

Kaposi Sarcoma: Global Disease Burden, Treatment Disparities, and Barriers to Care

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Abstract

Kaposi sarcoma (KS) is an angioproliferative malignancy caused by Kaposi sarcoma-associated herpesvirus (KSHV/HHV-8). Immunodeficiency due to HIV infection is a major risk factor, and KS remains one of the most common HIV-associated malignancies globally. KS burden of disease is disproportionately concentrated in sub-Saharan Africa (SSA), which accounts for over 70% of incident KS cases worldwide, driven by high HHV-8 seroprevalence and a persistent regional HIV burden. The age-standardized incidence rate is generally higher in males than in females; the highest male rates have been reported in Mozambique and Zambia, whereas the lowest are seen in South Asia, particularly India and Sri Lanka.

In high-income countries, widespread access to antiretroviral therapy (ART) has markedly reduced KS incidence and improved outcomes, transforming HIV-associated KS into a more manageable condition. In many low- and middle-income countries (LMICs) in SSA, patients frequently present with advanced disease, severe immunosuppression, and uncontrolled viremia. Contemporary data from East Africa suggest that one-year mortality for HIV-associated KS remains above 40%.

Treatment of advanced HIV-associated KS is based on ART combined with systemic chemotherapy, most commonly pegylated liposomal doxorubicin or paclitaxel. In LMICs, access to preferred agents is limited by cost, supply constraints, inadequate access to timely histopathologic diagnosis, infusion infrastructure, and trained personnel. Where liposomal anthracyclines are unavailable, paclitaxel has emerged as a

practical and effective alternative. Persistent barriers include delayed diagnosis, incomplete ART coverage and retention, stigma, and vulnerability to disruptions in international HIV funding and public health expenditures. Preventing HIV infection, promoting earlier diagnosis, expanding ART access, strengthening cancer care infrastructure, and improving chemotherapy availability are critical to improving outcomes. Emerging approaches, including telemedicine, digital decision support, and novel point-of-care diagnostic tools, may help extend expertise but will complement rather than replace sustained investment in health systems and workforce development.

Take Home Messages

1. Kaposi sarcoma (KS) remains a major cause of cancer morbidity and mortality in sub-Saharan Africa (SSA), driven by high HHV-8 seroprevalence, persistent HIV burden, late presentation, and gaps in access to antiretroviral therapy (ART) and oncology care.
2. ART is the cornerstone of HIV-associated KS management. In patients with advanced disease, adding systemic chemotherapy improves tumor response and clinical outcomes compared with ART alone.
3. Paclitaxel is a practical and effective chemotherapy option in resource-limited settings, particularly where pegylated liposomal doxorubicin is unavailable. Currently, the World Health Organization (WHO) endorses either paclitaxel or pegylated liposomal doxorubicin for people living with HIV and KS, and both medicines are included on the WHO Model List of Essential Medicines.
4. Recent disruptions in US global health funding affecting the President's Emergency Plan for AIDS Relief (PEPFAR), the US Agency for International Development (USAID), and related health programs are reducing public health capacity, threatening continuity of HIV treatment and prevention services, and increasing risks to commodity availability, potentially undermining KS control in low- and middle-income countries (LMICs).
5. Emerging tools—including point-of-care (POC) diagnostics, artificial intelligence-assisted skin pathology imaging, and expanded telemedicine—may improve access to expertise, but they will complement rather than replace sustained investment in health systems, workforce, and drug availability.

1. Introduction

Cancer is a major contributor to the global disease burden and is currently the second leading cause of death worldwide after cardiovascular disease.¹ The Global Burden of Disease (GBD) Collaborators estimate continued increases in cancer incidence and mortality through 2050, with a disproportionate rise in burden in low- and middle-income countries (LMICs), driven by demographic shifts and persis-

tent inequities in access to prevention, diagnosis, and treatment.

