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Prevalence and Pattern of Dermatophytosis in Patients with Human Immunodeficiency Virus Infection Seen in The University of Port Harcourt Teaching Hospital, (UPTH) Port- Harcourt

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ABSTRACT

Background: Immunosuppression due to various aetiologies have been associated with the occurrence of dermatophytosis. Several studies in the past have demonstrated that Human Immunodeficiency Virus (HIV) infection is a risk factor for the acquisition and severity of dermatophytosis.
Aim: This study examined the prevalence and pattern of dermatophytosis among patients with HIV infection in the University of Port - Harcourt Teaching Hospital, Port Harcourt, Southern Nigeria.
Method: This was a cross-sectional study carried out in Port Harcourt over a 6 month period involving 173 HIV sero-positive cases and 173 seronegative controls subjects. They were interviewed with a structured questionnaire and thereafter screened for the presence of dermatophytosis. Samples were collected from those with clinically diagnosed dermatophytosis and sent for mycology studies. Information were anaylsed using SPSS version 20.
Results: There was a higher prevalence of dermatophytosis in the HIV seropositive group when compared to the HIV seronegative control made up of HIV seronegative subjects. Most of the lesions seen were not markedly different from that seen in immunocompetent persons. 41.65% of the cases were found among those with Cd4 cell counts below 200cells/µl. Tinea corporis was the commonest lesion seen (50%). Trichophyton species was the commonest dermatophyte isolated, followed by Microsporum species.
Conclusion: The prevalence of dermatophytosis is significantly higher in HIV infected patients and commonly occurs in advanced stages of the disease. Tinea corporis is the most common lesion in this group of patients and Trichophyton species a common causative agent.

INTRODUCTION

Dermatophytosis is a superficial fungal infection of the skin, hair or nails. Dermatophytes are characterised by their ability to exist and grow in keratin, enabling them to invade the stratum corneum of the skin and keratinized structures such as hair and nails with minimal stimulation of the host's immune response. Dermatophytes belong to 3 genera:

- Trichophyton
- Microsporum
- Epidermophyton

The growth of dermatophytes in keratin is restricted to production of hyphae, which branch and segment into chains of spores called arthrospores or arthroconidia. Arthrospores are the main means of dissemination and propagation of the fungus and can remain viable and in the environment and exfoliated skin for many months and even years.

The type and extent of fungal invasion in dermatophytosis of the hair as well as its clinical features differs according to the species of fungi. The hyphae and arthrospores of some species remain within the hair shaft (endothrix) while others form a sheath of arthrospores around the shaft of the hair (ectothrix).

Dermatophytes grow best in a warm, humid environment and are therefore more common in the tropical and subtropical regions. The geographic distribution varies with the organism *Microsporum canis*, *Microsporum nanum*, *Trichophyton mentagrophytes, Trichophyton verrucosum* and *Trichophyton equinum* occur worldwide[1]. *Trichophyton simii* (found in monkeys) occurs only in Asia, and *Trichophyton mentagrophytes* var. *erinacei* is limited to France, Great Britain, Italy and New Zealand[1]. *Trichophyton schoenleinii* and *Trichophyton soudanense* are commonly found in Africa. In the West African sub-region, *Trichophyton soudanense, Microsporum audounii, Microsporum canis, Trichophyton violaceum,* and *Trichophyton rubrum* are common aetiological agents of dermatophytosis[1,2].

There are about 40 recognised specie of dermatophytes. Some are only able to infect man (anthropophilic), others are primarily animal pathogens (zoophilic) but can also infect man. Other species are found as saprophytes in the soil (geophilic), and cause sporadic infection in man and animals.

Dermatophytosis are mild communicable diseases with high morbidity and contribute to major health problems in the tropics and sub tropics especially in Nigeria. Most surveys on dermatophytosis done in the past have been carried out in school children and have concentrated mainly on tinea capitis. This maybe because of the social stigma attached to it, possibility of alopecia and other associated secondary diseases such as bacterial infection of the lesions.

The prevalence rate of dermatophytosis in the general population in a study done in Lagos is about 6.1%[3].

Transmission: Infection occurs by contact with arthrospores (asexual spores formed in the hyphae of the parasitic stage) or conidia (sexual or asexual spores formed in the "free living" environmental stage). Infection usually begins in a growing hair or the stratum corneum of the skin. Dermatophytes do not generally invade resting hairs, since the essential nutrients they need for growth are absent or limited. Hyphae spread in the hairs and keratinized skin, eventually developing infectious arthrospores. Transmission between hosts usually occurs by direct contact with a symptomatic or asymptomatic host, or direct or airborne contact with its hairs or skin scales. Infective spores in hair and dermal scales can remain viable for several months to years in the environment[4].

Geophilic dermatophytes, such as *Microsporum nanum* and *Microsporum gypseum*, are usually acquired directly from the soil rather than from another host[4].

Fomites are also another important means of transmission[5,6].

Factors affecting infection:

Factors inhibiting the growth of dermatophytes include saturated fatty acids in sebaceous glands[7].
Neutrophils and monocytes have been found to kill dermatophyte conidia, a process which depends on both intra-and extracellular mechanisms[8].

Antibodies to dermatophytes have not been found to be protective[9]; however development of cellular immunity via sensitized T-lymphocytes is a key factor in immunological defence[10]. Host immunity against dermatophytosis depends on both innate and acquired immune mechanism. Chronic infections are associated with poor T-lymphocyte function[11,12].

Other host factors affecting infection are as follows- genetic susceptibility, reduced immunity in old age, diabetes mellitus, Cushing's syndrome, and HIV infection[13-17].

Environmental factors like humidity and raised CO_2 tension favour dermatophyte invasion[18]. Raised temperature of more than 37^{o}C inhibits dermatophyte growth[19]. This is partly responsible for the lack of deeper penetration of the skin in dermatophytosis.

Clinical Features:

The disease produced by dermatophytes are described according to anatomic site involved viz – tinea capitis (scalp), tinea barbae (bearded skin of the face), tinea corporis (the body), tinea cruris (groin), tinea unguium (the nails), tinea manuum (hand) and tinea pedis (the feet). These infections may vary from mild inflammations to acute vesicular reactions.

The incubation period in humans is 1 to 2 weeks.

Tinea Capitis: This occurs predominantly in prepubertal children. Adult infection is rare. One risk factor for adult infection is immunosuppression from HIV or drugs. Microsporum and Trichophyton species are the main aetiological agents. The most common causative fungi is *Trichophyton tonsurans* and

Microsporum canis. Lesions vary from a dry, scaly patch of alopecia to the development of pustules and abscesses also known as kerion.

Tinea Barbae: This refers to dermatophytic infection of the bearded area of the face. It may present as folliculitis or as a severe inflammatory reaction consisting of papules, pustules, exudates and crusting. Common causative organisms include *Trichophyton veruccossum, Trichophyton violaceum, Trichophyton mentagrophytes, Trichophyton schoenleinii, Microsporum canis* and *Trichophyton rubrum.*

Tinea Corporis: This refers to dermatophytosis of the skin excluding the hair, nails and feet. Skin lesions may be dry and scaly or moist and crusty. As they enlarge their centres heal producing the classic annular lesions. Lesions may also be pustular, vesicular and occasionally granulomatous.
All dermatophytes can produce tinea corporis. Tinea incognito occurs if a topical steroid has been applied and the clinical appearance of the initial tinea lesion is altered, becoming less scaly, more extensive, pustular, pruritic, and painful.

Tinea Cruris: Dermatophytosis of the groin area. Infections occurs in proximal thighs, crural folds and extends onto the buttocks. Lesions are raised, sharply defined, erythematous and pruritic.
Common causative organisms include: *Trichophyton rubrum, Epidermophyton floccosum, Trichophyton mentagrophytes var interdigitale.*

Tinea Pedis: This refers to dermatophytosis of the foot including the plantar surface and toe web space. It presents with itching pain, maceration, and hyperkeratosis of soles and sides of the feet.
Causative organisms include *Trichophyton rubrum, Trichophyton mentagrophytes and Epidermophyton flocossum*.

Tinea Manuum: Dermatophytosis of the hand especially involving the palmar surface, it presents as a dry scaling eruption, may also presents as ulcerations and hyperkeratosis. *Trichophyton rubrum* is commonly implicated.

Tinea Unguium: Dermatophytosis of the nail plate. Also called onychomycosis. It usually starts at the tip of one or more nails. There is a gradual thickening, discoloration and crumbling of the affected nail which may eventually be completely destroyed. Causative fungi include *Trichophyton rubrum, Trichophyton mentagrophytes var interdigitale* and *Epidermophyton floccosum.*

Chronic dermatophyte infections may be the first manifestation of HIV and may suggest HIV infection because of increased severity of presentation, atypical clinical appearance or increased resistance to treatment.

The burden of HIV: HIV infection is a major challenge and health problem worldwide. It was first discovered in Los Angeles**,** California in young homosexual men who presented with disseminated Kaposi sarcoma and pneumocystic carinii infection in 1981[20,21,22].

It later became evident that this disease affected other population groups as well when some cases were reported in intravenous drug users. In 1983, almost 2 years later HIV was defined as the primary cause of Acquired Immunodeficiency Syndrome (AIDS)[23,24,25]. Since then it has become a global problem with rising incidence in various parts of the world.

Worldwide more than 34 million people are infected with 69% in Sub Saharan Africa. Globally, 0.8% of adults aged 15-49 years are living with the virus[26].

The prevalence rate for HIV in Nigeria is 4.1%[27] previously, now 1.4%.[26] The geo-political zone with the highest HIV sero-prevalenceis the North-central zone (7.5%); while the North-western zone has the lowest prevalence rate of 2.1%[27]. Benue state in the North central zone has the highest prevalence of 12.7% while Kebbi state in the North-western zone has the lowest prevalence of 1.0%[27]. A total of 3,459,363 are currently infected with the virus in this country[27]. Urban areas have a higher prevalence than rural areas[27]. Women, youths, and people with a low level of education are worst affected[27].

