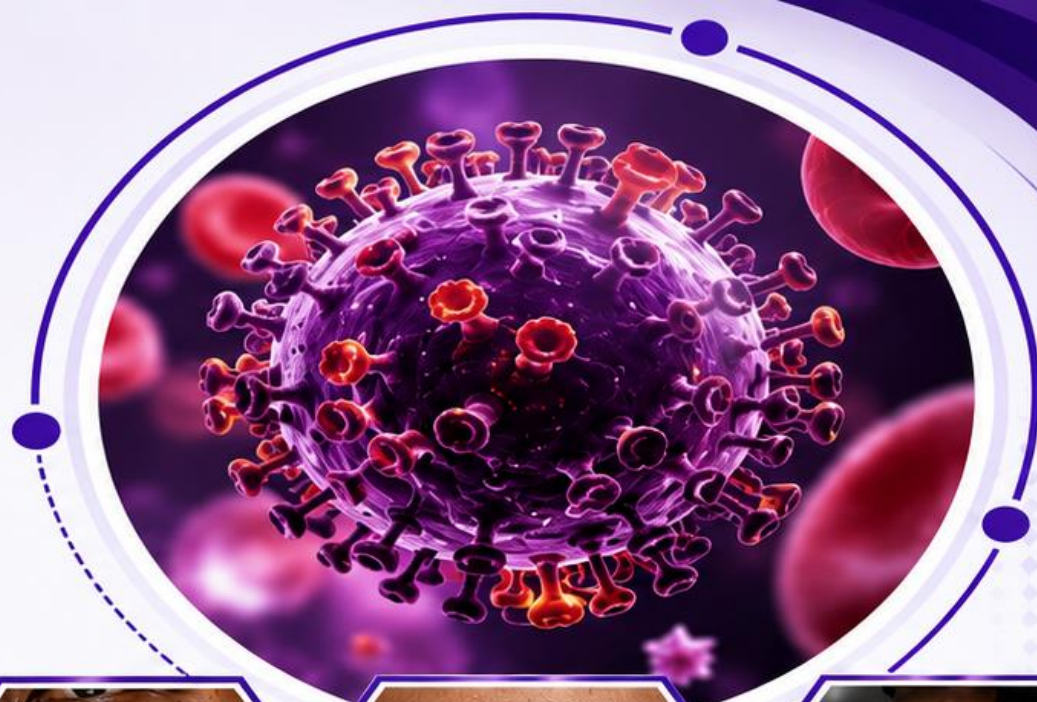


CUTANEOUS MANIFESTATIONS OF HIV



— Pr Ahmed Bouhamidi —



Learning Objectives



Describe the **cutaneous manifestations** of HIV primary infection



Identify the main **cutaneous infections** (bacterial, viral, fungal, parasitic) observed in people with HIV



Recognize the **inflammatory dermatoses** associated with HIV (seborrheic dermatitis, psoriasis, prurigo, etc.)



Diagnose the main **skin tumors** associated with HIV (Kaposi sarcoma, lymphomas, carcinomas)



Identify key **cutaneous signs** that warrant HIV serological testing



Correlate the type of skin manifestation with the degree of **immunosuppression** (CD4 count)



Prescribe appropriate additional tests and plan patient management



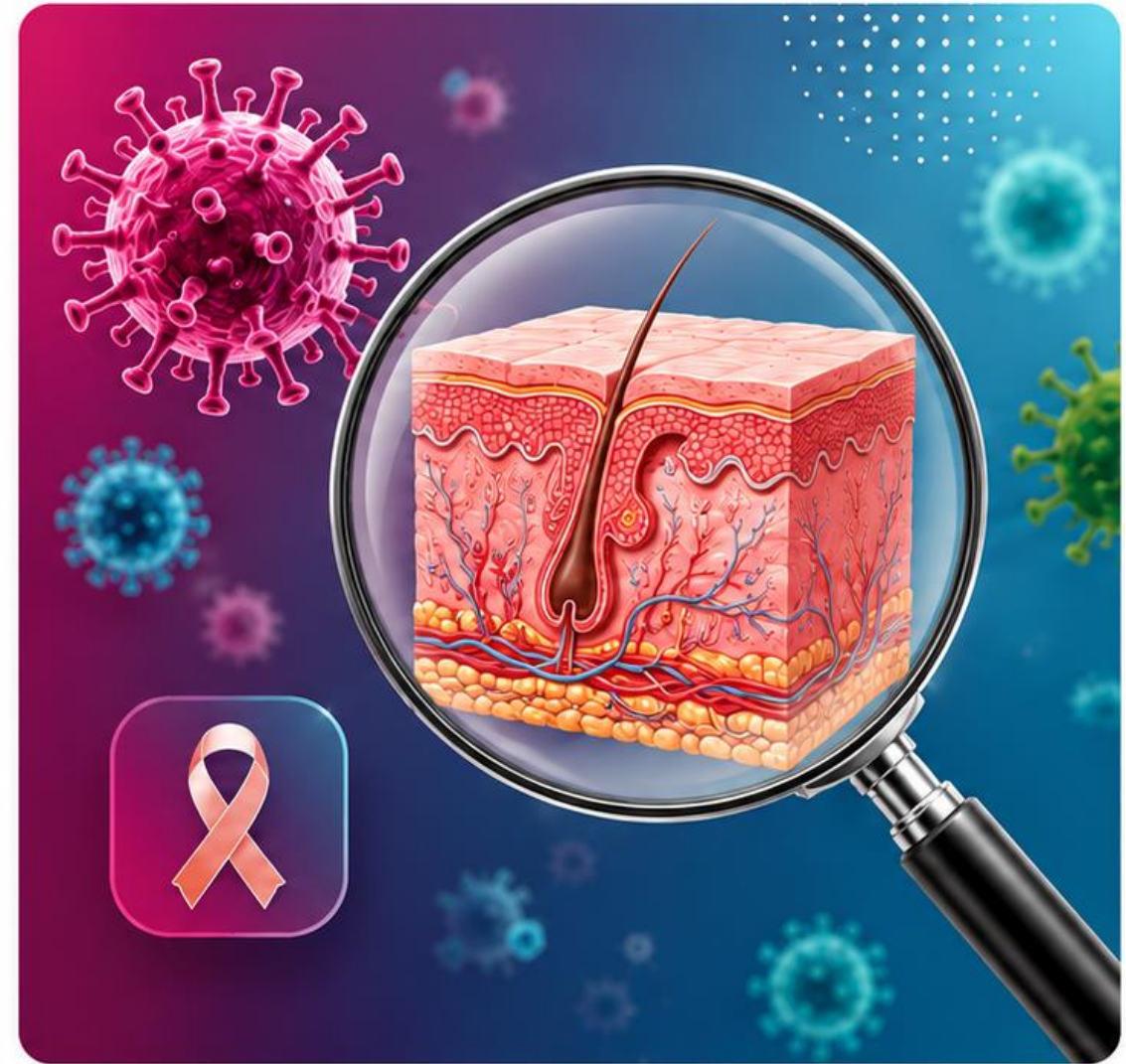
KEY MESSAGE

The skin is a reliable mirror of the immune status of people living with HIV — knowing how to read it often helps save valuable time in reaching a diagnosis.



Course Plan

- 01**  1. Introduction — epidemiology and challenges
- 02**  2. Pathophysiology — HIV, CD4 and immunosuppression
- 03**  3. Cutaneous manifestations of primary infection
- 04**  4. Inflammatory dermatoses
- 05**  5. Opportunistic skin infections (bacteria, viruses, fungi, parasites)
- 06**  6. HIV-associated skin tumors
- 07**  7. Oral and nail disorders
- 08**  8. Diagnostic approach and HIV serology
- 09**  9. Therapeutic management
- 10**  10. Interactive clinical cases and key messages



KEY MESSAGE

Ten steps, one guiding thread: recognize a dermatosis → think HIV → act.