Kaposi sarcoma (KS) is an angioproliferative malignancy caused by KS-associated herpesvirus (KSHV/HHV-8). The four widely recognized epidemiologic forms of KS are classic KS, which typically occurs in older men of Mediterranean origin and tends to follow a relatively indolent course; endemic KS, which occurs in persons in sub-Saharan Africa (SSA) and often involves lymph nodes; transplantation-associated KS; and epidemic HIV-associated KS.²⁻⁴ A fifth category, consisting of a nonepidemic indolent variant affecting HIV-negative men who have sex with men (MSM), has also been described in numerous case reports.^{3,4} (Table 1). While HHV-8 infection is necessary, it is not sufficient for disease development; immunosuppression—most notably due to HIV infection—is a critical cofactor in the most common type of KS (i.e., HIV-associated KS).

People living with HIV (PLHIV) have a substantially increased risk of several infection-related cancers compared with the general population. In 2022, an estimated 81,300 of 19 million cancer cases worldwide (0.4%) were attributable to HIV, driven largely by cervical cancer, KS, and intermediate- and high- grade B-cell non-Hodgkin lymphoma, with marked regional variation.⁵

The epidemiology of KS reflects the intersection of KSHV seroprevalence and HIV burden. Although KS incidence has declined substantially in high-income countries (HICs) following the widespread introduction of antiretroviral therapy (ART), it remains a major public health challenge in SSA. In 2020, Africa accounted for approximately 73% (25,010 of 34,270) of global KS incidence and about 87% (13,066 of 15,086) of KS-related deaths, with the highest burden observed in eastern and southern Africa.⁶ In contrast, KS has become relatively uncommon in HICs, where outcomes have improved substantially with ART and when needed, systemic chemotherapy. This stark divergence in disease burden and outcomes highlights marked global inequities. In HICs, early diagnosis and access to ART and systemic therapy have transformed KS into a largely manageable condition. In many LMICs, however, patients frequently present with advanced disease, severe immunosuppression, and limited access to timely diagnosis and effective treatment, contributing to substantially

Table 1: Clinical Categories of KS

Epidemiologic Type of KS	Typical Population	Clinical Features and Course
Classic KS (sporadic KS)	Older men of Mediterranean, Eastern European, or Middle Eastern ancestry; usually presents in the sixth to seventh decade of life	Predominantly involves the skin of the distal lower extremities. Visceral involvement is uncommon. Usually indolent and slowly progressive.
Endemic KS	Individuals in SSA, including both adult and pediatric forms	Adult form may resemble classic KS, with lower-extremity skin lesions and a variable course. Pediatric disease often presents with lymphadenopathy, minimal skin disease, and may be aggressive or rapidly fatal.
Epidemic KS / HIV-associated KS	PLHIV; initially recognized among MSM in HICs but common among men, women, and children in high-prevalence regions such as SSA	Frequently involves skin, oral cavity, gastrointestinal tract, lymph nodes, and lungs. May be aggressive without ART. Incidence declined substantially after widespread ART in HICs, but KS remains a major cancer burden in SSA
Iatrogenic / Transplant-associated KS	Patients receiving immunosuppressive therapy, particularly renal transplant recipients and other solid-organ transplant recipients	Usually cutaneous but may involve mucosal or visceral sites. May regress with reduction or modification of immunosuppression.
Nonepidemic KS in HIV-negative MSM (proposed fifth category)	HIV-negative MSM without overt immunosuppression	Primarily cutaneous disease; visceral involvement is uncommon. Typically, indolent and increasingly recognized as a possible distinct epidemiologic variant.

Abbreviations - SSA: sub-Saharan Africa; KS: Kaposi sarcoma; PLHIV: people living with HIV; MSM: men who have sex with men; HICs: high income countries; ART: antiretroviral treatment.

higher morbidity and mortality. Structural barriers—including constrained oncology infrastructure, limited access to timely histopathologic diagnosis, workforce shortages, and reliance on international HIV funding mechanisms—continue to impede optimal care delivery.

This review aims to (1) describe the global epidemiology and burden of HIV-associated KS; (2) outline its pathophysiology and key clinical features; (3) summarize contemporary treatment strategies across resource settings; and (4) analyze the health system, socioeconomic, and policy barriers that perpetuate disparities in outcomes, while highlight-

ing emerging approaches that may help mitigate these inequities.