Human Immunodeficiency Viral infection are commonly associated with a myriad of skin conditions. Examples include pruritic papular eruption, herpetic infections, Kaposi sarcoma and dermatophytosis and these skin conditions may be the first manifestation of the disease.

The development of cellular immunity via sensitised T-lymphocytes is a key factor in immunological defence against dermatophytic infections. HIV alters this defence by producing cellular immunodeficiency characterised by the depletion of Helper T-lymphocytes (CD4 cells) thereby predisposing to dermatophyte infections.

Animal models have shown that Langerhan's cells which act as antigen presenting cells to dermatophyte antigen are the first cellular targets of HIV. The virus fuses with these cells and spreads into deeper tissues[28,29]. The virus has also been identified in epidermal Langerhan cells in HIV infected patients[30], and these patients have a reduced number of such cells which leads to a compromise in the skin immune response which may result in multiple or extensive skin infections.

Acquired immune deficiency syndrome (AIDS) was first discovered as a novel disease in 1981.[21] Within 2 years of defining AIDS as a distinctive syndrome in 1981, the human immunodeficiency virus (HIV) was identified as the causative agent. HIV infection is acquired sexually, from blood or blood products, or vertically from an infected mother to her child during pregnancy, delivery or breastfeeding. The virus infects immunocompetent cells including CD4 T-cells and macrophages. It creates variable patterns of disease in individuals, groups and races. These diseases are characterized by evolving, sometimes fulminant immunodysfunction (AIDS) affecting many systems of the body.

Aetiology:

HIV belongs to the lentivirus group of the retrovirus family. There are two types, HIV-1 and HIV-2. HIV-1 is the most frequently occurring strain globally. HIV-2 is almost entirely confined to West Africa, although there is evidence of some spread to Europe, particularly France, Portugal and the Indian subcontinent[31]. HIV-2 has only 40% structural homology with HIV-1 and although it is associated with immunosuppression and AIDS, appears to take a more indolent course than HIV-1. Many of the drugs that are used in HIV-1 are ineffective in HIV-2. The structure of HIV is shown in figure 1 below.

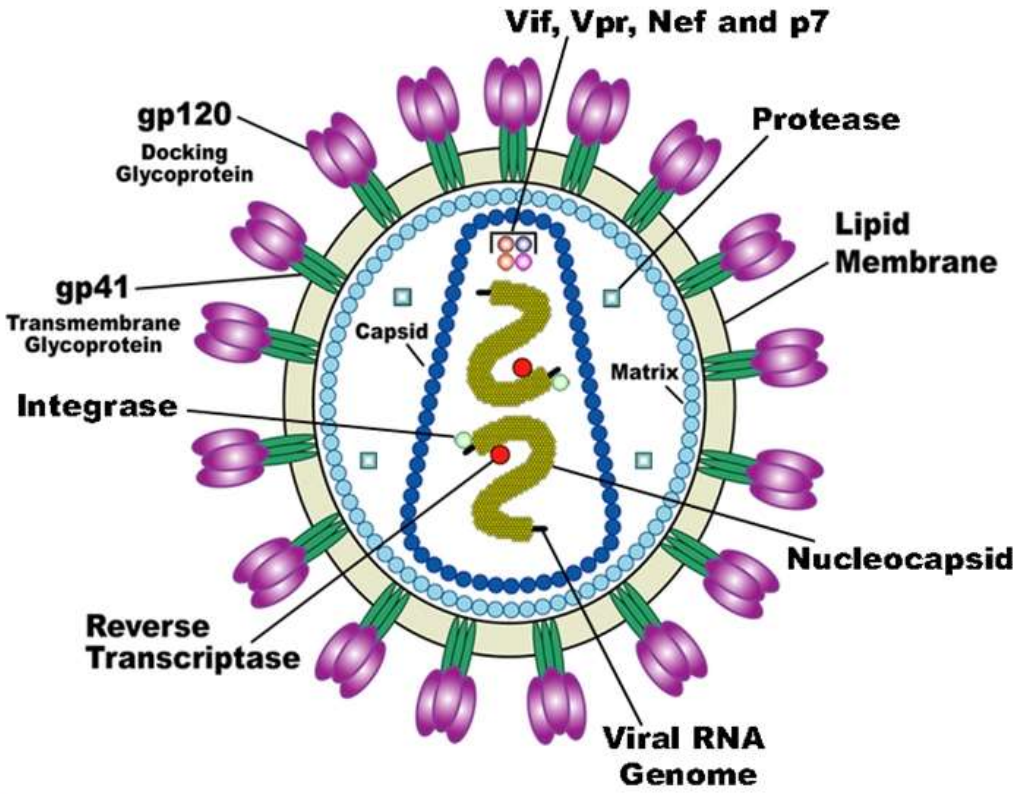


Figure 1: Diagram of Human Immunodeficiency Virus.

Retroviruses are characterized by the possession of the enzyme reverse transcriptase, which allows viral Ribonucleic Acid (RNA) to be transcribed into Deoxyribonucleic Acid (DNA), and thence incorporated into the host cell genome. Reverse transcription is an error-prone process with a significant rate of mis-incorporation of bases. This, combined with a high rate of viral turnover, leads to considerable genetic variation and a diversity of viral subtypes or clades. Upon entry into the target cell, the viral RNA genome is converted (reverse transcribed) into double-stranded DNA by a virally encoded reverse transcriptase that is transported along with the viral genome in the virus particle. The resulting viral DNA is then imported into the cell nucleus and integrated into the cellular DNA by a virally encoded integrase and host co-factors. Once integrated, the virus may become latent, allowing the virus and its host cell to avoid detection by the immune system. Alternatively, the virus may be transcribed, producing new RNA genomes and viral proteins that are packaged and released from the cell as new virus particles that begin the replication cycle anew. On the basis of DNA sequencing, HIV-1 is divided into three groups (M, N, and O), which probably represent three zoonotic transfers from the chimpanzee but do not differ clinically in humans[32].

Group M (major) subtypes (95% of infections worldwide) contains at least 8 subtypes (or clades), which are denoted A–J[33]. There is a predominance of subtype B in Europe, North America and Australia, but areas of central and sub-Saharan Africa have multiple M subtypes. Subtype G is predominant in Nigeria. Recombination of viral material generates an array of circulating recombinant forms (CRFs), which increases the genetic diversity that may be encountered.

Group N (new) is mostly confined to parts of West-Central Africa (e.g. Gabon).

Group O (outlier) subtypes are highly divergent from group M and are largely confined to small numbers centred in Cameroon.

Transmission:

Despite the fact that HIV can be isolated from a wide range of body fluids and tissues, more than 90% of HIV infections are transmitted via semen, cervical secretions and blood.

Sexual intercourse (vaginal and anal):

Globally, heterosexual intercourse accounts for the vast majority of infections, and coexistent sexually transmitted infections (STIs), especially those causing

genital ulceration, enhance transmission[34,35]. Transmission of HIV appears to be more efficient from men to women, and to the receptive partner in anal intercourse, than vice versa. In the United States, as of 2009, most sexual transmission occurred in men who had sex with men- with this population accounting for 64% of all new cases[36]. In central and sub-Saharan Africa (Nigeria inclusive) the epidemic has always been heterosexual and more than half the infected adults in these regions are women[26]. SouthEast Asia and the Indian subcontinent are experiencing an explosive epidemic, driven by heterosexual intercourse and a high incidence of other sexually transmitted diseases[37].

Mother-to-child (transplacentally, perinatally, breastfeeding):

Mother-to-child transmission is the third most common route of HIV infection globally[34]. Studies suggest that, without intervention, In the absence of treatment, the risk of transmission before or during birth is around 20% and in those who also breastfeed 35%[36]. As of 2008, vertical transmission accounted for about 90% of cases of HIV in children[36]. With appropriate treatment the risk of mother-to-child infection can be reduced to about 1%[36]. In the developed world interventions to reduce vertical transmission, including the use of antiretroviral agents, delivery by caesarean section and the avoidance of breast-feeding have led to a dramatic fall in the numbers of infected children. However, with the advent of highly active anti-retroviral therapy (HAART), mothers are encouraged to exclusively breastfeed their babies as the benefits outweigh the risks.

Contaminated blood, blood products and organ donations: Screening of blood and blood products was introduced in 1985 in Europe and North America. Prior to this, HIV infection was associated with the use of blood products (in haemophiliacs) and with blood transfusions. In some parts of the world where blood products may not be screened, and in areas where the rate of new HIV infections is very high, transfusion-associated infections continues to occur. HIV is transmitted in About 93% of blood transfusions involving infected blood[39].

Contaminated needles (intravenous drug misuse, injections, needle-stick injuries): The practice of sharing needles and syringes for intravenous drug use continues to be a major route of transmission of HIV in both developed countries and parts of South East Asia, Latin America and the states of the former Soviet Union. In some areas, including the UK, successful education and needle exchange schemes have reduced the rate of transmission by this route. Iatrogenic transmission from needles and syringes used in developing countries is reported. Healthcare workers have a risk of approximately 0.3% following a single needle-stick injury with known HIV-infected blood[40].

Immunology And Pathogenesis Of HIV Infection: Primary HIV infection results in natural or innate immune responses that are mobilized within hours of infection and include inflammation, non-specific activation of macrophages, natural killer cells and complement, and release of cytokines. After antigenic stimulation, acquired immune responses are primed. These responses emerge at the same time as clearance of viraemia and rebound of CD4 T cells is seen. These HIV-specific responses include specific humoral or antibody responses and specific cellular (T-lymphocyte) responses.

Specific humoral or antibody responses[41]: This consist of neutralizing antibodies to the envelope proteins of the virus and other non-neutralizing antibodies to internal viral proteins such as *gag*. Specific secretory Immunoglobulin A (IgA) mucosal antibodies are also produced. Neutralizing antibodies are usually measurable by 12 weeks after infection.