PART 1



Introduction

*History, epidemiology and
the place of dermatology*



HISTORY



EPIDEMIOLOGY



CURRENT
CHALLENGES



THE ROLE OF
THE DERMATOLOGIST

1900

First clinical
descriptions

1950

Revolutionary
antibiotics
in treatment

1980

HIV epidemic:
new
challenges

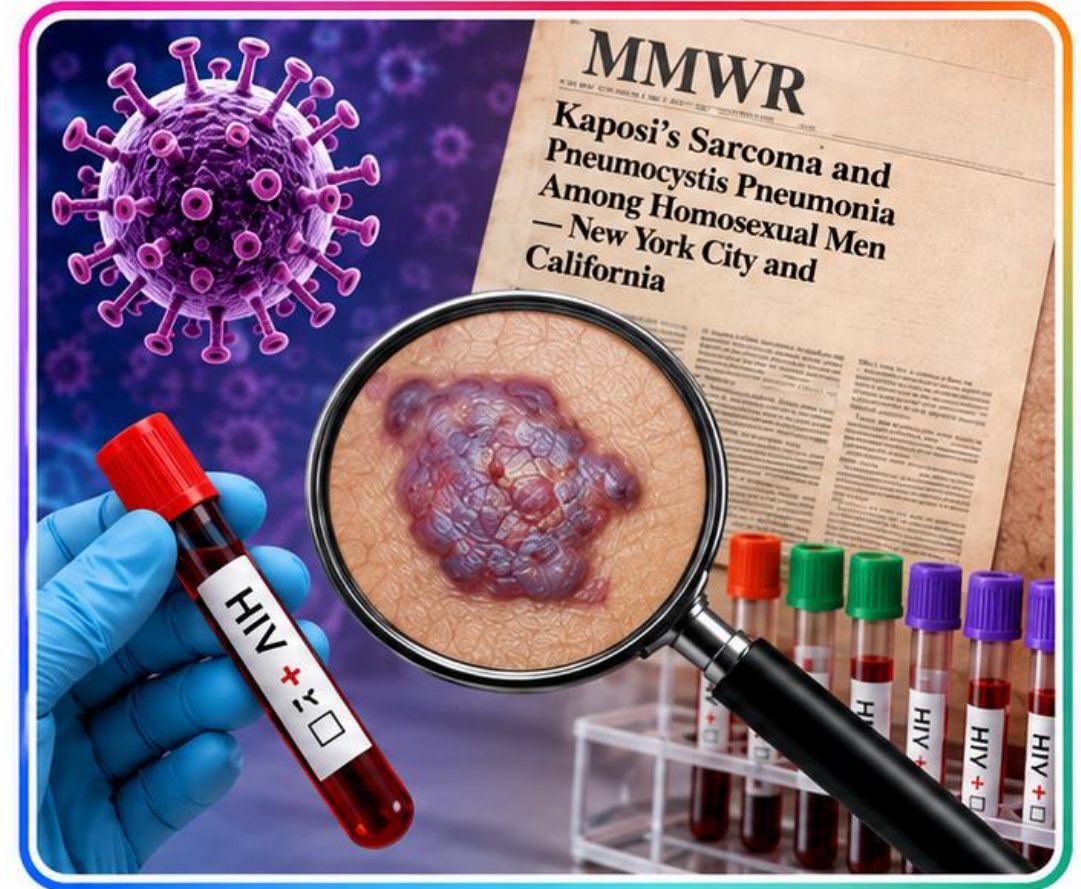
2000+

Global burden
and early
management



History: when the skin revealed the epidemic

- 
1981 (CDC, Los Angeles/New York): first cases of pneumocystis pneumonia and Kaposi sarcoma in young homosexual men — **initial alert.**
- 
1981–1982 Cutaneous Kaposi sarcoma is one of the first manifestations that led to the description of **acquired immunodeficiency syndrome (AIDS).**
- 
1983 Identification of **HIV** by the Pasteur Institute team (Barré-Sinoussi, Montagnier).
- 
1980s From the 1980s, dermatology established itself as a **sentinel discipline** in the diagnosis of HIV.
- 
Since 1996 Since the era of antiretroviral therapy (ART), the profile of dermatoses has changed: decline of severe forms, emergence of **immune reconstitution inflammatory syndrome (IRIS).**



KEY MESSAGE

The history of HIV begins in part with a dermatological observation:
Kaposi sarcoma in young patients.



Global Epidemiology of HIV



- ◆ **40.9 million** people were living with HIV worldwide in 2025 (UNAIDS/WHO), including 39.7 million adults and 1.3 million children



- ◆ **1.2 million** new infections in 2025, a 65% decrease since the peak in 1995 — but a worrying plateau in recent years



- ◆ **Sub-Saharan Africa** remains the most affected region, accounting for the majority of people living with HIV

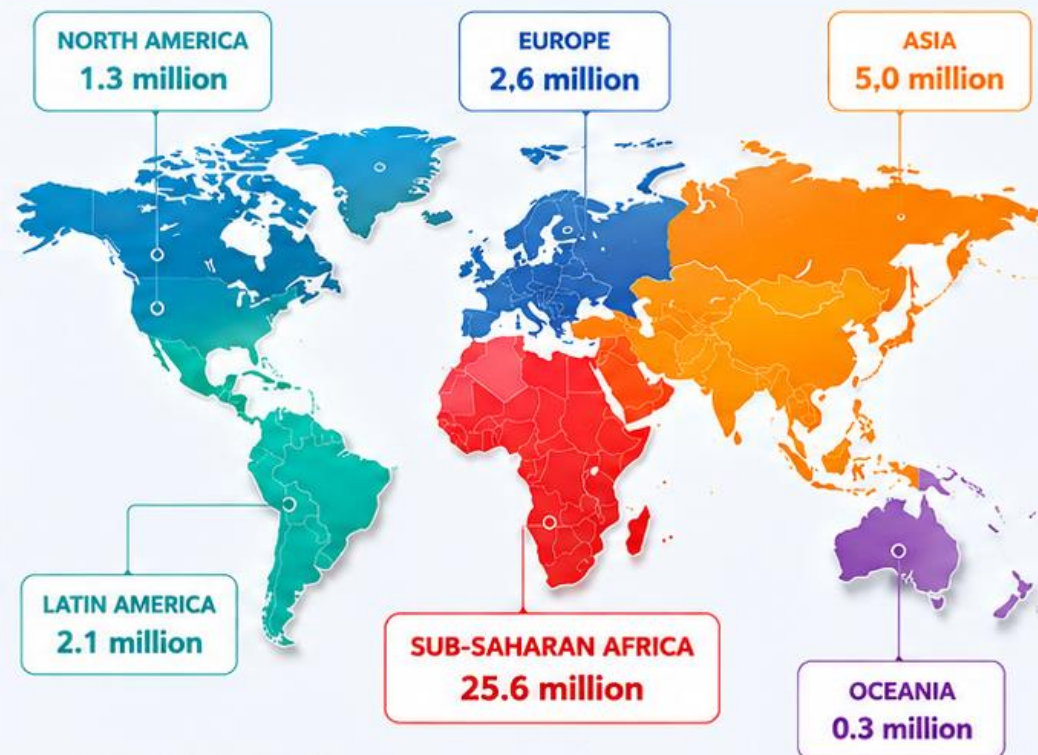


- ◆ Expanded access to antiretroviral therapy has **dramatically reduced** AIDS-related mortality since the early 2000s



- ◆ **WHO “95-95-95” strategy:**
95% of people living with HIV diagnosed, 95% on treatment, 95% with suppressed viral load

GLOBAL DISTRIBUTION OF PEOPLE LIVING WITH HIV IN 2025



Source: UNAIDS/WHO – Data 2025



KEY MESSAGE

Despite therapeutic advances, HIV remains a major public health issue — and the skin continues to be a frequent site of disease expression.



Situation in Africa



Sub-Saharan Africa concentrates the **majority** of people living with **HIV** worldwide



Particularly high prevalence in **Southern Africa** and **Eastern Africa**



High burden of **co-infections** (tuberculosis, viral hepatitis) that worsen cutaneous and systemic prognosis



Severe cutaneous manifestations (endemic Kaposi sarcoma, extensive warts, extensive dermatophytoses) remain more frequent in settings with limited access to ART



Strengthening community **screening programs** and **decentralization** of access to antiretroviral treatment

PREVALENCE OF HIV IN AFRICA BY SUB-REGION



KEY MESSAGE

In high-prevalence settings, **dermatoses are key indicators that should systematically prompt HIV screening.**



Situation in Morocco



- ♦ **Morocco** is classified among countries with a **concentrated** epidemic, mainly in key populations (sex workers, MSM, people who inject drugs)



- ♦ National strategy to fight AIDS led by the **Ministry of Health and the National AIDS Control Program (PNACP)**, with support from UNAIDS



- ♦ Strengthening of decentralized screening and free access to antiretroviral therapy within the framework of **universal health coverage**



- ♦ Moroccan dermatology is on the front line: many patients are still diagnosed during an opportunistic dermatosis (**young patients, widespread pruritus, recurrent candidiasis**)



- ♦ Need to maintain a high level of **clinical vigilance**, including outside groups at risk that are traditionally identified

HIV IN MOROCCO IN FIGURES (estimate 2024)



~ 23,000
people living with HIV (PLHIV)



~ 600
new infections per year



> 90 %
of PLHIV on antiretroviral therapy (ART)



Stable trend
of the epidemic in recent years



PNACP goal: to end the AIDS epidemic by 2030



KEY MESSAGE

In Morocco, the dermatologist can be the first — sometimes the only — health professional to raise the suspicion of HIV.



Why is dermatology central to the diagnosis of HIV?



- ◆ **More than 90%** of patients living with HIV will present with cutaneous and/or mucosal manifestations during the course of the disease



- ◆ **The skin** can be the first organ affected, often preceding any other clinical or biological sign



- ◆ **Certain dermatoses** have strong orientation value (in young subjects, extensive molluscum in adults, recurrent candidiasis)



- ◆ **The severity, extent and atypical pattern** of a “banal” dermatosis may reflect an underlying immunodepressed state



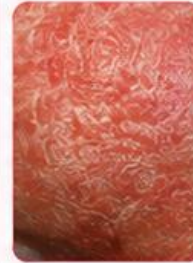
- ◆ **Cutaneous follow-up** also helps assess immune restoration under antiretroviral treatment

5 MAJOR CATEGORIES OF HIV-ASSOCIATED DERMATOSES



INFLAMMATORY

- Seborrheic dermatitis
- Psoriasis
- Xerosis



INFECTIOUS

- Candidiasis
- Dermatophytosis
- Herpes
- Zona



TUMORAL

- Kaposi sarcoma
- Cutaneous lymphomas



MUCOSAL

- Hairy leukoplakia
- Oral candidiasis
- Ulcerations



UNGUAL

- Paronychia
- Onychomycosis
- Beau's lines



KEY MESSAGE

In the presence of any severe, atypical, recurrent or unusual dermatosis for the patient's age: **think HIV.**