2. Epidemiology and Global Disease Burden

KS was first described by the Hungarian dermatologist Moritz Kaposi in 1872 in a German article titled ‘Idiopathic Multiple Pigmented Sarcoma of the Skin.’^{8,9} In 1981, reports of KS occurring in MSM in the US brought KS to the forefront of the emerging AIDS epidemic, and KS was soon recognized by the US Centers for Disease Control and Prevention (CDC) as an AIDS-defining malignancy.

nancy.^{10,11} During the early HIV epidemic, KS incidence increased dramatically. Among PLHIV in the US, the standardized incidence ratio (SIR) for KS exceeded 50,000 in the 1980s.¹² With the widespread adoption of effective ART and improved immune reconstitution, KS incidence has since declined, although relative risk remains markedly elevated; the SIR decreased from 358.98 in 2010–2014 to 213.87 in 2015–2019.¹²

Overall, HIV-associated KS epidemiology reflects the intersection of HIV burden, KSHV prevalence, healthcare-system capacity, and structural inequities, resulting in marked global and within-country disparities (Figures 1 and 2). The GBD collaborators estimated that there were 40 million PLHIV globally in 2021 with about 73% residing in SSA.¹³ HHV-8 prevalence is highest in SSA, intermediate in the Mediterranean and parts of Latin America, and lower in the general population in northern Europe, North America, and most of Asia.¹⁴ In the US, MSM and PLHIV are at increased risk for HHV-8 infection.¹⁵

Analysis of GLOBOCAN 2020 data from 185 countries demonstrated that Africa bears the overwhelming burden of KS worldwide, accounting for approximately 73% of incident cases.⁶ Incidence rates were highest in eastern and southern African countries such as Mozambique and Zambia and lowest in South Asia. Globally, KS predominantly affects males, and mortality patterns generally parallel incidence trends. Globally, 15,086 KS deaths were estimated in 2020, corresponding to an age-standardized mortality rate (ASMR) of 0.18 per 100,000 people in 2020 with substantially higher mortality rates observed in Africa.⁶

KS incidence in SSA and Latin America is likely under-ascertained because of the limited coverage of population-based cancer registries in the region.¹⁶ When comparing the periods 2001–2010 and 2011–2016, KS incidence showed marked inter-registry variability, with a crude average decline of approximately 6% across SSA.¹⁷ Post-2015 data from East Africa, during the WHO "Treat All" era—when ART was recommended for all PLHIV regardless of CD4+ cell count or clinical stage—showed persistently poor outcomes, with mortality exceeding 40% at one year after KS diagnosis.¹⁸ By comparison, US data demonstrated a more significant 34% reduction in KS incidence between 2000 and 2014 among PLHIV.¹⁹ In the US, five-year mortality

after KS diagnosis among PLHIV declined from 43.1% for those diagnosed in 2000–2002 to 29.5% in 2012–2015.²⁰ In Europe, KS incidence among patients younger than 65-years-old declined by approximately 40% in the early ART era, while five-year relative survival improved and exceeded 80% across age groups by 2005–2007.²¹ The incidence of KS among PLHIV in Latin America decreased from 55.1 per 1000 person-years in 2000 to 3.0 per 1000 person-years in 2017, with most of the decline occurring in the early ART era and rates stabilizing thereafter; five-year mortality after KS diagnosis remained stable at approximately 25%.²²

Of note, disparities in KS burden are not unique to LMICs and are also observed within HICs. Although KS incidence declined overall in the US during the modern ART era, important racial and geographic disparities persisted. In Surveillance, Epidemiology, and End Results (SEER)-based analyses of US men younger than 55 years from 2000 to 2013, KS incidence decreased significantly among White men, whereas rates among African American men declined less markedly overall and increased significantly among African American men in the South.²³ More recent CDC WONDER data from 1999 to 2020 showed 27,886 KS cases and 4,380 KS-related deaths in the US. Overall, the ASIR was 0.99 per 100,000 in men versus 0.10 in women, and the ASMR was 0.16 versus 0.01, respectively. Black men had the highest burden, with an ASIR rate of 2.23 per 100,000 and an ASMR of 0.40 per 100,000, both significantly higher than the corresponding rates in White men at 0.79 and 0.13 per 100,000, respectively.²⁴ Racial differences in mortality following KS may partly reflect disparities in linkage to and engagement in care, as well as later presentation to health facilities with more advanced disease among Black PLHIV.²⁰