In specific cellular (T-lymphocyte) responses[42], CD8 T lymphocytes or cytotoxic-lymphocytes (CTLs) form a primary component of the critical cellular immune response induced by HIV infection. CTLs are differentiated from existing CTL precursors, and express T-cell receptor molecules capable of recognizing specific viral epitopes presented in the context of human leukocyte antigen (HLA) or Major Histocompatibility Complex (MHC) molecules at the surface of infected target cells. Mature CTLs are functional 5–10 days after antigenic stimulation, recognizing, binding and then lysing the infected target cell. Virus-specific CTLs evolve faster than antibody responses and are often induced before seroconversion and before viral RNA has reached peak titres. Thus CD8 CTLs are temporally associated with the fall in viraemia during acute infection, and there is good evidence that CTLs play a major role in the control of HIV infection at this time and later in HIV disease. Evidence for strong CD8 antiviral pressure can be appreciated by the number and variety of strategies which viruses have evolved to avoid apoptosis and CTL recognition, thus prolonging the life of the virally infected cell and enabling viral replication and dissemination[43]. In addition to the lysis of infected cells, CD8 T cells can reduce viral replication by the production of soluble factors. These factors are not antigen specific but their production requires specific T-cell activation. Anti-HIV effects have been found for interferon (IFN) interleukin (IL)-10, IL-13, IL-16 and the C-C chemokines, macrophage inhibitory protein-1β, (MIP-1β) and regulated upon activation, normal T-cell expressed and secreted (RANTES). Such soluble factors may also have profound effects on other opportunistic infections including those affecting the skin. CD4 T-cell responses induced by HIV infection provide help to both HIV-specific CTLs and B cells. CD4 T-helper cells recognize antigen in the context of HLA class II molecules on the surface of antigen-presenting cells such as dendritic cells. CD4 responses to a variety of HIV proteins (including *env*, *gag* and *nef*) have been demonstrated in early disease, but immunological abnormalities in T-helper function occur very early in HIV infection, even before CD4 T-cell numbers diminish in the peripheral blood. Furthermore, advances in the understanding of HIV-1 pathogenesis reveal that mucosal tissues, primarily in the

gastrointestinal tract, are major sites for early viral replication and CD4 T-cell destruction, and this may represent the major viral reservoir[44]. Reduced proliferative capacity and diminished IL-2 production in response to stimulation by exogenous antigens (including those from HIV and other pathogens) is one of the hallmarks of HIV disease.

On recognition of their specific antigen, naïve CD4 T cells differentiate from a common (Th0) precursor into T-helper (Th)1cells, which differentially secrete interleukin 2 (IL-2), IFN-γ, transforming growth factor-β and IL-12 and can activate macrophages and 'help' CTLs, or into Th2 cells, which secrete IL-4, IL-5, IL-6 and IL-10 that can activate B cells to proliferate and differentiate into antibody-producing plasma cells. Central to the cellular immune response is the dendritic cell, which is the most potent antigen-presenting cell. However, such cells on mucosal surfaces (Langerhans' cells) may be some of the first targets in transmission; as well as transporting viral antigens across mucosal barriers and presenting them to CD4 cells, dendritic cells may themselves become infected with HIV, and their function compromised.

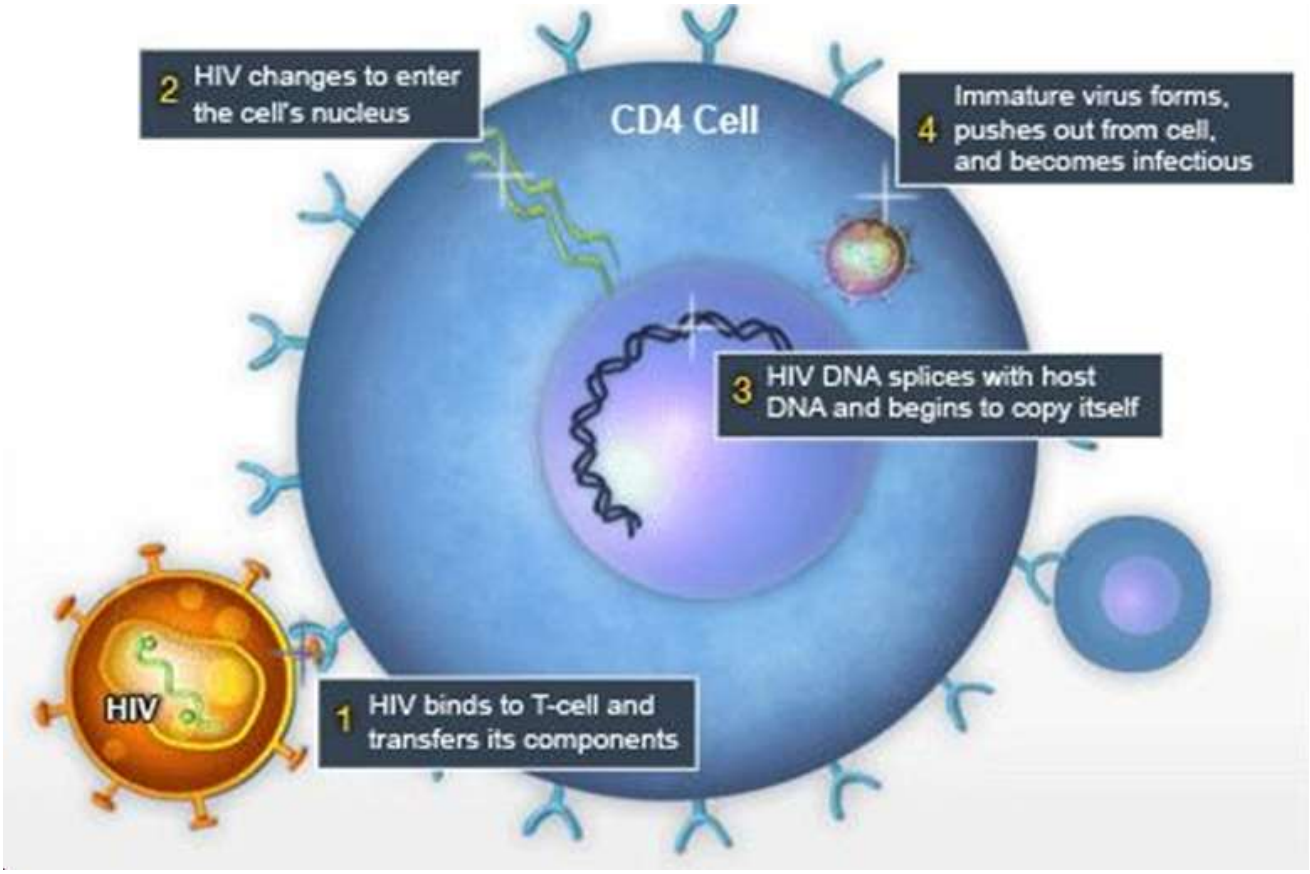


Figure 2: Schematic depiction of HIV attacking a CD4 lymphocyte

Clinical Features of HIV infection:

WHO Clinical Staging of HIV/AIDS and Case Definition:

Clinical staging and case definition of HIV for resource-constrained settings were developed by the World Health Organisation (WHO) in 1990 and revised in 2007. Staging is based on clinical findings that guide the diagnosis, evaluation, and management of HIV/AIDS, and it does not require a CD4 cell count. This staging system is used in many countries to determine eligibility for antiretroviral therapy, particularly in settings in which CD4 testing is not available. Clinical stages are categorized as 1 through 4, progressing from primary HIV infection to advanced HIV/AIDS (see Table 1 below). These stages are defined by specific clinical conditions or symptoms.

TABLE 1: WHO CLINICAL STAGING OF HIV/AIDS FOR ADULTS AND ADOLESCENTS[45]

Asymptomatic
Acute retroviral syndrome

Clinical Stage 1

Asymptomatic
Persistent generalized lymphadenopathy

Clinical Stage 2

Moderate unexplained weight loss (<10% of presumed or measured body weight)
Recurrent respiratory infections (sinusitis, tonsillitis, otitis media, and pharyngitis)
Herpes zoster
Angular cheilitis
Recurrent oral ulceration
Papular pruritic eruptions
Seborrheic dermatitis
Fungal nail infections

Clinical Stage 3

Unexplained severe weight loss (>10% of presumed or measured body weight)
Unexplained chronic diarrhea for >1 month
Unexplained persistent fever for >1 month (>37.6ºC, intermittent or constant)
Persistent oral candidiasis (thrush)
Oral hairy leukoplakia
Pulmonary tuberculosis (current)
Severe presumed bacterial infections (e.g., pneumonia, empyema, pyomyositis, bone or joint infection, meningitis, bacteremia)
Acute necrotizing ulcerative stomatitis, gingivitis, or periodontitis
Unexplained anemia (hemoglobin <8 g/dL)
Neutropenia (neutrophils <500 cells/µL)
Chronic thrombocytopenia (platelets <50,000 cells/µL)

Clinical Stage 4

HIV wasting syndrome, as defined by the CDC
Pneumocystis pneumonia
Recurrent severe bacterial pneumonia
Chronic herpes simplex infection (orolabial, genital, or anorectal site for >1 month or visceral herpes at any site)
Esophageal candidiasis (or candidiasis of trachea, bronchi, or lungs)
Extrapulmonary tuberculosis
Kaposi sarcoma
Cytomegalovirus infection (retinitis or infection of other organs)
Central nervous system toxoplasmosis
HIV encephalopathy
Cryptococcosis, extrapulmonary (including meningitis)
Disseminated nontuberculosis mycobacteria infection
Progressive multifocal leukoencephalopathy
Candida of the trachea, bronchi, or lungs
Chronic cryptosporidiosis (with diarrhea)
Chronic isosporiasis
Disseminated mycosis (e.g., histoplasmosis, coccidioidomycosis, penicilliosis)
Recurrent non_typhoidal *Salmonella* bacteremia
Lymphoma (cerebral or B-cell non-Hodgkin)
Invasive cervical carcinoma
Atypical disseminated leishmaniasis
Symptomatic HIV-associated nephropathy
Symptomatic HIV-associated cardiomyopathy
Reactivation of American trypanosomiasis (meningoencephalitis or myocarditis)

HIV infection produces a panorama of mucocutaneous manifestations, from the macular roseola – like rash seen with the acute sero-conversion syndrome to an array of severe and extensive skin lesions seen as the disease progresses[46].