PART 2



Pathophysiology

From the virus to cutaneous immunosuppression



THE VIRUS



IMMUNE
RESPONSE



PROGRESSIVE
IMMUNOSUPPRESSION



CUTANEOUS
MANIFESTATIONS



HIV
infection



Viral
replication



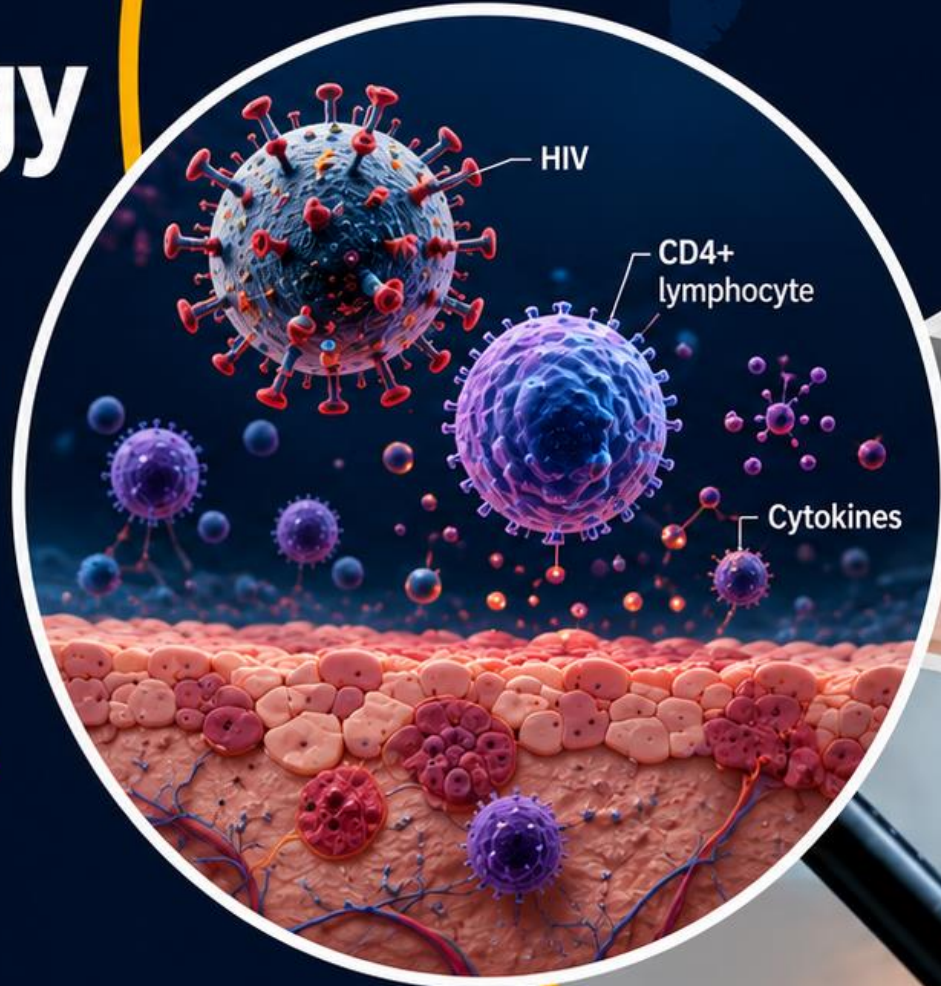
CD4+
depletion



Cellular
immunosuppression



Cutaneous
manifestations



HIV-1 and HIV-2: Two viruses, two evolutionary profiles



HIV is a **retrovirus** of the Lentivirus family, which targets cells expressing the CD4 receptor (CD4+ lymphocytes, macrophages, dendritic cells)



HIV-1: responsible for the vast majority of infections worldwide, high genetic variability (groups M, N, O, P)



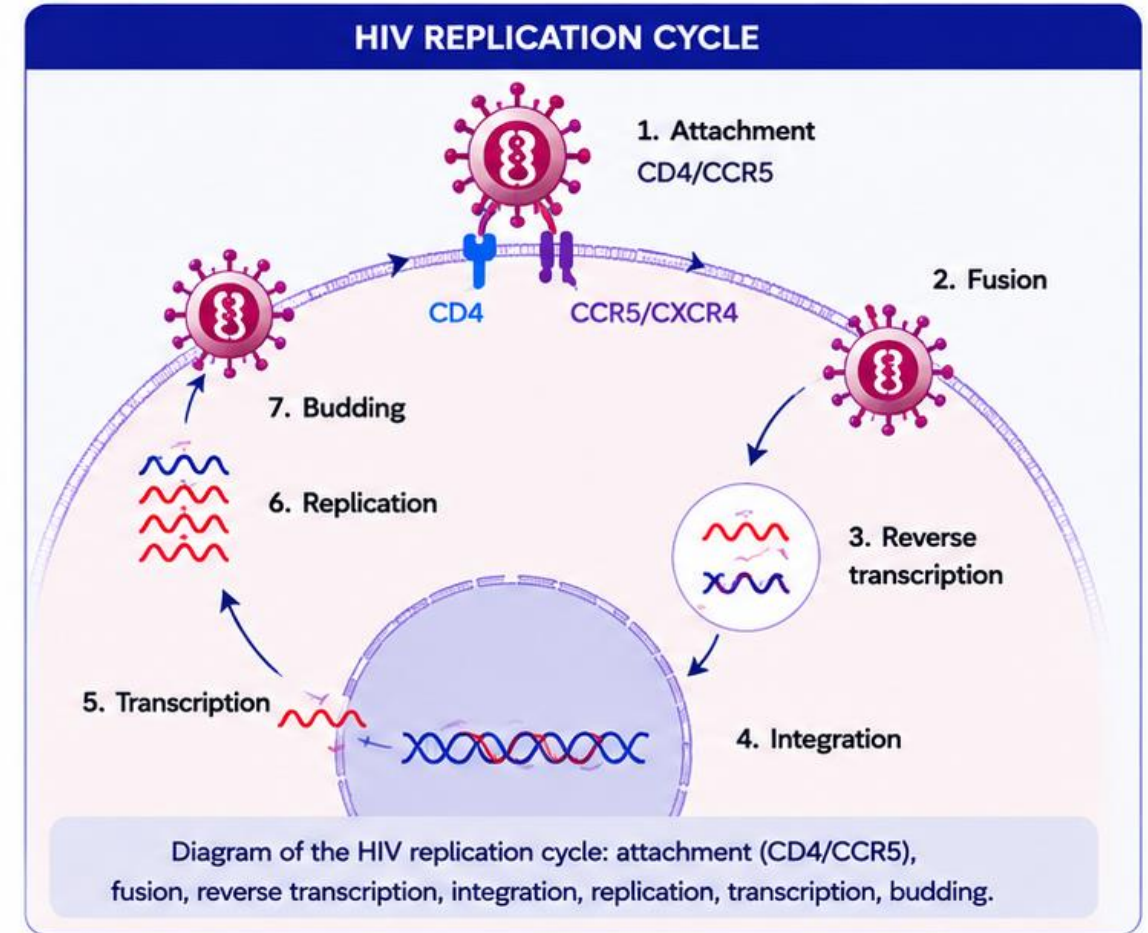
HIV-2: lower prevalence, concentrated in West Africa, generally slower progression to immunosuppression



The viral cycle (entry via CD4/CCR5-CXCR4, reverse transcription, integration, replication) leads to the progressive destruction of CD4+ T lymphocytes



CD4 depletion is the central mechanism underlying the cutaneous and mucosal manifestations observed



KEY MESSAGE

All the cutaneous semiology of HIV is explained by a single central mechanism: the progressive decline of CD4+ T lymphocytes.

Progression of immunosuppression and appearance of dermatoses



> 500
cells/mm³

Pre-infection / asymptomatic phase

- ◆ Primo-infection exanthem, early seborrheic dermatitis, follicular boils



350 – 500
cells/mm³

Mild to moderate immunosuppression

- ◆ Herpes zoster, molluscum contagiosum, psoriasis, warts (HPV), severe seborrheic dermatitis



200 – 350
cells/mm³

Moderate to severe immunosuppression

- ◆ Recurrent oral candidiasis, oral hairy leukoplakia, prurigo, eosinophilic folliculitis



< 200
cells/mm³

AIDS — severe immunosuppression

- ◆ Extensive Kaposi sarcoma, cryptococcosis, disseminated leishmaniasis, severe/profuse (e.g., Norwegian) scabies, opportunistic infections

↓ CD4 lymphocyte count (cells/mm³)



KEY MESSAGE

The lower the CD4 count, the more dermatoses become severe, extensive and opportunistic.

Summary Table: CD4 Count and Dermatologic Manifestations

CD4 COUNT (cells/mm ³)	LEVEL OF IMMUNOSUPPRESSION	TYPICAL CUTANEOUS AND MUCOSAL MANIFESTATIONS
 > 500	Pre-infection / asymptomatic phase 	 Pre-infection exanthem, follicular boils, early seborrheic dermatitis
 350 – 500	Mild to moderate immunosuppression 	 Herpes zoster, molluscum contagiosum, warts (HPV), severe seborrheic dermatitis, psoriasis
 200 – 350	Moderate to severe immunosuppression 	 Recurrent oral candidiasis, oral hairy leukoplakia, prurigo, eosinophilic folliculitis
 100 – 200	Severe immunosuppression 	 Kaposi sarcoma, extensive dermatophytoses, chronic ulcerative herpes
 < 100	Very severe immunosuppression 	 Extensive (e.g., Norwegian) scabies, cutaneous cryptococcosis, disseminated histoplasmosis, giant molluscum of the face
 < 50	Extreme immunosuppression 	 Severe and disseminated opportunistic infections, cutaneous lymphomas



KEY MESSAGE

Certain “sentinel” dermatoses—such as herpes zoster in a young adult, extensive molluscum contagiosum, or recurrent candidiasis—should **prompt consideration of HIV infection** and assessment of immune status.

→ These presentations should prompt **HIV testing** and, when infection is confirmed, immunological assessment including the **CD4 count**.



PART 3

Manifestations of acute HIV infection

Acute retroviral syndrome



GENERAL SIGNS

- Fever
- Asthenia
- Headache
- Myalgias / arthralgias
- Pharyngitis



LYMPHADENOPATHY

- Generalized
- Diffuse
- Persistent



CUTANEOUS MANIFESTATIONS

- Maculopapular exanthem
- Urticaria
- Papular eruption
- Pruritus



OTHER INVOLVEMENT

- Oral ulcers
- Diarrhea
- Hepatosplenomegaly



HIV
Infection



Viral
replication



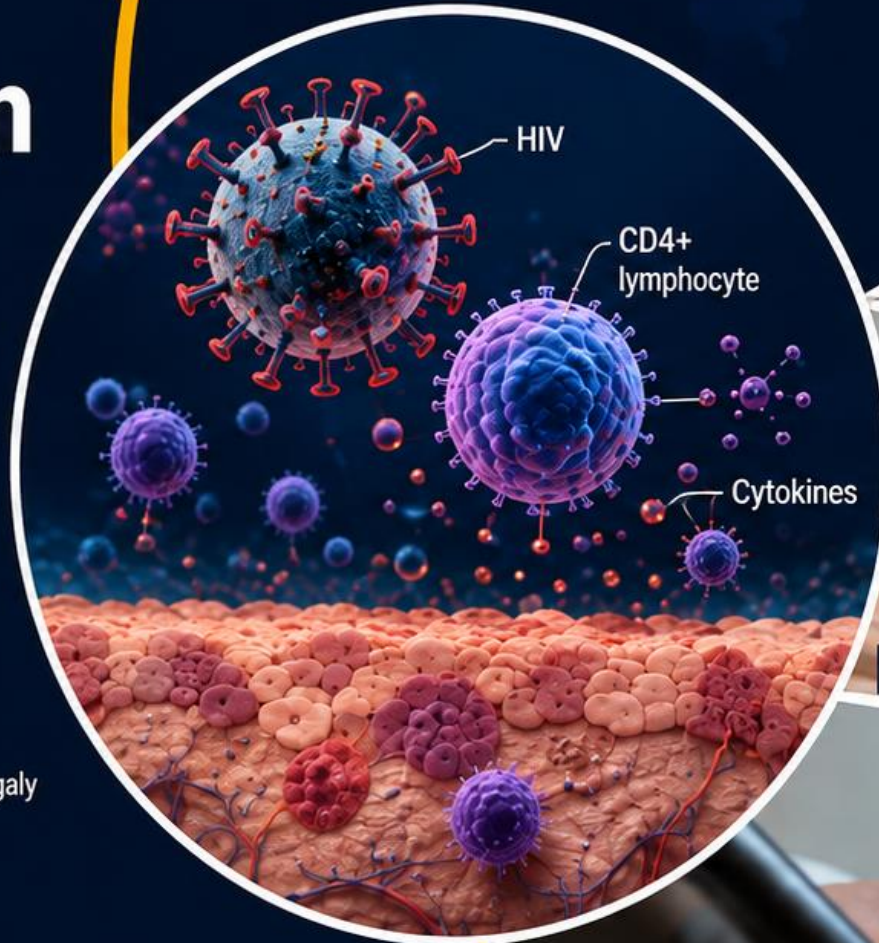
CD4+
depletion



Cellular
immunosuppression



Cutaneous
manifestations



SKIN RASH



CLINICAL EXAMINATION



PATIENT CARE

Acute retroviral syndrome: exanthem and general signs



Occurs 2 to 4 weeks after exposure,
in 40–90% of patients (often unrecognized or attributed to another viral illness)



