. Atypical presentation in unusual sites such as the trunk and extremities may occur in HIV patients. It can be extensive and resistant to conventional treatment.

The diagnosis is by clinical examination. The treatment is by combination of topical imidazole with
topical steroid, as pityrosporum yeast is thought to play an important role in this papulosquamous disorder.

**Atopic dermatitis**

In patients with HIV infection, the typical presentation is the recurrence of atopic dermatitis which have been in remission for some years, when they become immunological deficient. Erythroderma had been reported. Typically the skin rash presents as itchy poorly demarcated erythematous papovesicular lesion with weeping, scale crust that have a characteristic distribution. There are also different protean minor manifestations.

The diagnosis is by clinical examination (strict criteria as proposed by Hannaffin or British Working Group should be used to qualify the diagnosis of atopic dermatitis). The treatment is by emollient, topical steroid, and occasionally phototherapy and short course of systemic steroid.

**Xerosis, ichthyosis, and astematotic dermatitis**

Dry skin is very common in patient with HIV infection, and is probably the commonest cause of pruritus in this group of patients. Typically the skin is dry and flaky with or without excoriation marks. Its aetiology is not certain.

The diagnosis is mainly on clinical ground but it is very important to rule out other important dermatological conditions that can also present with pruritus. The treatment is by proper skin care and liberal use of emollient.

**Pruritic papular eruption**

This was originally described as a specific entity that affected HIV infected individual of African black ethnic origin. However, it is now believed to be a result skin diseases of heterogeneous causes.

Eosinophilic folliculitis is the more well clarified entity and is one of the most common pruritic dermatosis to develop in patient with HIV infection. Patients generally present with widespread excoriated follicular papules that involve the trunk, extremities, and head and neck. Intact pustules are unusually be seen. It is important to look for other important conditions that may present with pruritus such as scabies, NHL or dermatitis herpetiformis.

The diagnosis is by clinical and histopathology. Treatment is with oral isotretinoin, metronidazole, itraconazole or UVB have been reported with variable success.

**Cutaneous drug reaction**

Cutaneous drug eruptions are the most common manifestation of drug hypersensitivity. The incidence of drug reaction is higher in patients with HIV infection. Septrin, anti-TB drugs and NNRTIs are the well reported culprits.

Moribilliform rash is probably the commonest type of drug rash. Stevens-Johnson syndrome and toxic epidermal necrolysis are also well known. Penile ulceration associated with foscarnet, pentamidine associated skin ulceration, urticarial and lichenoid rash have also been described.

The diagnosis is mainly by clinical assessment supplemented by histopathology. Treatment includes withdrawal of culprit drug and supportive management that is most important.

**Nail and hair**

Proximal white subungual onychomycosis is thought to be highly suggestive of HIV infection. The incidence of candida and scytalidium (and other non-dermatophyte filamentous fungus) related onychomycosis is increased. Yellow nail syndrome, nail ridging and opacity and Beau's line are well reported. Melanonychia is associated with zidovudine treatment. Hair abnormalities have been described in patients with HIV infection. These include telogen effluvium, premature graying, diffuse thinning, alteration of texture and alopecia areata.

**Miscellaneous dermatoses**

Generalized hyperpigmentation is very common in patient with long standing HIV infection. Porphria cutanea tarda, granuloma annulare, pityriasis rubra pilaris, vasculitis are all described in patients with HIV infection.

**PRINCIPLES OF MANAGEMENT**

As skin diseases are so common and the spectrum of dermatoses in HIV infected individual is so perplexing, it is not to be expected that the correct diagnosis and significance can always be reached without difficulty. It is important to differentiate the
type of primary lesion, the configuration and distribution of the lesions affecting the individual.

Investigation armament is similar to that as for patient without HIV infection. However, special stain, culture technique, immunohistochemical or molecular technique may be necessary for the diagnosis of certain conditions, such as Warthin-Starry stain is required for the diagnosis of bacillary angiomatosis.

Majority of the topical treatment can be used in person with HIV infection. Systemic therapy include phototherapy should be used with caution. The treatment course may be longer than that as required for non-HIV infected. The response to treatment may be sub-optimal and relapse on cessation of treatment is not uncommon. The disease course of some of the skin conditions may not run parallel to that of HIV infection. On the other hand, some skin condition may improve on control of HIV infection with HAART such as Kaposi Sarcoma, and occasionally psoriasis.

The diagnosis of many dermatological conditions may not be straight forward and the histological pattern of individual skin disease may not be specific. Follow-up the disease course, therapeutic trial and input from various disciplines are essential for the diagnosis of some unusual dermatoses.

Impact of HAART in management of HIV infection

HAART is the abbreviation of 'Highly Active Anti-Retroviral Therapy' or commonly known as cocktail therapy. It is usually composed of three anti-retroviral agents. A commonly used combination comprises one (or two) protease inhibitor (indinavir/nelfinavir, and ritonavir/saquinavir which are not as commonly used as the first line protease inhibitor) or one non-nucleoside reverse transcriptase inhibitor (efavirenz or nevirapine) together with two nucleoside analogue type of reverse transcriptase inhibitors (zidovudine, didanosine, zalcitabine, lamivudine, stavudine).

HAART has significantly improved both the morbidity and mortality of patient with HIV infection. Development of AIDS has been prolonged. The number of opportunistic infections is decreased. Patients can discontinue prophylaxis for certain opportunistic infections as their CD4 count is raised by the treatment. Majority of patients on HAART are ambulatory and can go back to their work nowadays.

Dramatic improvement of certain dermatological conditions, which typically occur with a more depressed level of cellular immunity in patients with HIV infection, are well documented coincident with immune reconstitution by the use of HAART. These include infective conditions like molluscum, wart, neoplastic conditions like Kaposi sarcoma and inflammatory condition like psoriasis.

There could be a new spectrum of dermatological conditions related to HAART such as lipodystrophy, drug related skin rash and those related to immune reconstitution. Close liaison with various disciplines is important in the overall management of these group of patients.

References