Olumide et al, in a seroprevalence survey of HIV I, HIV II and Human T-Lymphotrophic Virus-1(HTLV I) among patients with skin disease and Sexually Transmitted Disease (STD) in a dermatology clinic found a rising seroprevalence to HIV I and HIV II over a 2 year period. This further highlights the increasing trend in HIV associated skin disease[47]. In another study to access the changes in the pattern of skin disease in Kaduna North Central Nigeria over a 6 year period. HIV related skin disease constituted about 4.3% of all the cases of skin diseases seen[48]. In a study by Ogunbiyi et al to assess the prevalence of skin disease in Ibadan, Nigeria it was also revealed that there is an increase in HIV associated skin disease[49].

Fungal infections are a common complication in HIV infection and include dermatophytosis, deep mycosis and yeast infections[50].

Nnoruka et al in a study to access the pattern of skin disease in HIV positive patients and their correlation with CD4 cell counts found out that dermatophytosis constituted 24.3% of all cases of skin diseases seen in this group of patients. Four hundred and seventy-seven HIV sero-positive patients were used in the study. The mean CD4 cell count of patients with dermatophytosis was 437.3±177 cells/µl[51].

In a study in India by Shobhana et al[52], 410, HIV seropositive patients were screened for skin disease. It was found that 40% had mucocutaneous involvement at presentation. Mean age of the study population was 29 years and male to female ratio was 2.5:1. The common mucocutaneous morbidities detected include oral candidiasis (36%), dermatophytosis and gingivitis (13% each), herpes zoster (6%), herpes simplex and scabies (5% each). A striking feature noted in 36% of the males was straightening of the hairs. Genital herpes was the commonest genital ulcer disease. Lesions associated with a declining immunity include oral candidiasis, oral hairy leukoplakia and herpes zoster with median CD4 cell counts of 94, 62 and 192 cells/µl respectively. It was concluded from his study that a recognition of the protean mucocutaneous diseases in HIV/AIDS helps in earlier diagnosis of HIV and serve as a measure of the immune status of the individual.

Dermatophytoses are common cutaneous fungal infections in HIV infected patients and can occur at any stage of the illness, and show clinical variations[50,53]. In the immunocompetent host, various risk factors have been identified for the acquisition of dermatophytosis. These include poverty[54,55], close contact with animals and soil[55-57]. (especially for geophillic and zoophillic dermatophytes). Other risk factors include male sex[57], poor sanitary conditions[58,55]. Use of poorly sterilised barbing equipment have also been identified as an important risk factor for transmission of tinea capitis in this environment and in one study was responsible for the high prevalence of tinea capitis in the community[59]. An important risk factor for tinea pedis infection is the frequent wearing of occlusive footwears. The warm humid environment surrounding the feet provides a conducive environment for the growth of dermatophytes for this group of persons[60].

Invasion of the skin by dermatophytes begins with adherence of arthroconidia to the keratinocytes, followed by penetration through and between cells and development of a host response[61].

Adherence of dermatophytes to keratinocytes takes about 2 hours to complete, during which germination and penetration of the keratinocytes occur. Hyphal prolongation follows shortly afterwards and proceeds radially[61,62]. The limitation of invasion of dermatophytes to the stratum corneum is due to the presence of a fungistatic factor in the tissue fluids and serum[63,64].

Dermatophytes produce a variety of proteolytic enzymes which play a role in the invasion of the stratum corneum, hair and nails[65-68]. There is some heterogeneity in substrate preference of dermatophytes. While all dermatophytes invade stratum corneum, different species vary widely in their ability to invade nail and hair.

Trichophyton rubrum rarely invades hair but frequently invades nail. *Epidermophyton. floccosum* never invades hair and only occasionally invades nail[69].

The commonest clinical presentations of dermatophytosis in immunocompetent persons are tinea corporis and tinea capitis[69].

Various studies have demonstrated unusual clinical presentations and higher prevalence rates of dermatophytosis among HIV seropositive patients. In one study by Goodman et al, it was 4 times higher in HIV positive patients[59]. In that study, 117 HIV seropositive patients were recruited. Dermatophytosis was seen in 30% of the patients. Other common skin diseases seen were: candidiasis (47%), seborrheic dermatitis (32%), acquired icthyosis or xerosis (30%) and herpes simplex infection (22%).

Kaviarasan et al[70], studied the prevalence and clinical variations of dermatophytosis in HIV infected persons . A total of 185 HIV infected persons were screened and a diagnosis of dermatophytosis was made in 41 cases. Prevalence of dermatophytosis was 22.2%. Male: female ratio 3:1, mean age of the patients was 30.7 years. Tinea Corporis was the commonest dermatophyte infection (53.7% of cases) followed by tinea cruris (49.9%), tinea pedis (17.1%), tinea facei (14.7%). Tinea manuun was noted in 0.5% while 6% of the cases had tinea unguium.

Out of the 22 patients with tinea coporis 19 (8.36%) were staged as full blown AIDS - WHO Clinical Stage IV. Ten (45.45%) of them presented with the anergic form of tinea corporis, proximal subungual onychomycosis, thought to be pathognomonic of AIDS was seen in 3 cases.

In another study by Ekong et al (utilising 4 centres in Nigeria)[71]. to evaluate the types and clinical presentations of superficial and deep fungal infections

in HIV/AIDS patients, 288 patients were screened, aged 37±14.5 years. Mean baseline CD4 cell count was 450 cells/µl. It was found that 69% of the patients had dermatophyte infections 35% with tinea corporis, 28% with tinea pedis. Patient with low CD4 count had more severe fungal infections while those on Anti-retroviral (ARV) drugs had milder diseases.

Dermatophytosis in HIV infection can present as atypical lesions due to immunodeficiency. Skin lesions may be disseminated, facial tinea mimicking seborrheic dermatitis, palmoplantar lesions with significant hyperkeratosis, lesions practically without erythema and with a prevalence of desquamation simulating xerosis[72,73].

In HIV dermatophytosis can also present as extensive and deep lesions[74]. A case of an HIV positive 23 year old male was reported with a CD4 cell count of 335 cells/µl with multiple large erythematous circinate and pustular plaques on his abdomen, back, arms, and legs. *Trichophyton mentagrophyte* was isolated and biopsy showed suppurative deep dermatophytosis and folliculitis. The patient responded to itraconazole therapy for 2 weeks[74].

In another report by Kwon et al[75], a 44 year old HIV positive man with *Trichophyton rubrum* infection presenting with widespread invasive multiple tumor like eruptions. The patient responded to oral terbinafine therapy with complete remission of his lesions.

Raquel et al also presented a case of an HIV positive 33 year old Brazilian woman with an exacerbated inflammatory response to *Trichophyton rubrum* infection of her left arm. This patient was treated with oral fluconazole which she responded to[76].

Uncommon dermatophytes such as geophillic and zoophillic organisms have also been detected as causative agents of tinea corporis in HIV infected patients. In a study by Porro et al, tinea corporis infection with atypical presentation caused by *Microsporum gypseum*, a geophillic dermatophyte were reported in 2 patients with AIDS. *Microsporum gypseum,* is an unusual causative organism for human dermatophytosis[77].

In another report by Nenoft et al, an HIV positive 15 year old boy from Uganda was found to have several dry and hyperkeratotic lesions of the forearms and left hand with circinate, erythematous and scaly morphology. *Microsporum gypseum* was also isolated in this patient as a causative organism of this tinea coporis[78].

Nunman et al also reported another similar case of extensive Tinea corporis infection caused by *Microsporum gypseum* in a 36 year old HIV positive woman. The lesions were said to be generalised, psoriasiform and refractory to treatment with ketoconazole and itraconazole[79].

Zoophilic organisms have also been reported as causative agents of tinea corporis in HIV infected patients. Menon et al reported a case of an HIV infected patient who presented with a chronic non inflammatory non pustular extensive infection caused by the zoophillic dermatophyte *Trichophyton verrucossum*[80].

Lowinger Seoane et al also described another case of disseminated cutaneous dematophytosis caused by another zoophillic organism. *Trichophyton mentagrophytes* and *Microsporum canis* in an HIV infected patient[81].

Tinea capitis is rare in adults. This may be due to the fact that the quantity of fungistatic saturated fatty acids in sebum increases in puberty[82].

It has also been found that dermatophyte colonisation of hair disappears at puberty, this may result from the colonisation by *pityrosporum orbiculare* interfering with dermatophyte contamination, and the thicker calibre of adult hair may protect against dermatophyte invasion[82,83].

Tinea capitis in adults generally occurs in patients who are immuncompromised and those infected with HIV[84]. It is uncommon in immunocompetent adults and when it occurs the clinical features may be atypical and this may delay the diagnosis[84]. It may resemble bacterial folliculitis, folliculitis decalvans, dissecting cellulitis or the scarring related to discoid lupus erythematosus[84].

However, tinea capitis in men even if HIV positive is uncommon. A few cases of tinea capitis have been described

in HIV infected patients. Lateur et al presented 2 cases of adult black African males with HIV infection with tinea capitis[85].

In another report, by Bournerias et al[86], 2 HIV sero-positive men presented with unusual *M.icrosporum canis* infection. Both had tinea capitis presenting as alopecia in one and scaling of the scalp in the other. One also had tinea unguium caused by *Microsporum canis*. Both were treated with oral itraconazole for several months and only one was cured.

One of the earliest superficial fungal infections to emerge in HIV infected persons is onychomycosis. These group of patients are more likely to develop it when the CD4 lymphocyte level falls to approximately 400 cells/µl[87].

In the HIV positive patient prevalence of onychomycosis ranges from 11% to 67%[88]. It often starts as a proximal white subungual onychomycosis (PWSO) and quickly spreads to the other nails of fingers and toes[88,89,90]. If left untreated it can lead to systemic infection to which the immunocompromised host cannot respond.