Maculopapular exanthem, morbilliform, non-pruritic most often, involving trunk, face and sometimes palms/soles



Associated pseudo-influenza-like syndrome: fever, asthenia, myalgia, pharyngitis, lymphadenopathy



Usual duration: 1 to 3 weeks,
spontaneous resolution



This clinical picture, although not specific, must systematically raise the question of a recent high-risk exposure



Maculopapular exanthem
of the trunk



Facial involvement



Palmar involvement



KEY MESSAGE

In any febrile exanthem with pseudo-influenza-like syndrome in a young adult, **systematically consider a recent high-risk exposure** and offer HIV testing (viral load and p24 antigen).

Mucosal ulcers of acute HIV infection



Oral ulcers: painful aphthae or erosions of the buccal mucosa, jugal mucosa, palate or pharynx



Genital ulcers: non-specific erosions or ulcers, which may mimic a classic sexually transmitted infection (herpes, syphilis)



These ulcers may be isolated or associated with exanthem and pseudo-influenza-like syndrome



Broad differential diagnosis: herpetic primary infection, primary syphilis, oral aphthae — requiring a complete STI workup



Mucosal ulcers during a febrile syndrome increase the suspicion of acute HIV infection



KEY MESSAGE

Any mucosal ulcer associated with a febrile syndrome should prompt elimination of **acute HIV infection**, in addition to the usual STI workup.

Typical mucosal ulcers in acute HIV infection



Oral ulcer
(*buccal mucosa*)



Palatal ulcer



Pharyngeal ulcers

PART 4

Inflammatory Dermatoses

*Mucocutaneous dermatoses,
but more frequent and more severe*



MORE FREQUENT

- Atopic dermatitis
- Psoriasis
- Seborrheic dermatitis
- Rosacea
- Alopecia areata



MORE SEVERE

- Widespread involvement
- Chronicity
- Functional impact
- Frequent relapses



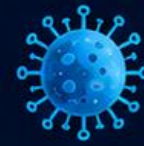
MAJOR IMPACT

- Quality of life
- Mental health
- Comorbidities
- Healthcare costs



OBJECTIVES

- Control inflammation
- Restore the barrier
- Prevent relapses
- Global management



TRIGGERING FACTORS

Genetic and environmental factors



INNATE AND ADAPTIVE INFLAMMATION

Innate and adaptive activation



PERSISTENCE

Chronic inflammation



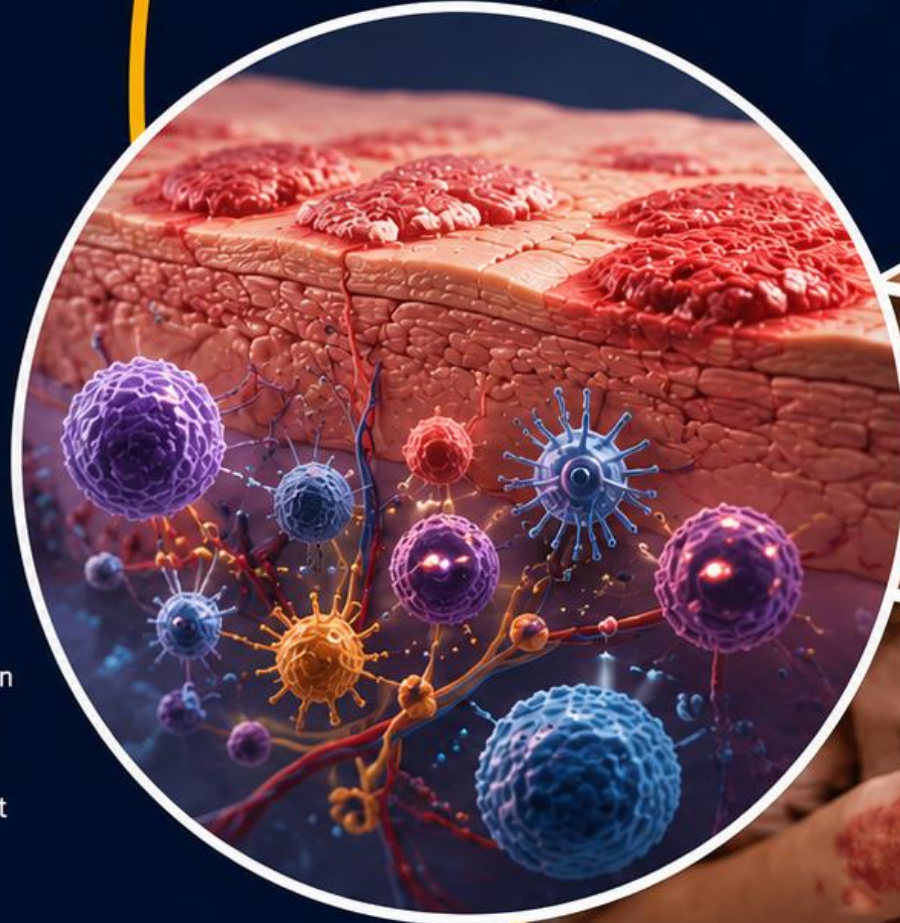
ALTERATION

Impaired skin barrier and dysbiosis



CLINICAL MANIFESTATIONS

More frequent, more extensive and more severe forms



Seborrheic dermatitis



Very high prevalence among PLWH

(up to 40–80% depending on the degree of immunodepression), vs. about 3–5% in the general population



Pathophysiology: proliferation of *Malassezia* spp. favored by cutaneous immune dysregulation



Clinical: well-demarcated erythematous-squamous oily plaques on the face (nasolabial folds, eyebrows), scalp and presternal area — often more extensive and inflammatory in the general population



May be an **early warning sign**, sometimes before the diagnosis of HIV seropositivity



Treatment: topical antifungals (ketoconazole), short-course topical corticosteroids, control of viral load with ARVs



KEY MESSAGE

A severe, extensive, or treatment-resistant seborrheic dermatitis in a young adult should prompt **consideration of HIV infection**.

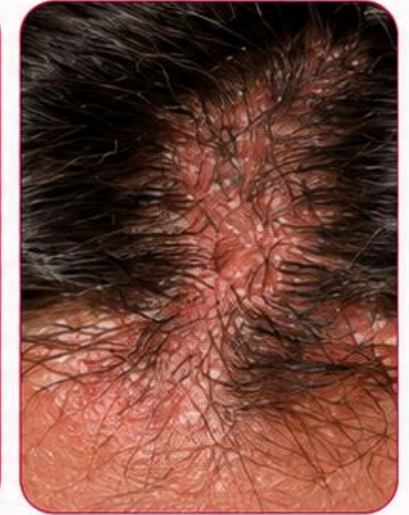
Severe seborrheic dermatitis of the face in an HIV+ patient (nasolabial folds, eyebrows)



Nasolabial folds



Eyebrows



Scalp

Psoriasis during HIV infection



- ◆ **May be revealed, aggravated or triggered de novo** by HIV infection, in contrast to other autoimmune diseases that are associated with immunosuppression



- ◆ **Forms are often more severe, extensive and treatment-resistant**, with a higher prevalence of psoriatic arthritis



- ◆ **Specific forms described:** inverted psoriasis, erythrodermic psoriasis, pustular psoriasis



- ◆ **Treatment must take immune status into account:** prefer phototherapy and ARVs as first-line therapy, use caution with conventional immunosuppressants



- ◆ **Biologic therapies (anti-TNF, anti-IL-17/23)** may be considered under effective ART coverage, in a multidisciplinary setting

Extensive psoriasis in an HIV+ patient



Erythrodermic psoriasis



Inverted psoriasis



Pustular psoriasis



KEY MESSAGE

HIV is one of the rare situations where **severe immunodepression**, rather than causing it, is more likely to **trigger an inflammatory dermatosis** such as psoriasis.

Xerosis, eczema, and prurigo



- ◆ **Cutaneous xerosis:** very frequent, often early, favored by alteration of the skin barrier and sometimes by ART



- ◆ **Eczema / atopic dermatitis-like:** may appear or worsen in adults, sometimes atypical



- ◆ **Prurigo (“papular pruritic eruption”):** excoriated papules, very pruritic, symmetrical, predominantly on the limbs — associated with moderate to severe immunodepression ($CD4 < 200-350$)



- ◆ **Chronic pruritus of unexplained cause in an HIV+ patient** should prompt search for specific causes (diabetes, eosinophilic folliculitis, drug toxicity) before considering prurigo linked to HIV



- ◆ **Symptomatic treatment:** emollients, antihistamines, topical corticosteroids, phototherapy in refractory forms, optimization of antiretroviral therapy

Frequent cutaneous manifestations in an HIV+ patient



Cutaneous xerosis



Eczema
(atopic dermatitis-like)



Prurigo
(pruriginous papules)



KEY MESSAGE

Prurigo in HIV is a **significant marker of immunodepression** and requires **monitoring of CD4 count**.