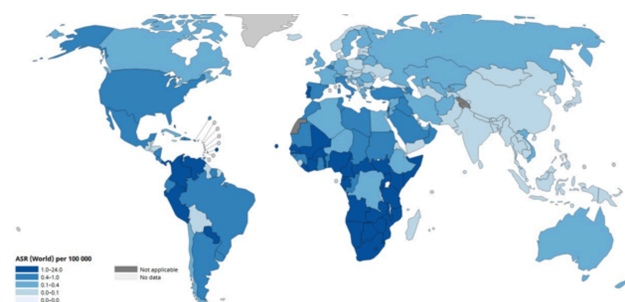


Figure 1. Global age-standardized incidence rates of Kaposi sarcoma among adults aged ≥ 20 years, 2022

Table 2: Staging Classification for HIV-Associated KS

ACTG Category	Good Risk (All of the following)	Poor Risk (Any of the following)
Tumor extent	T0: KS confined to skin and/or lymph nodes, and/or minimal (non-nodular) oral KS	T1: Ulcerated KS, KS-associated edema, nodular oral KS, or KS involving any non-nodal visceral organ
Immune status	I0: CD4+ T-cell count ≥ 150 cells/ μ L	I1: CD4+ T-cell count < 150 cells/ μ L
Systemic illness	S0: KPS ≥ 70 No B symptoms (unexplained fever, weight loss, night sweats, or diarrhea) No history of opportunistic infections including no oropharyngeal candidiasis	S1: KPS < 70 Presence of B symptoms History of opportunistic infections and/or oropharyngeal candidiasis Other HIV-related illness (e.g., neurologic disease, lymphoma)

Abbreviations - KS: Kaposi sarcoma; KPS: Karnofsky performance status.

Adapted from: International Agency for Research on Cancer. *Cancer Today. GLOBOCAN 2022, World Health Organization, 2024*(7)

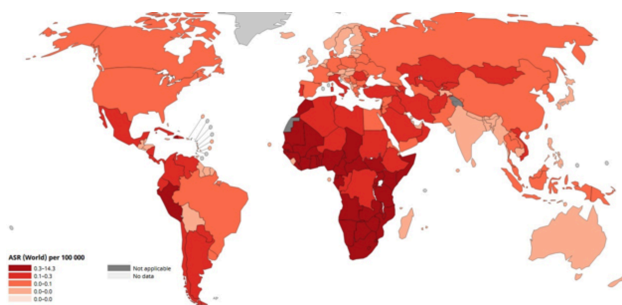


Figure 2. Global age-standardized mortality rates of Kaposi sarcoma among adults aged ≥ 20 years, 2022

Adapted from: International Agency for Research on Cancer. *Cancer Today. GLOBOCAN 2022, World Health Organization, 2024*(7)

3. Pathophysiology and Clinical Spectrum

3.1 Mechanisms/Pathophysiology

In 1994, Chang et al. identified the etiologic agent of KS as a novel human herpesvirus, HHV-8/KSHV, using representational difference analysis of DNA sequences present in KS tissue in patients with AIDS.²⁵ This double-stranded DNA virus can infect several cell types, including B-cells,

monocytes and endothelial cells. Like other herpesviruses, HHV-8 has latent and lytic replication programs. During latency, only a limited set of viral genes is expressed including ORFK12, ORF71, ORF72, ORF73 (latency-associated nuclear antigen [LANA]), ORFK10.5 and several viral microRNAs. In contrast, lytic replication involves expression of the full viral gene repertoire, production and release of progeny virions, and death of the infected cells.¹⁴ The switch from latency to lytic replication is mediated by the replication and transcription activator (RTA), encoded by ORF50. A variety of physiologic signals can activate RTA including hypoxia, oxidative stress, certain cytokines and chemical stimuli. HHV-8 encodes genes that evade innate cellular defenses like apoptosis and cell-cycle arrest, thereby subverting antiviral immune responses and promoting proliferation of infected cells. Some viral genes also mimic human proteins with angiogenic and inflammatory functions. Although these adaptations primarily enhance viral survival, they can also drive the development of HHV-8-associated tumors and other proliferative disorders.