Proximal subungual onychomycosis is said to be an indicator of HIV disease[91,92]. Although it can also occur in other immunocompromised patients.

Another common presentation of onychomycosis in HIV infected persons is the one hand, 2 feet tinea (that is affectation of one hand and two feet) which is relatively uncommon in the general population[91,92].

In a study of onychomycosis in 62 HIV infected persons, Dompmatin et al found out that the most frequent aetiologic agents were dermatophytes (in 58%). The rest are caused by *candida albicans* and *Pityrosporum. Ovale*[93].

Proximal white subungual onychomycosis in HIV patients is generally caused by *Trichophyton rubrum*[92,93]. However, there are rare reports of lesions caused by other species such as *Trichophyton mentagrophytes, Epidermophyton floccosum* and *Microsporum gypseum*[93]. In immunocompetent

persons, the commonest organism is *Trichophyton mentagrophytes*[92].

Other dermatophyte infections seen in HIV infection (although less frequently) are tinea cruris, tinea facei, tinea pedis, and tinea manuum[70].

From the foregoing it can be seen that dermatophyte infections are quite common in HIV infections and may present in atypical clinical forms. However, a few authors have postulated that these skin diseases may not occur any more frequently in HIV infected persons than in the normal population[94,95].

In a study by Torssander J. et al[26], the prevalence of dermatophytosis in HIV infected persons was 37.3% as compared to 31.8% in the HIV negative population. The difference was not statistically significant. More studies need to be done to ascertain this.

AIMS AND OBJECTIVES

(1) To evaluate the prevalence of dermatophytosis in patients with HIV infection at the university of Port Harcourt teaching hospital, Port Harcourt.
(2) To assess the pattern and clinical variations of dermatophytosis seen in HIV infected patients.

MATERIALS AND METHOD

Study Area/Population:

This is a cross sectional study assessing the prevalence and pattern of dermatophytosis among patients with HIV infection in the University of Port - Harcourt Teaching Hospital, Port Harcourt.

One hundred and seventy-three patients who presented with newly diagnosed HIV infection in the wards and at the Anti-retroviral clinic of the University of Port Harcourt Teaching Hospital, Port Harcourt were recruited into the study over a period of seven months and their consent obtained to participate in the study.

The University of Port-Harcourt Teaching Hospital, is located at Alakahia in the outskirts of Port-Harcourt city. It is a major referral centre in the South-south region of the country which comprises Bayelsa, Edo, Delta, Cross River, Akwa-Ibom and Rivers states. Between 10 and 20 new cases of HIV are seen weekly. The adult and paediatric ARV clinics and medical wards are located within the teaching hospital premises in Alakahia.

Sample Size Determination:

The sample size was obtained using the formula below:

$$n_1 = \frac{2 \times Z^2 \times p \times q}{d^2}$$

$$n_1 = \frac{2Z^2pq}{d^2}$$

Where n_1 = sample size

P = prevalence rate of dermatophytosis in Nigeria

6.1% = 0.061[3]

q = (1 – p) = 1 – 0.061 = 0.939
z = standard error deviate, set at 1.96, corresponding to 95% confidence level
d = difference obtained = 0.05

$$\text{Sample size is } \frac{2 \times (1.96)^2 \times (0.061)(0.939)}{(0.05)^2} = 173$$

Criteria for inclusion was:

(1) Patients who are HIV positive and not yet on anti-retroviral therapy.
(2) The patients that gave their consent.

Exclusion criteria:

(1) Patients that did not give their consent
(2) Patients who are HIV negative
(3) Diabetic patients.

Control population: One hundred and seventy-three patients that were screened for HIV infection and found to be seronegative were used as control. The cases and control were matched for age and sex.
The subjects also had their CD4 cell counts assayed and documented. A fasting blood sugar was done to exclude diabetes.

Laboratory Methodology:

Hiv Screening:

The HIV I & II rapid test strip is a qualitative membrane based immunoassay for the detection of antibodies HIV I & II in whole blood, plasma or serum. The membrane is pre-coated with recombinant HIV antigens. During testing, whole blood, serum or plasma react with HIV antigen coated particles in the test strip. The mixture then migrates upwards on the membrane chromatographically by capillary action and reacts with recombinant HIV antigen on the membrane in the test line region. If the specimen contains antibodies to HIV I & II, a coloured line will appear in the test line region indicating a positive result. If the specimen does not contain HIV I and/or II antibodies, a coloured line will not appear in the test region indicating a negative result.

Five mls of whole blood were collected from the subjects via vene-puncture in a syringe.

The blood was allowed to clot and the serum collected and tested using the Rapid HIV test strip

Materials provided in the test strip kit include:

Test strips
Disposable specimen droppers
Buffer
Test cards
Package inserts

Materials that are required but not provided include – specimen collection containers
Lancets
Centrifuge
Timer

Procedure:

A pre-test counselling was done after obtaining the patient's consent. Thereafter, the test is performed on their specimens.

The test strip, specimen, buffer and/or controls were allowed to equilibrate to room temperature. (15–30^{0}C) prior to testing.

The test strip were removed from the foil pouch and used as soon as possible.

The tape was peeled off from the test card and the test strip stuck in the middle of the test card.

The dropper was used to transfer one drop of serum to the 'specimen pad' of the test strip and then 2 drops of buffer added and the timer started.

The result was read after 15 minutes when the red line(s) is expected to appear if the result is positive – One in the control region (C) and one in the test region (T).

If the result is negative only one red line appears in the control region (C). None appears in the test region (T).

The result is invalid if the control line fails to appear. This may result from insufficient specimen volume or wrong procedural techniques.

Determine® HIV ELISA kit was used for this screening. The specimens that were positive were were retested for confirmation with a different kit (Immunocomb®). A post-test counselling was done thereafter. A high level of confidentiality was maintained throughout the testing period.

CD4 Cell Determination:

The CD4 cell count of the subjects was determined using the Partec flow cytometry and recorded as CD4 cells per microliter of blood. The reagents and consumables were used according to the manufacturer's instructions. The CyFlow Counter uses a 'no lyse, no wash' procedure for CD4 counting.

Fifty microlitres of EDTA-anticoagulated blood were added to 10 µl of monoclonal antibodies. After 15 min of incubation, 1ml of no lyse dilution buffer was added and the sample tube was attached to the CyFlow Counter for automated counting. Results were available in 2 min and were expressed in a histogram (CD4+ cells/µl).

Mycology Studies:

All the subjects that were recruited into the study were interviewed using a questionnaire (ADDENDUM I), from which bio data, social and clinical history were collected. History of contact with persons with skin disease was also obtained and their CD4 counts documented. The patients were then examined physically by me for the presence of skin, hair or nail lesions with particular reference to the site of involvement and morphology of skin lesions. The various clinical diagnosis were supplemented with relevant laboratory investigations. Patients with clinical diagnosis of any form of dermatophytosis had specimen obtained and sent for mycological studies.

Materials:

1. Paper for specimen collection.
2. Blunt scalpels.
3. Face masks.
4. Disposable gloves.
5. Test tubes.
6. Cotton wool.
7. Light microscope.
8. Microscopes slides and cover slips.
9. Lactophenol cotton blue.
10. 70% alcohol.
11. Potassium hydroxide (10% and 20%).
12. Saboraoud dextrose agar.
13. Chloramphenicol.

Procedure:

All suspected lesions were first cleaned with methylated spirit. Skin specimen were obtained by scraping with a sterile scalpel blade from the advancing edge of the lesions .

Strands of hair from scalp lesions were epilated with artery forceps and submitted with the root end.

Nail specimens were obtained by clipping affected nails as proximally as possible through the entire thickness of the nails using a pincer type nail clipper.

All specimens were collected into folded paper and sent to the laboratory for mycological studies.

Specimens of skin, hair and nail were then digested in a solution of potassium hydroxide on a microscope slide. A solution of 10% potassium hydroxide (KOH) was used for skin, while 20% KOH was used for hair and nails. This made the tissue layers thin enough to enable the hyphae or other fungal elements to be seen. A cover slip was placed on the specimen and it was allowed to stand for 30 minutes. Nail clippings were allowed to stand for 2 to 3 hours.

Thereafter the specimens were examined under direct microscopy to identify the morphology of the fungi present. They were viewed under low power initially and the entire specimen carefully scanned after which any suspicious was viewed under high power.

Some portions of the specimen were inoculated into a slope of 5ml saboraoud's dextrose agar (SDA) in a test-tube plugged with non-absorbent cotton wool, held in a slant and incubated at room temperature (26^{o}C). Chloramphenicol was incorporated into the specimen to prevent growth of bacteria. The test-tube was labelled with patients' identification number, date and nature of specimen.

The specimen was examined weekly for up to one month. When sufficient colony growth appeared, 1 – 2 drops of lactophenol cotton-blue was put on a slide, and a sterile needle pick of the colony was mounted on the slide and examined under the microscope for the characteristic types of vegetative structure, asexual spores and hyphae present.

Data Analysis:

The data obtained from the results were analysed using appropriate statistical analysis through the statistical programme for social sciences (SPSS) version I8 package.
Statistical significance was documented at $p<0.05$.

RESULTS

Study Population:

A total of one hundred and seventy three HIV seropositive patients and one hundred and seventy three seronegative controls were recruited for this study.

Demographic Data:

All of the subjects are Nigerians and resident in Rivers state.

The cases and controls were matched for age and sex.

One hundred and five of the cases were females while 68 were males. Male to female ratio = 1:1.54, While male to female ratio for the control is 1:1.74 .This difference is not statistically significant ($p>0.05$).

The ages of the cases ranged from 2-75 years with a mean age of 33.3036 ± 12.61914. For the control the age range is from 2-77 years with a mean age of 35.4404 ± 14.02180. This difference is not statistically significant ($p>0.05$). Other details are as shown in the table below.