Eosinophilic folliculitis (HIV eosinophilic folliculitis)



Specific inflammatory dermatosis described almost exclusively in HIV+ patients, typically in an advanced stage of immunodepression ($CD4 < 250-300$)



Clinical: very pruritic papulo-pustular follicular eruption, predominantly on the face, neck, upper trunk and limbs



Histological diagnosis: perifollicular infiltrate rich in eosinophils, to distinguish from other folliculitis (bacterial, Malassezia, Demodex)



Differential diagnoses to systematically rule out: bacterial folliculitis (*Staphylococcus aureus*), *Malassezia* folliculitis, scabies, toxidermia



Treatment often difficult: dermocorticoids, antihistamines, narrowband UVB phototherapy, isotretinoin in low doses in refractory forms; clear improvement under effective ART

Eosinophilic folliculitis in HIV

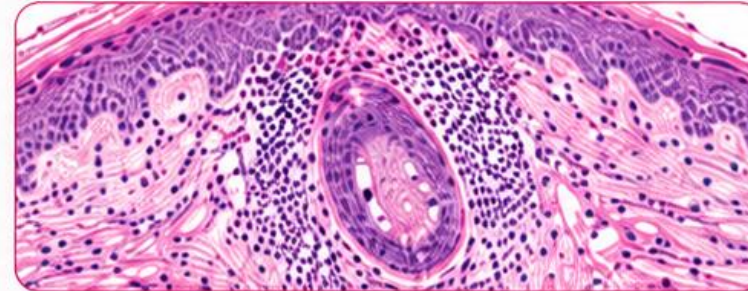
Face



Upper trunk



Upper limb



Histology:
perifollicular infiltrate
rich in eosinophils
(H&E, $\times 200$)



KEY MESSAGE

Eosinophilic folliculitis is an **almost pathognomonic** dermatosis of advanced HIV immunodépression and requires an **urgent immunovirological assessment**.

PART 5

Opportunistic cutaneous infections

Bacteria, viruses, fungi, parasites



BACTERIA

- Staphylococci
- Streptococci
- Mycobacteria
- Bartonella



VIRUSES

- Herpes simplex
- Varicella-zoster
- HPV
- Molluscum contagiosum



FUNGI

- Candida spp.
- Dermatophytes
- Malassezia spp.
- Cryptococcus spp.



PARASITES

- Sarcoptes scabiei
- Leishmania spp.
- Toxoplasma gondii
- Strongyloides spp.



CLINICAL ISSUES

- Early diagnosis
- Complications
- Drug resistance
- Adapted treatment



IMMUNODEFICIENCY

Immune deficiency
(e.g., HIV)



LOSS OF IMMUNE CONTROL

Reduced skin
defenses



OPPORTUNISTIC COLONIZATION

Proliferation of
opportunistic agents



CUTANEOUS INFECTIONS

Various clinical
manifestations



DIAGNOSIS AND MANAGEMENT

Early identification
and tailored treatment



VIRUSES

FUNGI

BACTERIA

PARASITES

Impétigo, furunculosis and cellulitis



- ◆ **Increased frequency** of cutaneous infections due to *Staphylococcus aureus* and *Streptococcus pyogenes* in PLWH, particularly in cases of immunodepression



- ◆ **Impetigo**: crusted skin lesions, often extensive and recurrent



- ◆ **Furunculosis**: deep involvement of the pilosebaceous follicle, recurrent and multiple forms to consider as a warning sign



- ◆ **Cellulitis / bacterial dermohypodermatitis**: sometimes more severe presentation, higher risk of complications (abscess, bacteremia) in case of advanced immunodepression



- ◆ **Management**: antibiotic therapy guided by antibiogram, screening for MRSA carriage in cases of recurrence, optimization of virological control

Impetigo



Crusted
honey-colored lesions

Furunculosis



Deep inflammatory
nodule with pustule

Cellulitis



Diffuse erythema, edema,
warmth, pain



KEY MESSAGE

Benign cutaneous bacterial infections, but recurrent or unusually severe infections, should prompt **investigation of an underlying immunodeficiency**.

Bacillary angiomatosis



- ◆ **Infection:** due to *Bartonella henselae* or *Bartonella quintana*, quasi-specific to immunocompromised patients (CD4 count often < 100)



- ◆ **Clinical:** red–violaceous vascular papules or nodules that may mimic Kaposi sarcoma or a botryomycoma



- ◆ **May be associated with:** a systemic condition (hepatic peliosis, bone involvement, fever)



- ◆ **Diagnosis:** skin biopsy with Warthin–Starry staining, specific PCR; histology shows vascular proliferation with bacterial aggregates



- ◆ **Treatment:** prolonged antibiotic therapy (macrolides or doxycycline), with a good clinical response in the absence of specific treatment

Violaceous angiomatoid nodules

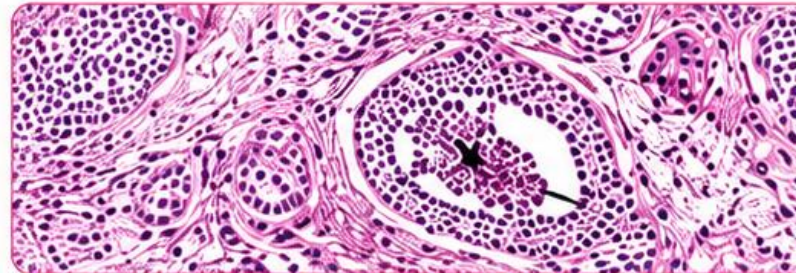


Comparison with Kaposi sarcoma



Kaposi sarcoma

Histology (skin biopsy)



Vascular proliferation in the dermis with bacterial aggregates (arrow).



KEY MESSAGE

In the presence of a vascular lesion suggestive of Kaposi sarcoma in a severely immunocompromised patient, bacillary angiomatosis is a key differential diagnosis that should not be overlooked — **it is treated with antibiotics.**

Herpes Simplex (HSV-1, HSV-2)



◆ **Very common infection** in people living with HIV, with more frequent recurrences and more extensive lesions.



◆ **In severely immunocompromised patients (low CD4 count):** chronic, extensive, sometimes pseudotumoral ulcerations persisting for more than 4 weeks may occur.



◆ **Typical locations:** perioral, genital, and perianal.



◆ **Bidirectional interaction with HIV:** genital herpes increases the risk of both HIV acquisition and transmission.



◆ **Treatment:** acyclovir, valacyclovir, or famciclovir; in cases of acyclovir resistance associated with severe immunosuppression, foscarnet may be required.

Chronic perianal herpetic ulceration



Clinical appearance: chronic perianal herpetic ulceration in a severely immunocompromised patient.



AIDS-defining criterion: ulcerations lasting more than 4 weeks (CDC).



Key point: lesions may be more extensive, atypical, and recurrent in people living with HIV.



KEY MESSAGE

A herpetic ulcer persisting for more than one month in a patient living with HIV is an **AIDS-defining condition and requires **intensified antiviral management**.**

Zoster (Varicella-Zoster Virus)



- ◆ **Incidence:** more than 15-fold higher in people living with HIV without effective antiretroviral therapy; remains 3 to 4 times higher than in the general population even with ART (NIH OI Guidelines)



- ◆ **Classic warning sign:** zoster in a young subject (< 50 years) without any other identified cause of immunosuppression



- ◆ **Possible particular clinical forms:** multidermatomal, necrotizing, recurrent, or disseminated (cutaneous dissemination) in cases of severe immunodepression



- ◆ **Complications to look for:** post-herpetic neuralgia, ocular involvement (ophthalmic zoster), central nervous system involvement



- ◆ **Treatment:** valacyclovir or acyclovir, intravenous route in severe or disseminated forms; management of associated pain

Thoracic multidermatomal zoster in a young person living with HIV



Zoster in a subject < 50 years without another cause of immunodepression: classic warning sign.



Possible severe forms: multidermatomal, necrotizing, recurrent, or disseminated.



Complications: post-herpetic neuralgia, ocular involvement, central neurological involvement.



Early treatment and optimal virological control are essential.



KEY MESSAGE

Any zoster in a person **under 50 years of age**, in the absence of another obvious cause of immunosuppression, should prompt **HIV serologic testing**.

Molluscum Contagiosum



- ◆ **Infection:** caused by a poxvirus, usually benign and self-limiting in immunocompetent individuals (especially children).



- ◆ **In immunocompromised patients with HIV infection (low CD4 count):** lesions may be numerous, large (“giant molluscum”), and often localized on the face.



- ◆ **Extensive or atypical molluscum contagiosum** in an adult should raise suspicion of underlying HIV infection.



- ◆ **Differential diagnosis in severely immunocompromised patients:** disseminated cutaneous cryptococcosis (similar umbilicated lesions).



- ◆ **Treatment:** curettage, cryotherapy, or topical therapy; marked improvement may occur with immune restoration following effective ART.

Profuse facial molluscum contagiosum in a severely immunocompromised patient living with HIV



KEY MESSAGE

Profuse and giant molluscum contagiosum in an adult is not benign: it should prompt investigation for HIV infection and, in cases of severe CD4 lymphopenia, consideration of concurrent cutaneous cryptococcosis.

Human Papillomavirus (HPV) Infections



- ◆ **Increased prevalence and persistence** of HPV infections in people living with HIV, with a higher risk of co-infection by oncogenic genotypes.



- ◆ **Cutaneous warts and anogenital condylomas** are often more numerous, more extensive, and more resistant to treatment.



- ◆ **Increased risk of intraepithelial neoplasia** and HPV-related epidermoid carcinomas (cervix, anus, oropharynx) — warrants enhanced screening (Pap smear, cervical cytology, anal cytology).



- ◆ **HPV vaccination is recommended** in people living with HIV, including those beyond the usual age ranges, in accordance with current guidelines.



- ◆ **Treatment of lesions:** cryotherapy, electrocautery, immunomodulators (e.g., imiquimod), podophyllotoxin — depending on the location.

Clinical manifestations of HPV infections in a patient living with HIV



Flat warts
multiple on the face



Accumulated condylomas
extensive perianal



Genital condylomas
multiple



Intraepithelial lesion
anal (AIN)

Recommended enhanced screening in a patient living with HIV



Regular cervical
screening
(Pap smear)
according to
guidelines



Anal cytology
in patients
at risk



Surveillance of
oropharyngeal lesions
if risk factors
are present



KEY MESSAGE

In a patient living with HIV, HPV infection warrants **enhanced screening** for precancerous lesions, particularly **cervical and anal**.

Oral Hairy Leukoplakia (EBV)



- ◆ **Lesion caused by replication of Epstein-Barr virus (EBV)** in the lingual epithelium.