TABLE 2: AGE STRATIFICATION OF CASES AND CONTROLS

AGE GROUP(Years)	CASES (%)	CONTROLS(%)	TOTAL POPULATION
	2.9%)		
2-5	5(2.9%)	4(2.3%)	9(2.6%)
6-10		5(2.9%)	10(2.9%)
11-15	2(1.1%)	3(1.7%)	5(1.4%)
16-20	2(1.1%)	3(1.7%)	5(1.4%)
21-25	30(17.0%)	31(17.9%)	61(17.6%)
26-30	33(19.0%)	30(17.0%)	63(18.2%)
31-35	30(17.0%)	27(15.6%)	57(16.4%)
36-40	24(13.8%)	25(14.4%)	49(14.1%)
41-45	17(9.8%)	18(10.4%)	35(10.1%)
46-50	14(8.0%)	16(9.2%)	30(8.6%)
51-55	3(1.7%)	4(2.3%)	7(2.0%)
56-60	3(1.7%)	3(1.7%)	6(1.7%)
61-65	1(0.55%)	1(0.55%)	2(0.5%)
66-70	1(0.55%)	1(0.55%)	2(0.5%)
>71	3(1.7%)	2(1.1%)	5(1.4%)
TOTAL	**173(100%)**	**173(100%)**	**346(100%)**

TABLE 3: DEMOGRAPHIC DATA OF HIV SERO-POSITIVE CASES AND THE HIV SERO-NEGATIVE CONTROLS

VARIABLE	HIV SERO-POSITIVE CASES(%)	HIV SERO-NEGATIVE CONTROLS(%)
Gender	68(39.3%)	63(36.4%)
Male	105(60.6%)	110(61.8%)
Female		
Total	173(100%)	173(100%)
Marital status		
Ever married	104(60.1%)	87(50.2%)
Never married	69(39.8%)	86(49.7%)
Total	173(100%)	173(100%)
Occupation		
Civil servant	20(11.3%)	20(11.5%)
Student	12(7.1%)	18(10.4%)
Self-employed	16(9.5%)	24(13.8%)
Trader	26(14.88%)	16(9.2%)
Armed forces	6(3.57%)	2(1.1%)
Unemployed	19(10.7%)	22(12.7%)
Others	74(42.7%)	71(41%)
Total	173(100%)	173(100%)
Educational status		
None		
Primary	13(7.5%)	12(6.9%)
Secondary	29(16.6%)	18(10.4%)
Tertiary	72(41.0%)	50(28.9%)
	59(33.9%)	93(53.7%)
Total	**173(100%)**	**173(100%)**

Cd4 Cell Count of the Subjects:

The mean CD4 cell count of the cases is 355.3.The range is between 21 and 1,260. The meanCD4 cell count for the control is 865.3 with a range of 778-1000.This difference is statistically significant($p<0.05$) . Forty-one percent of the cases have a CD4cell count between 200-500 cells/µl. Other details are as in the table below.

TABLE 4: CD4 CELL COUNT GROUPING ACCORDING TO SERO-STATUS

Cd4 Cell Count(Cells/µl)	Cases(%)	Controls(%)	Total(%)
<50	8(4.6%)	0	8(4.6%)
50-200	50(28.9%)	0	50(28.9%)
200-500	72(41.6%)	0	72(41.6%)
>500	43(24.8%)	173(100%)	216(62.4%)
Total	**173(100%)**	**173(100%)**	**346(100%)**

Medical History of the Cases And Controls:

Sixty three (36.9%) of the cases had a prior history of use of bleaching cosmetics compared to 41 (23.81%) of the control. This is not statistically significant(p.0.05). Hydroquinone is the most commonly abused bleaching agent as shown in the table below.

Five (2.8%) of the cases had a history of close contact with somebody with a skin disease compared to 12 (6.9%) of the control.

TABLE 5: MEDICAL HISTORY OF CONTROLS AND CASES

VARIABLE	CASES (%)	CONTROLS(%)	TOTAL(%)
History of use of bleaching cosmetics	63(36.3%)	41(23.8%)	104(30.0%)
Type of bleaching cosmetics			
Steroids	11(6.5%)	12(7.1%)	23(5.2%)
Hydroquinone	53(30.3%)	37(21.4%)	90(26.0%)
History of contact with a person with skin disease	5(2.8%)	12(6.9%)	17(4.9%)

Clinical Findings In The Cases And Controls:

Sixty of the cases (34.6%) had various skin lesions on physical examination with a mean duration of symptoms of about 71 weeks compared to 12 (6.9%) of the control whose mean duration of symptoms was about 8 weeks This is statistically significant ($p<0.05$).

Twenty-eight of the cases with various skin lesions (16.0%) had sought various forms of treatment compared to only 5 (2.8%) of the control.

TABLE 6: CLINICAL FINDINGS IN THE CASES AND CONTROLS

VARIABLE	CASES	CONTROLS
Presence of skin lesions	60(34.6%)	12(6.9%)
Mean duration of symptoms (in weeks)	71.47±17.69	8.22 ± 3.11
Previous treatment	28(16.01%)	5(5.9%)

HIV Staging:

The HIV-seropositive patients were staged using the WHO clinical staging as follows: 43(24.8%) patients had stage 1 disease, 72(41.6%) had stage 2 disease, 50(28.9%) had stage 3 disease while 8(4. 6%) had stage 4 disease.

Prevalence of Various Skin Lesions Among The Cases And Control Groups:

.

The commonest skin lesion seen in the cases was pruritic papular eruption of HIV followed by dermatophytosis-24(13.8%) and 12 (6.9%) respectively. The commonest skin lesions in the control group were dermatophytosis, acne vulgaris and furunculosis. There is a significantly higher prevalence of dermatophytosis in the cases compared to the control (p,0.05) Other details are in table 7 below

TABLE 7: PREVALENCE OF VARIOUS SKIN LESIONS IN THE HIV POSITIVE CASES AND CONTROLS

TYPE OF SKIN LESION	CASES(%)	CONTROLS(%)	TOTAL POPULATION(%)
Pruritic papular eruption	24(13.8%)	_	24(6.9%%)
Dermatophytosis	12(6.9%)	5(2.8%)	17(4.9%))
Acne vulgaris	4(2.3%)	4(2.3%)	8(2.3%)
Kaposi sarcoma	2(1.15%)	_	2(0.57%)
Herpes genitalis	3(1.7%)	_	3(0.86%)
Furunculosis	3(1.7%)	2(1.15%)	5(1.4%)
Herpes zoster	3(1.7%)	-	3(0.86%)
Warts	2(1.15%)	-	2(0.5%)
Molluscum contagiosum	2(1.15%)	-	2(0.5%)
Fixed drug eruption	1(0.5%)	-	1(0.25%)
Tinea versicolor	1(0.5%)	2(1.15%)	3(0.86%)
Keloids	1(0.5%)	-	1(0.25%)
Urticaria	1(0.5%)	-	1(0.25%)
Epidermodysplasia verruciformis	1(0.5%)	-	1(0.25%)
Total	**60(34.6%)**	**12(6.9%)**	**72(20.8%)**

Cd4 Cell Counts of the HIV Seropositive Cases With and Without Skin Lesions

The mean CD4 cell count of the HIV seropositive cases with skin lesions is 224.86 compared with those without skin lesions which is 404.72.. Patients with herpes genitalis, molluscum contagiosum and epidermodysplasia verruciformis have the lowest CD4 cell counts ((below 200). Other details are as in the table below.

TABLE 8: MEAN CD4 CELL COUNTS OF THE HIV POSITIVE CASES WITH VARIOUS SKIN LESIONS

TYPE OF SKIN LESION	NO. OF CASES	MEAN CD4 CELL COUNT
Pruritic papular eruption	24(13.8%)	199.54
Dermatophytosis	12(6.9%)	226.2
Acne vulgaris	4(2.3%)	370.75
Kaposi sarcoma	2(1.15%)	365.5
Furunculosis	3(1.7%)	317.67
Herpes zoster	3(1.7%)	203
Warts	2(1.15%)	272.5
Herpes genitalis	3(1.7%)	88
Urticaria	1(0.5%)	567
Keloids	1(0.5%)	400
Fixed drug eruption	1(0.5%)	526
Tinea versicolor	1(0.5%)	611
Molluscum contagiosum	2(1.15%)	113
Epidermodysplasia verruciformis	1(0.5%)	51
Total	**60(34.6%)**	**224.86**

TABLE 9: DEMOGRAPHIC CHARACTERISTICS OF CASES WITH DERMATOPHYTOSIS

VARIABLE	TOTAL NO WITH TINEA CORPORIS	TOTAL NO WITH TINEA UNGUIUM	TOTAL WITH TINEA MANUUM	TOTAL WITH TINEA PEDIS	TOTAL PER AGE GROUP
Age					
2-20	0	2	0	0	2
21-40	2	0	1	0	3
41-60	2	0	1	2	5
>61	2	0	0	0	2
Total	**6(50%)**	**2(16.6%)**	**2(16.6%)**	**2(16.6%)**	**12(100%)**
Gender					
male	4	0	2	2	8
Female	2	2	0	0	4
Total	**6(50%)**	**2(16.6%)**	**2(16.6%)**	**2(16.6%)**	**12(100%)**

TABLE10: DEMOGRAPHIC CHARACTERISTICS OF THE CONTROL WITH DERMATOPHYTOSIS

VARIABLE	TOTAL NO WITH TINEA CORPORIS	TOTAL NO WITH TINEA UNGUIUM	TOTAL WITH TINEA MANUUM	TOTAL WITH TINEA CAPITIS	TOTAL PER AGE GROUP
Age					
2-20	1	0	0	1	1
21-40	0	0	0	0	10
41-60	1	1	0	0	5
>61	0	0	1	0	0
Total	**2(40%)**	**1(20%)**	**1(20%)**	**1(20%)**	**5(100%)**
Gender					
Female	2	1	1	0	4(80%)
Male	0	0	0	1	1(20%)
Total	**2(40%)**	**1(20%)**	**1(6.25%)**	**1(20%)**	**5(100%)**

TABLE 11: DERMATOPHYTOSIS AMONG THE CASES STRATIFIED BY CD4 CELL COUNT

CD4 COUNT	TOTAL NO OF TINEA CORPORIS N(%)	TOTAL NO OF TINEA UNGUIUM N(%)	TOTAL NO OF TINEA MANUUM N(%)	TOTAL NO OF TINEA PEDIS N(%)	TOTAL WITH DERMATOPHYTOSIS(%)
0-200	4(33.3%)	0	1(8.3%)	0	5(41.6%)
201-400	2(16.6%)	2(16.6%)	1(8.3%)	1(8.3%)	6(50%)
401-600	0	0	0	0	0
601-800	0	0	0	1(8.3%)	1(8.3%)
801-1000	0	0	0	0	0
1001-1200	0	0	0	0	0
TOTAL	**6(50%)**	**2(16.6%)**	**2(16.6%)**	**2(16.6%)**	**12(100%)**

PLATES 1-5: CLINICAL IMAGES OF VARIOUS TYPES OF DERMATOPHYTOSIS SEEN AMONG THE SUBJECTS

PLATE 1: Tinea Unguium in an HIV sero-positive woman.

PLATE 2: Tinea manuum in a young HIV seropositive male.

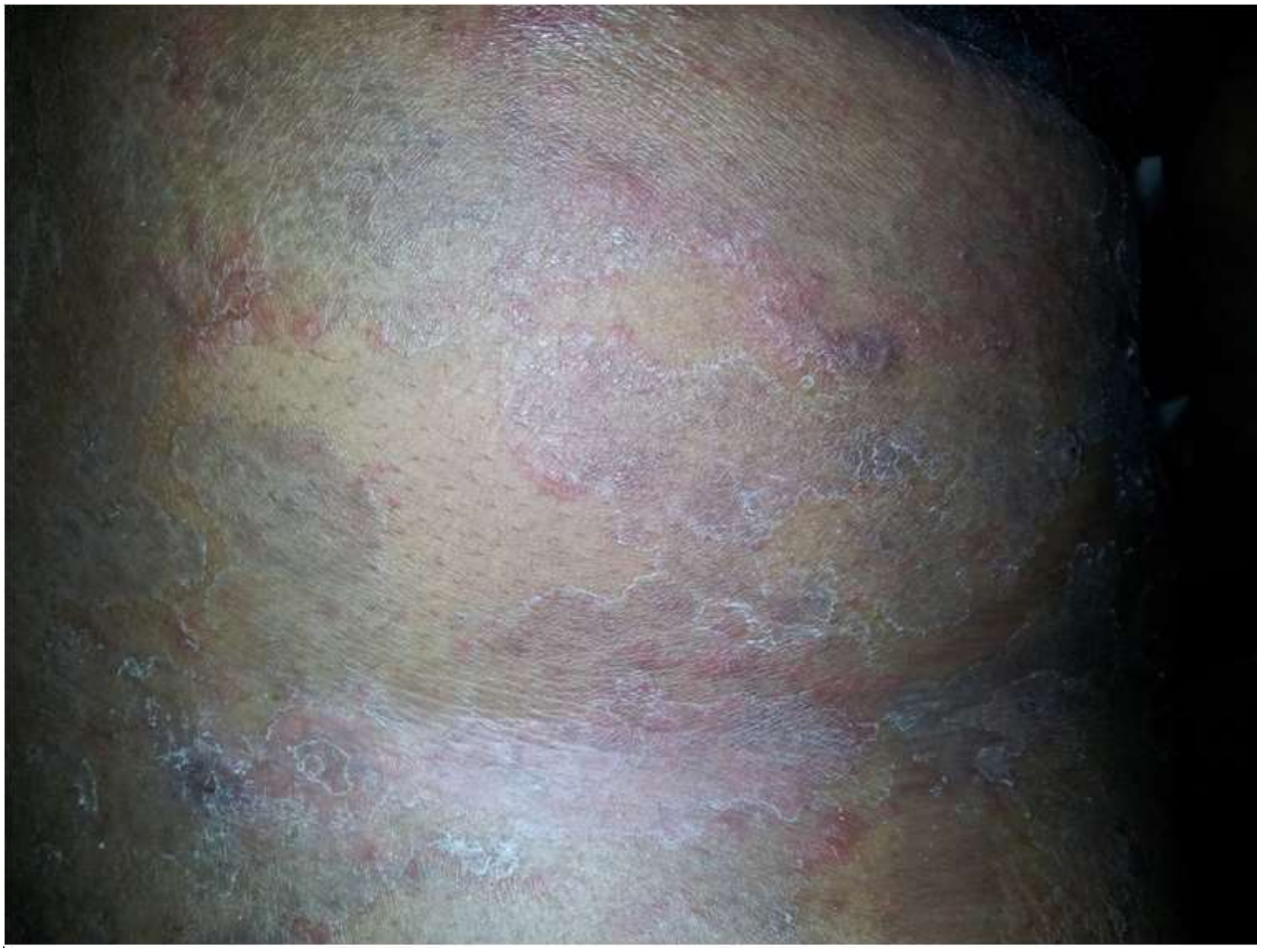


PLATE 4: Tinea pedis in an HIV seropositive patient

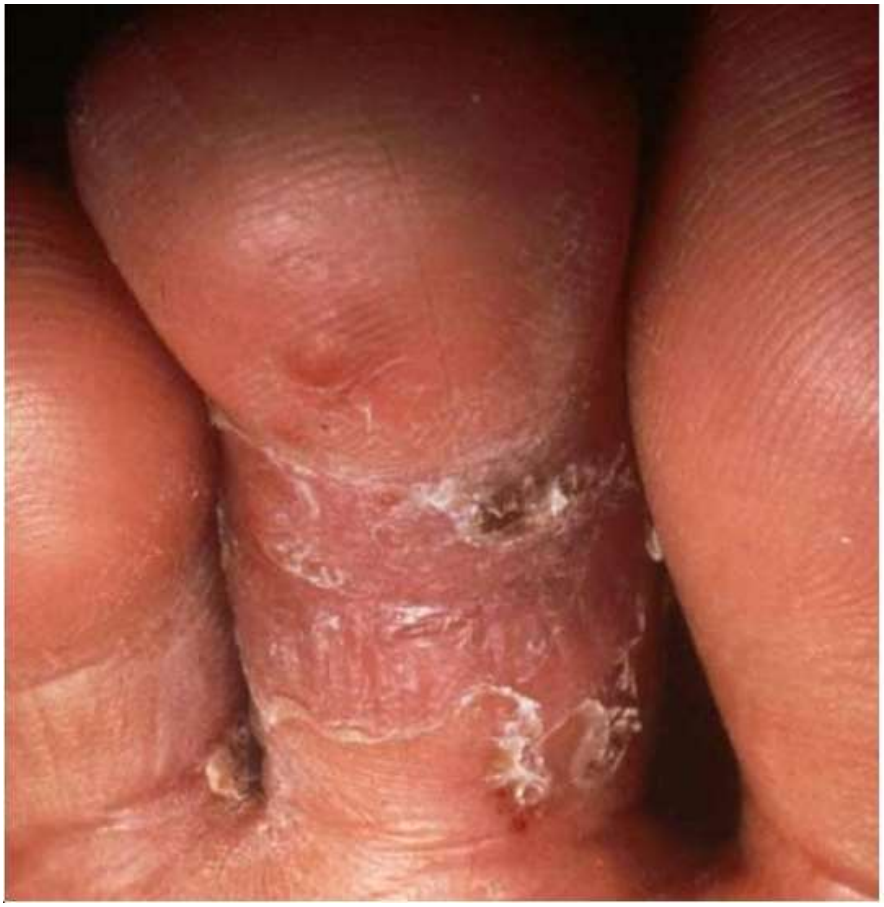
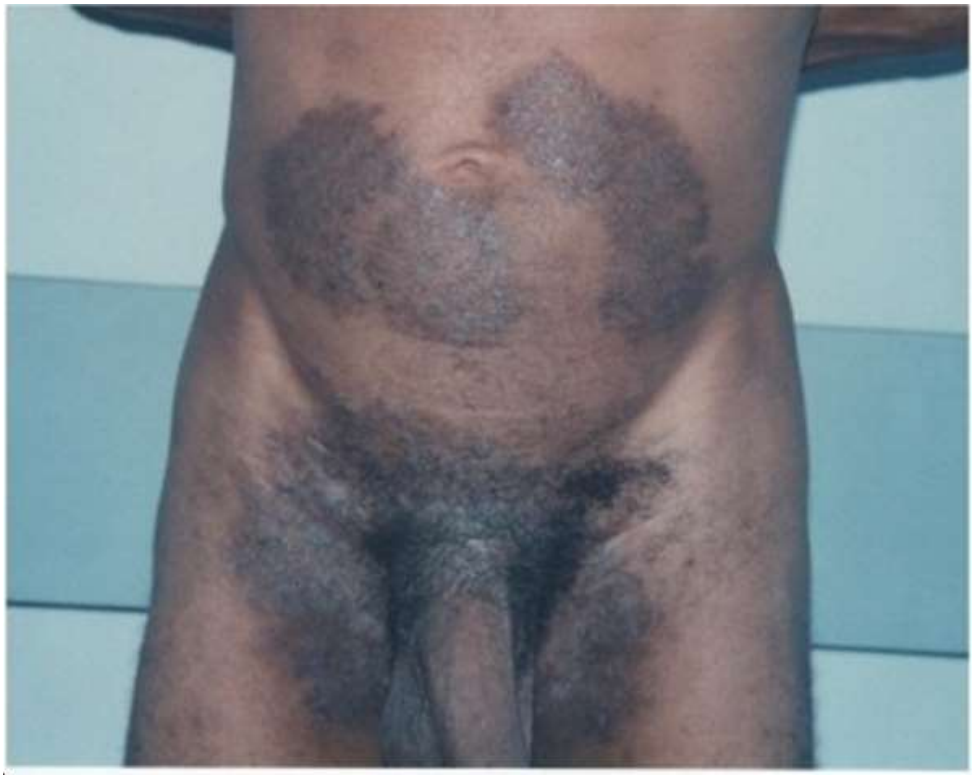


PLATE 5: Tinea corporis in a young HIV seropositive male

MYCOLOGY:

(1) Potassium Hydroxide Wet Mount:

Eleven of the specimens from the HIV sero-positive cases (91.6%) were positive for fungal hyphae while 4 of the control group(80%) were positive for fungal hyphae.