- ◆ **Clinical features:** white, vertical, non-scrapable plaques on the lateral borders of the tongue (unlike oral candidiasis which can be scraped off), seating on the lateral borders of the tongue.



- ◆ **Usually asymptomatic**, often discovered incidentally during an oral examination.



- ◆ **Highly specific marker of significant HIV-related immunosuppression** (although rare exceptional cases exist in immunocompetent individuals).



- ◆ **Treatment:** usually not necessary (improvement after HAART); topical treatment or podophyllin may be considered in selected cases

Oral hairy leukoplakia
(lateral borders of the tongue)



KEY MESSAGE

Oral hairy leukoplakia, a non-scrapable lesion, is a **highly specific marker of HIV-related immunosuppression** — helping to differentiate it from **oral candidiasis**.

Candidiasis



- ◆ **Oropharyngeal candidiasis:** removable white plaques leaving an erythematous mucosa (pseudomembranous candidiasis), or erythema without plaque (erythematous form).



- ◆ **Esophageal candidiasis:** dysphagia, odynophagia — AIDS-defining condition (CDC); should be systematically investigated in cases of dysphagia.



- ◆ **Cutaneous candidiasis:** intertrigo of large skin folds, chronic paronychia.



- ◆ Recurrent or treatment-resistant oropharyngeal candidiasis is strongly associated with a low **CD4** count (**CD4 < 200–350** cells/ μ L).



- ◆ **Treatment:** topical antifungals (nystatin, miconazole) as first-line therapy; oral fluconazole in extensive or esophageal forms.

Pseudomembranous oral candidiasis
(removable white plaques on the palate and tongue)



KEY MESSAGE

Recurrent oropharyngeal candidiasis in a young adult, in the absence of an obvious predisposing factor (diabetes, corticosteroid therapy), should prompt investigation for HIV infection.

Dermatophytoses and Pityriasis Versicolor



- ◆ **Dermatophytoses:** more extensive, atypical and treatment-resistant in immunocompromised patients; frequent onychomycosis, often bilateral.



- ◆ **An inverse correlation** has been reported between the extent of dermatophytoses/candidiasis and CD4 count in several cohort series.



- ◆ **Pityriasis versicolor:** hypo- or hyperpigmented macular lesions with fine scaling, favored by immunosuppression and heat/humidity.



- ◆ **Definitive clinical diagnosis** is based on direct mycological examination (squamous scraping, Wood's lamp for pityriasis versicolor).



- ◆ **Treatment:** topical or systemic antifungals (terbinafine, itraconazole) according to the extent of lesions.

Extensive dermatophytosis of the trunk



Pityriasis versicolor



KEY MESSAGE

The extent and treatment resistance of cutaneous and unguinal mycoses in a young adult should alert to a possible underlying immunosuppression.

Deep mycoses with cutaneous expression

Mycosis	Terrain / CD4	Cutaneous presentation	Diagnostic & treatment
 Cryptococcosis	CD4 count often < 100	• Umbilicated papules • Mimicking profuse molluscum • Sometimes ulcerated 	• Biopsy + India ink stain • Cryptococcal antigen • Amphotericin B / fluconazole
 Disseminated histoplasmosis	CD4 count often < 100–150 , endemic areas	• Umbilicated papules or nodules • Mucocutaneous ulcers • Multi-organ involvement 	• Biopsy + culture • Urinary antigen • Itraconazole or amphotericin B depending on severity



KEY MESSAGE

Any umbilicated papulo-nodular lesion in a severely immunocompromised patient (CD4 < 100**) requires ruling out a deep mycosis before concluding simple molluscum.**

Profuse (Norwegian) Scabies and Demodicosis



- ◆ **Profuse scabies (Norwegian scabies):** hyperkeratotic and hypoinflammatory form occurring in severely immunocompromised patients (CD4 count often < 150)



- ◆ **Clinical features:** diffuse crusted hyperkeratosis, may be pruriginous or not (unlike classic scabies), extremely contagious (considerable parasitic burden)



- ◆ **High risk of nosocomial transmission:** imperative contact isolation



- ◆ **Demodicosis:** proliferation of *Demodex folliculorum* favored by immunosuppression, facial pustular folliculitis-like eruption



- ◆ **Treatment of profuse scabies:** oral ivermectin combined with a topical scabicide treatment, several repeated courses, rigorous environmental decontamination

Profuse (Norwegian) scabies



Diffuse crusted hyperkeratosis
of the hands

Demodicosis



Pustular folliculitis
of the face



KEY MESSAGE

Profuse scabies, which may be non-pruriginous but is extremely contagious, is a **diagnostic emergency** and a sign of **severe immunosuppression** requiring **immediate isolation** measures.

Skin tumors associated with HIV

Kaposi's sarcoma, lymphomas, carcinomas



MOST FREQUENT TUMORS

- Kaposi's sarcoma
- Cutaneous lymphomas
- Epidermoid carcinomas
- HPV-associated epidermoid carcinomas



RISK FACTORS

- Advanced immunodeficiency (low CD4 count)
- High viral load
- Co-infections (HPV, EBV, HHV-8)
- UV exposure



CLINICAL FEATURES

- Multiple lesions
- Rapid evolution
- Various sites
- Poor prognosis related to immune status



MANAGEMENT

- Optimal antiretroviral therapy
- Local and systemic treatments
- Regular dermatologic surveillance
- Prevention and screening



HIV
Infection



CD4+
Immunodeficiency ↓



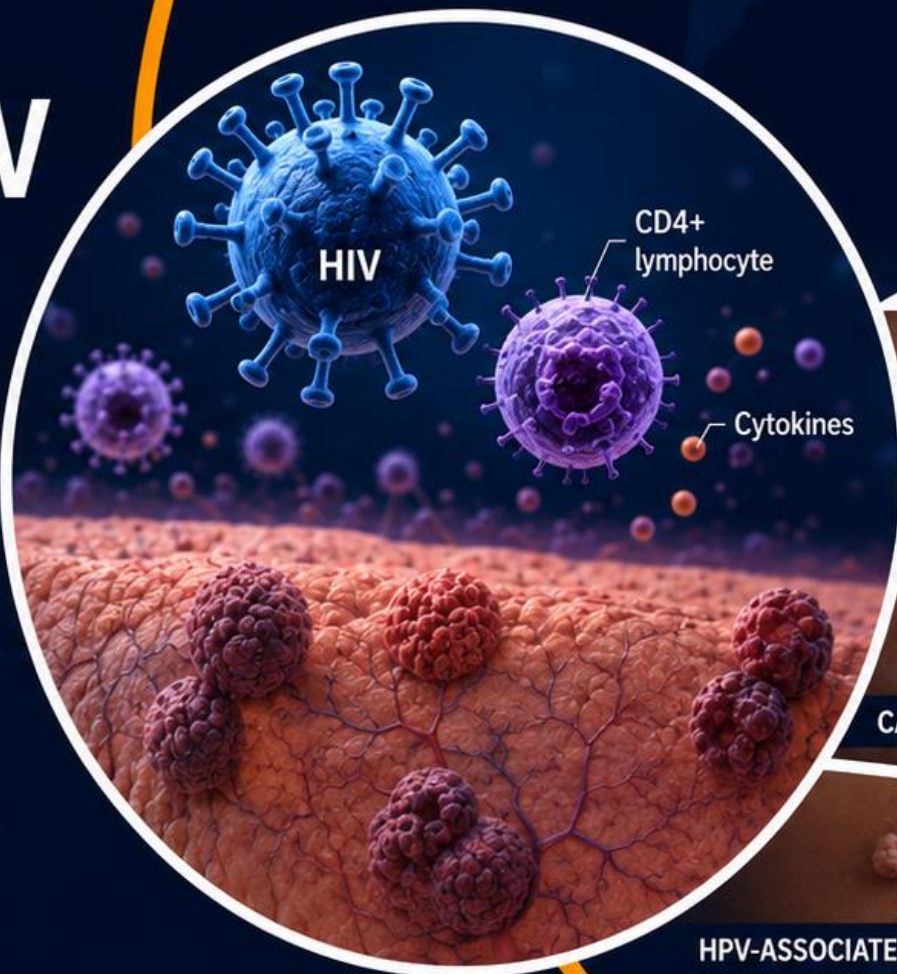
Immune
dysregulation



Viral and
inflammatory
oncogenesis



Associated
cutaneous
tumors



KAPOSI'S SARCOMA

LYMPHOMAS

CARCINOMAS

HPV-ASSOCIATED
TUMORS

Kaposi Sarcoma



- ◆ **Vascular tumor** related to HHV-8 (human herpesvirus type 8), epidemic form associated with HIV — AIDS-defining condition (CDC)



- ◆ **Clinical features:** violaceous/red-brown macules, papules or nodules, often multiple, which may involve the skin, mucous membranes (palate), and internal organs (lungs, GI tract)



- ◆ **May occur at variable CD4 counts** but the risk increases markedly below **200–500/mm³**



- ◆ **Diagnostic confirmation:** skin biopsy (atypical vascular proliferation, spindle cells, HHV-8/LANA-1 positivity on immunohistochemistry)



- ◆ **Treatment:** immune restoration with ART as first-line therapy (may suffice in limited forms), chemotherapy in disseminated or visceral forms

Kaposi Sarcoma



Multiple cutaneous
maculo-nodular lesions



Mucosal involvement
(palate)



Visceral involvement
(lung)



KEY MESSAGE

Kaposi sarcoma remains the **most characteristic** cutaneous tumor of HIV and systematically requires an **assessment of extent** and **initiation or optimization of antiretroviral therapy**.

Cutaneous lymphomas and HIV-associated carcinomas



- ◆ **Non-Hodgkin lymphomas:** higher risk in PLWH, particularly at advanced immunosuppression; possible cutaneous involvement (nodules, infiltrated plaques)



- ◆ **Primary cutaneous lymphoma** remains rare but should be considered in the presence of an atypical and treatment-resistant cutaneous lesion



- ◆ **Epidermoid carcinomas:** increased incidence, especially in sun-exposed areas and in HPV-related sites (anus, anogenital region)



- ◆ **Basocellular carcinomas:** also increased frequency, sometimes with more aggressive presentation



- ◆ Any persistent, ulcerated, or evolving cutaneous tumor in a PLWH must be biopsied without delay

Examples of HIV-associated cutaneous tumors

Non-Hodgkin cutaneous lymphoma



Epidermoid carcinoma



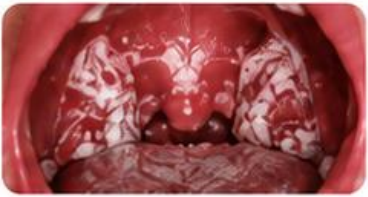
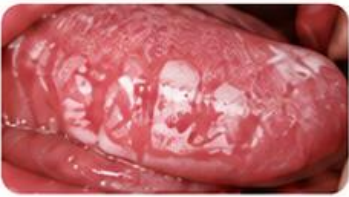
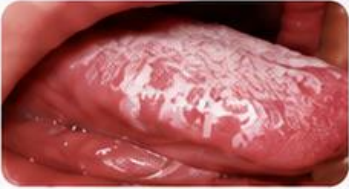
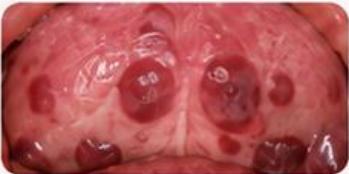
Basocellular carcinoma



KEY MESSAGE

In PLWH, the threshold for biopsy of a cutaneous tumor must be low: the risk of skin cancer is significantly increased compared to the general population.

Oral manifestations during HIV infection

Lesion	Key clinical features	Differential diagnostic element
 Oropharyngeal candidiasis	 • White or erythematous deposits often diffuse • On the oral mucosa and palate	• Detachable on scraping
 Oral hairy leukoplakia	 • Vertical white patches, lateral borders of the tongue	• NOT detachable on scraping (EBV)
 Aphthoid ulcerations	 • Painful erosions, jugal/palatal mucosa	• Rule out herpes, primary HIV infection
 Oral Kaposi sarcoma	 • Violaceous macules/nodules, palate mostly++	• Biopsy + HHV-8/LANA-1



KEY MESSAGE

Systematic examination of the oral cavity is an integral part of the dermatologic examination of HIV+ patients: it often guides the diagnosis and reflects the patient's immune status.

Nail manifestations

Manifestation	Key clinical features	Clinical photograph	Diagnosis & management
 Melanonychia	- Longitudinal bands or diffuse nail pigmentation - May be related to HIV infection itself or secondary to certain antiretroviral treatments, particularly zidovudine		 Clinical assessment  Adjust or reassess treatment if necessary
 Onychomycosis	- Common fungal infections - Often bilateral and extensive - May be resistant to standard treatment		 Mycological examination (KOH preparation, culture)  Prolonged systemic antifungal therapy
 Leukonychia	- Partial or complete whitish discoloration of the nail plate - Reported during HIV infection		 Clinical assessment  Investigate associated causes  Treat the underlying cause whenever possible
 Candidal paronychia	- Painful inflammation of the periungual fold - Often associated with candidiasis of the skin folds - Erythema, edema, sometimes oozing		 Clinical assessment  Investigate associated candidiasis  Topical ± systemic antifungal therapy
 Systematic nail examination	- An integral part of a complete dermatological examination - May provide additional diagnostic clues		 Interpret according to the clinical context and immune status / CD4 count  Guide further investigations when necessary



KEY MESSAGE

Nail examination, often overlooked, is an integral part of the comprehensive dermatological assessment of people living with HIV and may reveal **opportunistic** or **treatment-related manifestations**.

PART 7

Diagnostic approach

From alert signs to HIV serology



ALERT SIGNS

- Skin eruptions
- Adenopathies
- Unexplained weight loss
- Prolonged fever
- Recurrent infections



INITIAL EVALUATION

- Risk assessment
- Clinical examination
- Biological workup
- Clinical staging evaluation



SCREENING TESTS

- Rapid tests
- Combined Ag/Ab tests
- Self-tests
- High sensitivity



SEROLOGIC CONFIRMATION

- Confirmatory test
- Differentiation
- Recent / old infection



ALERT SIGNS

Clinical and biological signs



INITIAL EVALUATION

History, clinical examination, initial workup



SCREENING TESTS

Rapid tests, combined Ag/Ab tests



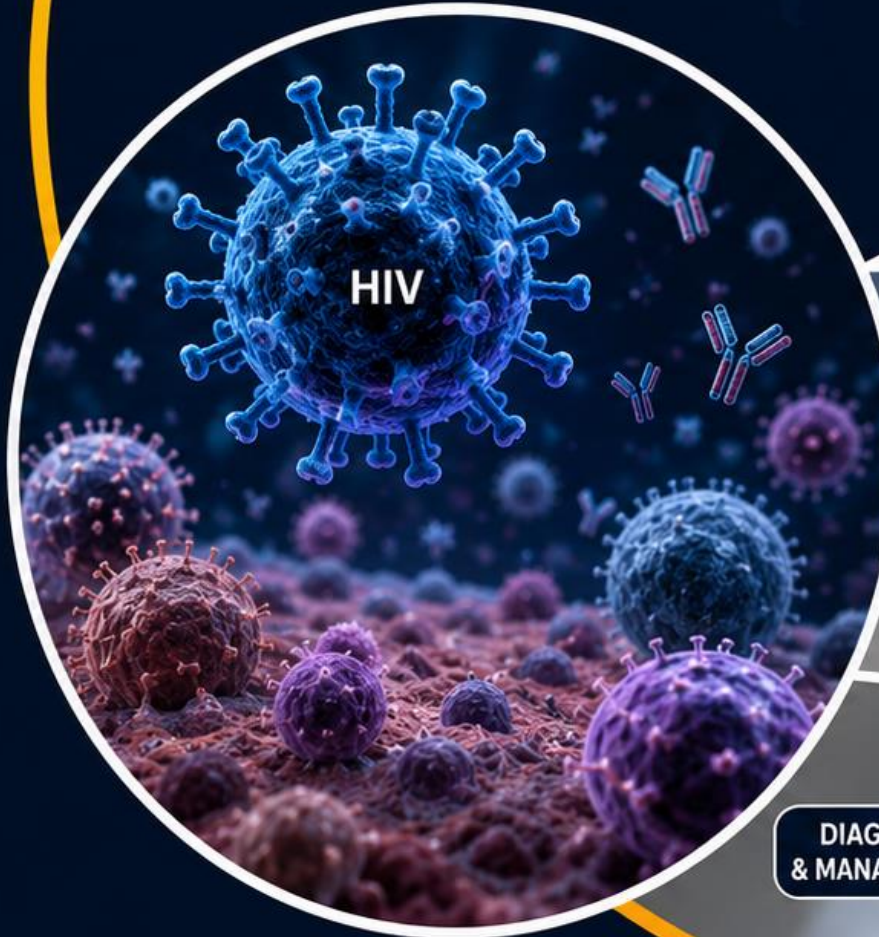
SEROLOGIC CONFIRMATION

Confirmatory tests



INTERPRETATION & FOLLOW-UP

Diagnostic interpretation, management



SKIN SIGNS



BIOLOGICAL WORKUP



RAPID TEST



DIAGNOSIS & MANAGEMENT



Cutaneous signs that should prompt HIV serology

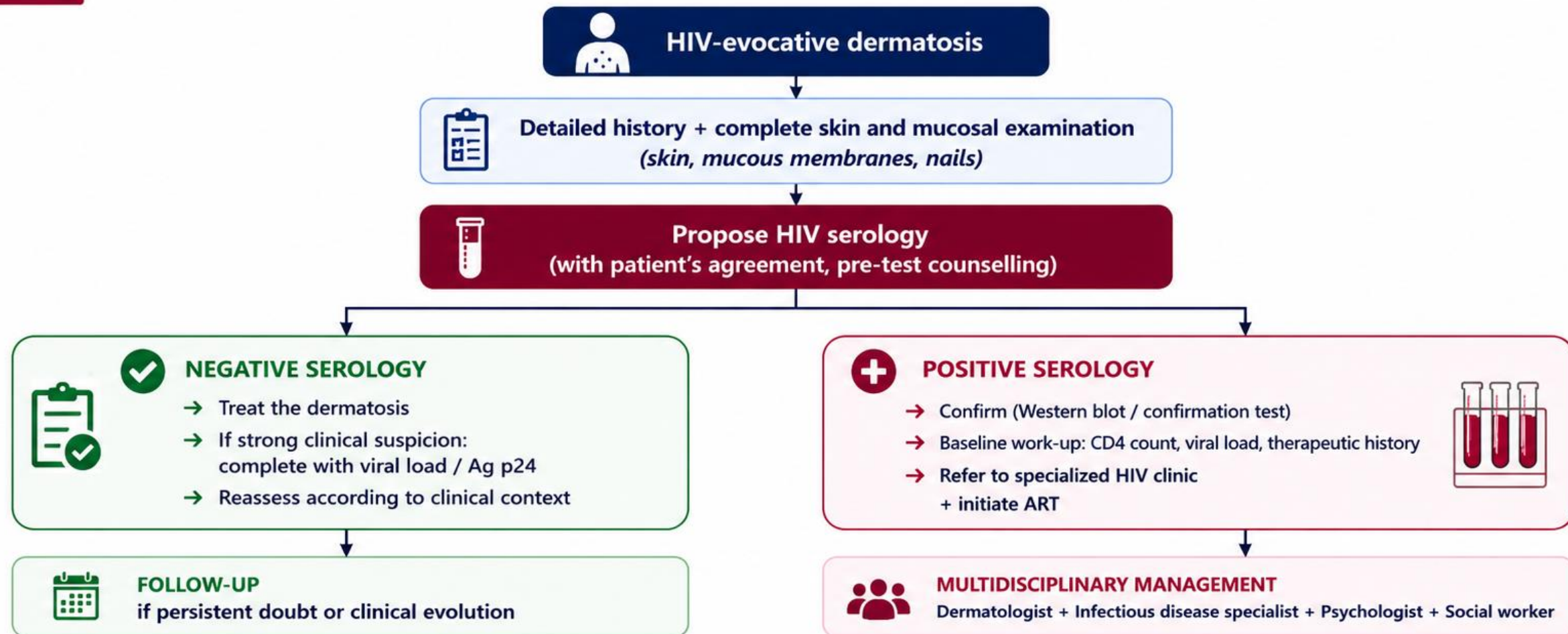
Evocative cutaneous sign	Context element raising suspicion
 Herpes zoster	 In a subject < 50 years old , without another cause of immunodepression
 Extensive molluscum contagiosum	 Numerous, large lesions on the face in an adult
 Severe seborrheic dermatitis	 Widespread, resistant to usual treatment
 Recurrent oral candidiasis	 In the absence of diabetes , prolonged corticosteroid therapy or antibiotics
 Prurigo nodularis / eosinophilic folliculitis	 Unexplained chronic pruritus , excoriated symmetric papules
 Kaposi sarcoma	 Any atypical violaceous vascular lesion
 Chronic herpes (> 1 month)	 persistent or extensive herpetic ulceration
 Oral hairy leukoplakia	 Non-scrapable white plaques on the lateral borders of the tongue
 Extensive dermatophytosis	 Bilateral involvement, resistant to standard antifungal treatment
 Diffuse pruritic hyperkeratosis (Norwegian scabies)	 Diffuse crusted hyperkeratosis, highly pruritic skin



KEY MESSAGE

These ten “sentinel” cutaneous signs should be part of the diagnostic reflex of all medical students: **their recognition enables early screening for HIV.**

Decision tree for an HIV-evocative dermatosis



KEY MESSAGE

Any clinical suspicion must be followed by pre-test counselling and rapid orientation, whatever the result — HIV serology should **never be proposed without accompaniment**.