(2) Culture:

Five of the specimens from the HIV sero-positive cases (41.6%) grew dermatophytes.Out of this number, 2 (16.6%) were *Trichophyton mentagrophyte;* 2(16.6%) were *Trichophyton soudanenses* and 1(8.3%) was *Microsporum auodunii.* Two of the specimens (16.6%) showed no growth, while 5(41.6%) specimens grew non-dermatophytic fungi such as *Aspergillus fumigatus, Aspergillus niger,* and *Penicillium chrysogenum.*

For the control, 2 (40%) of the specimen showed no significant growth, while 3 of the specimens grew non-dermatophytic fungi (*Aspergillus niger, Aspergillus fumigatus and Aspergillus flavus).* Other details are in the tables below.

TABLE 12: MYCOLOGY RESULTS OF HIV POSITIVE CASES WITH CLINICALLY DIAGNOSED DERMATOPHYTOSIS

SPECIES	FREQUENCY	PERCENTAGE
Microsporum auodonii	1	8.3%
Trichophyton soudanenses	2	16.6%
Trichophyton mentagrophyte	2	16.6%
Other species (Aspergillus,penicillium)	5	41.6%
No significant growth	2	16.6%
Total	**12**	**100%**

TABLE 13: MYCOLOGY RESULTS OF THE CONTROLS WITH CLINICALLY DIAGNOSED DERMATOPHYTOSIS

SPECIES	FREQUENCY	PERCENTAGE
Non-dermatophytic fungi	3	60%
No significant growth	2	40%
TOTAL	5	100%

PLATES 7-9: Dermatophytes cultured from the specimens collected from the subjects

PLATE 7: Culture of *Microsporum audounii* in Saboraoud's dextrose agar (SDA).

PLATE 8: Culture of *Trichophyton mentagrophyte* in SDA.

PLATE 9: Culture of *Trichophyton soudanense* in SDA.

DISCUSSION

This study was carried out to assess the prevalence and pattern of the various types of dermatophytosis among HIV infected patients as compared to apparently healthy seronegative controls.

Various skin conditions are associated with HIV infection. Epidemiologic studies have shown that almost all persons with HIV infection will have skin disorders at some point during their disease.[96] Skin disorders commonly encountered in HIV-infected patients may be the first manifestation of HIV disease. Up to 90% of HIV-infected persons suffer from skin diseases during the course their of illness[97]. In a recent cross-sectional study of 186 HIV positive patients, 175 (94%) suffered from one or more cutaneous disorders[98]. The most common skin disorder identified was fungal infection, followed by eczema and seborrhoeic dermatitis. The spectrum of skin disorders depends on: (a) immunologic stage, as reflected by CD4 count (b) concurrent use of Highly Active Anti-Retroviral Therapy (HAART) (c) pattern of endemic infections.

In general, declining immunity is associated with increased number and severity of skin disorders[99]. Skin lesions are more likely to have unusual appearances in advanced HIV infection.

In this study the prevalence of several skin diseases was found to be significantly higher in the HIV seropositive group compared to the seronegative controls (34.6% vs 6.9%).The mean duration of the lesions was also longer for the cases compared to the control group (71 weeks vs 8weeks). In addition, the mean CD4 cell count of the HIV seropositive cases with skin disease was significantly lower than those without any skin disease thus indicating the importance of immunosuppression in the development of skin disease in such patients. Dermatophytosis is the second commonest skin lesion in the HIV seropositive group (next to pruritic papular eruption of HIV). It has a significantly higher prevalence among the cases when compared to the control group -6.9% vs2.8%, ($p<0.05$). The prevalence of dermatophytosis in the HIV positive cases used in this study is 6.9%. Previous studies done in the past revealed prevalence rates between 6.06% and 30%[50-52,70,100-102]. The relatively low prevalence observed in this study may be attributable to the relatively fewer number of subjects studied and to the low frequency of contact with infected persons as observed in this study (only 2.8% of cases admitted to having any history of contact with other persons with skin lesions).

Cases with dermatophytosis have a mean CD4 cell count of 226.2cells/µl, this figure can be said to be much lower when compared to other studies where mean CD4 cell counts in HIV positive patients with dermatophytosis ranged from 267-450 cells/µl[51,103,104]. Of the cases with clinical dermatophytosis, 41.6% % have CD4 cell count between 0 and 200 cells/µl. From these findings it can be concluded that dermatophytosis is directly related to the degree of immunosuppression in HIV sero-positive patients.

Tinea corporis is the commonest dermatophytic lesion seen among the cases. In previous studies sited earlier it was also found to be the commonest in the setting of HIV infection. This is in keeping with other studies where tinea corporis or capitis where found to be the commonest dermatophytosis affecting HIV patients.[70,97,102,105] All of the cases seen in this study had a CD4 cell count below 400cells/µl (two thirds below 200 cells/µl). Other studies done in the past revealed a similar high prevalence in patients with low CD4 cell count. Most of the lesions seen were of the classical annular types with active edges and healing centers or tinea incognito. A female among the control with tinea incognito admitted to chronic application of bleaching creams (see plate 4). Two of the cases with tinea corporis had extensive involvement of the trunk, limbs and flexures, with hyperpigmented, thick scaly plaques (see plate 6). This is in keeping with findings from previous studies that have demonstrated atypical presentations of tinea corporis in immunosuppressed persons with HIV infection. The paucity of such atypical lesions (in this study) described in earlier literature[72-74], may be attributed to the fact that a significant number of the patients(16.1% of the cases) have utilized one form of antifungal treatment or another prior to presentation.

Another common type of dermatophytosis seen among the cases was tinea unguium, the patients with these lesions had a CD4 count of 400 cells/µl and below. Moreover, all of the cases with onychomycosis fall within the 21-40 year age group who are more prone to trauma compared to other age groups (prior trauma is a risk factor for onychomycosis[106]. It is rare in HIV positive children. In a recent study on prevalence of dermatophytosis in HIV positive children in Nigeria, Tinea unguium constituted just 5% of the total no of dermatophytosis seen in the entire population.[105]

The typical lesions seen in HIV- proximal white subungual onychomycosis and superficial white onychomycosis were not seen in this study. However, the patients seen had nail dystrophy, discolouration, onycholysis and nail destruction (see plate 2). Onychomycosis in the setting of HIV infection usually involves the toe nails, however in this study, the cases seen were in the finger nails. This may be attributed to the fact that most of the cases seen were women (61.2 %) who are more exposed to moisture in the course of their household chores than their male counter parts.

Tinea capitis, a common dermatophyte infection in children was not seen in any HIV positive child. This may be attributed to the fact that only a small proportion of children were used in this study. A 13 year old school boy in the control group had it. He presented with patchy alopecia.

Tinea manuum and tinea pedis were encountered less frequently in this study(16.6% each) and the morphology of the lesions seen were not significantly different from those seen in immunocompetent HIV negative persons. No case of tinea cruris was seen in this study. This may be attributed to the fact that a relatively fewer number of males were used in this study since it occurs more frequently in the male sex.[107]

There is a low mycology yield of specimens cultured in this study. This may be attributed to adulteration of the culture media or prior treatment of the skin lesions by the subjects.

The commonest dermatophyte isolated in this study was trichophyton species (33.2% of isolates) This is in keeping with findings in several other studies where it has been found to be the commonest aetiologic agent of dermatophytosis in HIV infected patients.[17,102,108,109]

Microsporum species was another common dermatophyte isolated (8.3%). This is also a common dermatophyte seen in HIV patients from previous studies[97,110]. It occurs in severe immunosuppression and can be invasive.

Aspergillus species was isolated in some (41.6%) of the HIV infected patients with dermatophytosis. However, this organism is not commonly associated with dermatomycosis in HIV. It may be an incidental finding or a contaminant.[111]

Penicillium species were also isolated in some(41.6%) of the patients. These may either be due to the contamination, increased susceptibility to non-dermatophyte infections in HIV infected persons due to immunosuppression or environmental factors that favour the growth of non-dermatophytic fungi.

CONCLUSION

This study was carried out to determine the prevalence and clinical variations of the various types of dermatophytosis among HIV seropositive patients seen in Portharcourt, Southern Nigeria.

A significantly higher prevalence of dermatophytosis as well as other skin lesions was observed in the HIV positive cases when compared to the control and most of the patients had advanced HIV infection as evidenced by a low CD4 cell count (below 200cells/µl). Thus it can be concluded that the occurrence of dermatophytosis in HIV infected persons is positively associated with the degree of immunosuppression.

The age group with the highest prevalence of dermatophytosis is the 21-40 and 41-60 year age group. Males have a higher prevalence than females.

The commonest lesion found in this study was tinea corporis. Other dermatophyte lesions seen were tinea capitis, tinea manuum , pedis and onychomycosis..

The various atypical dermatophytic lesions found in HIV infected persons as reported in other studies were not commonly seen. Most of the skin lesions seen in this study were not much different from the classical lesions seen in HIV seronegative persons with the exception of 2 cases of disseminated and atypical presentation of tinea corporis. Proximal white subungual onychomycosis, said to be pathognomonic of HIV/AIDS was not seen in any patient enrolled for this study.

The commonest aetiologic agents for dermatophytosis in the group of HIV infected patients studied are trichophyton species and microsporum species which are known to be common aetiologic agents of dermatophytosis in HIV infected patients from previous studies. Other fungi that were commonly

isolated include aspergillus species, and penicillium species but these are of doubtful significance.

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