Complementary investigations



HIV serology (4th generation Ag/Ab combination test):
first-line screening test



Plasma viral load (HIV RNA) and p24 antigen:
useful in the setting of suspected acute/primo-infection
(serological window period)



CD4 lymphocyte count:
assess the degree of immunosuppression
and guide management



Skin biopsy with histological examination
(± HHV-8 immunohistochemistry, specific stains):
essential for any tumoral or atypical lesion



Microbiological tests guided by the clinical context:
HSV/VZV PCR, mycological sampling,
fungal serologies (cryptococcal antigen)



KEY MESSAGE

The diagnostic work-up must follow a structured approach: first, first-line **serological screening**, then **immunovirological** and **histological** assessment guided by the clinical context.

PART 8

Therapeutic management

Antiretroviral therapy and specific treatments



ANTIRETROVIRAL THERAPY (ART)

- Inhibition of viral replication
- Reduction of viral load
- Immune recovery
- Prevention of progression



SPECIFIC TREATMENTS FOR CO-INFECTIONS

- Tuberculosis
- Viral hepatitis
- Opportunistic infections
- Prophylaxis and adapted treatments



GLOBAL MANAGEMENT

- Regular clinical follow-up
- Therapeutic education
- Psychological support
- Nutrition and hygiene
- Adherence to treatment



THERAPEUTIC OBJECTIVES

- Suppress viral replication
- Restore immune function
- Improve quality of life
- Prevent transmission



VIRAL REPLICATION

HIV actively replicates



ART TREATMENT

Inhibition of viral replication



VIRAL LOAD REDUCTION

Decrease in viral load



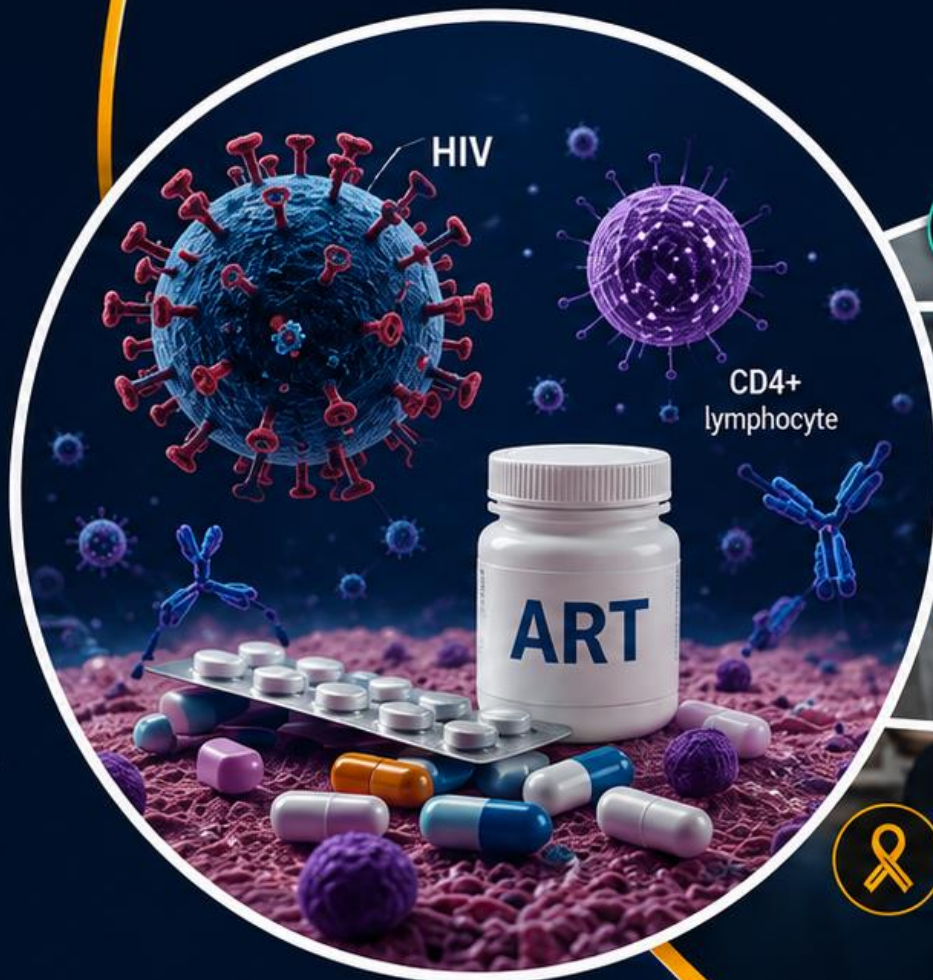
IMMUNE RECOVERY

Increase in CD4+ count



HEALTH & QUALITY OF LIFE

Sustainable infection control



ANTIRETROVIRAL TREATMENT



TREATMENT OF CO-INFECTIONS



CLINICAL FOLLOW-UP AND SUPPORT



PREVENTION AND EDUCATION

Algorithme thérapeutique : trois piliers complémentaires



1 • Traitement antirétroviral



Initiation précoce dès le diagnostic confirmé, quel que soit le taux de CD4 (recommandations OMS/EACS)



Trithérapie de référence adaptée au profil du patient



Restauration immunitaire progressive
→ régression de nombreuses dermatoses



2 • Traitement spécifique de la dermatose



Antifongiques, antiviraux ou antibiotiques ciblés selon l'étiologie



Traitement local (dermocorticoïdes, antiseptiques) en complément



Chirurgie / chimiothérapie pour les formes tumorales étendues (Kaposi, lymphome)



3 • Prévention des infections opportunistes



Prophylaxie primaire selon le taux de CD4 (ex. cotrimoxazole si $CD4 < 200$)



Vaccinations recommandées (HPV, pneumocoque, hépatite B)



Éducation thérapeutique : observance, dépistage des partenaires, suivi dermatologique régulier



MESSAGE CLÉ

Le traitement antirétroviral précoce reste le socle de la prise en charge : il traite la cause et fait **régresser la majorité des dermatoses** associées au VIH.

Dix vignettes cliniques — testez le raisonnement diagnostique

N°	Vignette clinique	Diagnostic attendu	N°	Vignette clinique (à discuter en groupe)	Diagnostic attendu
1	Homme 28 ans, fébrile + énanthème maculo-papuleux du tronc et du visage, suivant 3 semaines après un rapport à risque.	 Primo-infection VIH (syndrome rétroviral aigu)	6	Femme 29 ans, dépôts blanchâtres buccaux détachables au grattage, dysphagie.	 Candidose oropharyngée ± œsophagienne
2	Femme 35 ans, plaques érythémato-squameuses grasses du visage résistantes au traitement habituel.	 Dermite séborrhéique sévère liée au VIH	7	Homme 38 ans, hyperkératose croûteuse diffuse des mains et pieds, peau prurigineuse.	 Gale profuse (norvégienne)
3	Homme 40 ans, nodules violacés multiples des membres inférieurs et lésion du palais.	 Sarcome de Kaposi	8	Homme 34 ans, papulo-pustules très prurigineuses du visage et du tronc, CD4 à 180/mm ³ .	 Folliculite à éosinophiles
4	Homme 32 ans, éruption vésiculeuse douloureuse en bande, hémithorax droit.	 Zona	9	Femme 41 ans, plages blanches linguales non détachables au grattage.	 Leucoplasie orale chevelue
5	Homme 45 ans, papules ombiliquées profondes du visage, CD4 à 80/mm ³ .	 Molluscum contagiosum profus (éliminer cryptococcose)	10	Homme 30 ans, papules excoriées symétriques et très prurigineuses des membres.	 Prurigo du VIH



MESSAGE CLÉ

Face à une **dermatose** évoquant le déficit immunitaire, pensez **VIH**.
L'analyse clinique rigoureuse oriente efficacement le **diagnostic juste**.



Partage
d'expérience



Raisonnement
clinique



Bon diagnostic



Meilleure
prise en charge

Take-Home Messages

1		More than 90% of PLWH will develop a cutaneous or mucosal manifestation during the course of their illness.	
2		Skin may be the first organ to reveal HIV infection.	
3		Zoster before age 50 = systematically propose HIV serology.	
4		Molluscum contagiosum , recurrent candidiasis and disseminated prurigo are sentinel signs.	
5		The severity of dermatoses correlates with the degree of CD4 decline.	CD4 ↓ 
6		Severe seborrheic dermatitis and resistant psoriasis may reveal HIV.	
7		Eosinophilic folliculitis = marker of advanced immunodepression.	
8		Oral ulcer > 1 month = criterion suggestive of AIDS.	

9		Oral hairy leukoplakia : NON-scrapable lesion, must not be confused with candidiasis.	
10		Kaposi sarcoma = hallmark tumor associated with HHV-8.	
11		Bacillary angiomatosis : key differential diagnosis of Kaposi, treatable with antibiotics.	
12		Pruritic papular eruption : highly contagious, pruritic — immediate isolation.	
13		Any tumoral skin lesion in a PLWH must be biopsied without delay.	
14		Early antiretroviral therapy remains the cornerstone of treatment.	
15		Screening must always be accompanied by pre- and post-test counseling .	



TAKE-HOME MESSAGE

*The skin and mucous membranes are **mirrors of the immune system**: every sign matters.*