KS lesions are characterized histologically by proliferation of spindle cells of endothelial origin, with abnormal vascularity, inflammatory infiltrates, and variable fibrosis. Extravasated red blood cells and hemosiderin deposition contribute to the characteristic violaceous appearance of the lesions.² The

Table 3: Response Evaluation for HIV-Associated KS

Response Category	ACTG Criteria for HIV-associated KS
Complete response (CR)	No detectable residual disease and no tumor-related edema for at least 4 weeks.
Partial response (PR)	No new mucocutaneous lesions, visceral disease, or worsening tumor-related edema. Existing sites show a $\geq 50\%$ reduction in one or more of the following: (1) number of lesions, (2) form of lesions (flattening of raised lesions), or (3) sum of the products of the largest perpendicular diameters of five measurable lesions. Note: If residual tumor-related edema or effusion is present despite otherwise meeting CR criteria, the response is classified as PR.
Stable disease (SD)	Does not meet criteria for complete response, partial response, or progressive disease.
Progressive disease (PD)	Increase of $>25\%$ in the size of existing lesions and/or the number of existing lesions that have become more nodular or plaque-like, or development of new sites of disease. New or increasing tumor-related edema or effusion

Abbreviations - KS: Kaposi sarcoma; ACTG: AIDS Clinical Trial Group.

spindle cells are infected with KSHV and express LANA, a viral protein that helps tether KSHV episomes to host chromatin. HHV-8 also encodes multiple viral proteins that promote cell proliferation, cytokine production, angiogenesis, and immune evasion, thereby contributing to KS pathogenesis.

Most primary infections by HHV-8 appear to be asymptomatic. Beyond KS, HHV-8 is linked to several lymphoproliferative and inflammatory disorders, including plasmablastic Multicentric Castleman Disease (MCD), primary effusion lymphoma (PEL), HHV-8-positive diffuse large B-cell lymphoma, and KSHV-associated inflammatory cytokine syndrome (KICS).

3.2 Transmission of HHV-8

The exact routes of HHV-8 transmission remain incompletely defined. HHV-8 has been detected in both saliva and genital secretions from infected individuals, but saliva is thought to be the predominant route of transmission. In HICs, transmission appears to occur mainly through sexual contact

among MSM, with higher risk associated with a greater number of sexual partners and receptive anal intercourse. In African countries, nonsexual transmission, particularly childhood household transmission through saliva, and possibly perinatal transmission, may also play an important role.^{3,15,26}

3.3 Clinical Spectrum of HIV-KS

The epidemiologic subtypes of KS show considerable clinical overlap, and HIV-associated KS can present along a broad spectrum ranging from indolent to aggressive, disseminated disease. KS typically presents on the skin or mucosa as isolated or multiple violaceous, non-blanching macules, plaques, papules, or nodules. Lesions are usually painless, although they may be tender when located in pressure-bearing areas; they may also ulcerate, become verrucous or exophytic, or invade adjacent tissues.⁴ Ocular KS can affect the eyelid or conjunctiva. KS can cause marked lymphedema through multifactorial mechanisms, including lymphatic obstruction, regional lymphadenopathy,

increased lymphatic permeability, impaired lymphatic drainage, and HHV-8-driven endothelial proliferation that may occlude lymphatic lumens. Persistent lower-extremity lymphedema can be complicated by reduced mobility, contractures, diffuse serous drainage, skin ulceration, and recurrent cellulitis.¹⁵

Extracutaneous involvement may affect visceral organs and, less commonly, brain, heart and bone. Gastrointestinal KS is often asymptomatic but may present with pain, bleeding, dysphagia or feeding difficulty, diarrhea, obstruction, or malabsorption.³ Pulmonary KS may present with dyspnea, cough, hemoptysis, pleural effusions, or reduced functional capacity.

With advanced immunosuppression, HIV-associated KS is more common, more aggressive, and more likely to involve visceral organs, mucosal sites, and lymph nodes, and to be associated with lymphedema. However, HIV-associated KS can also occur in people with well-controlled HIV infection, including those with preserved CD4+ cell counts and suppressed viral loads.²⁷ In such cases, which often occur at older ages, KS is thought to reflect accelerated immunosenescence in PLHIV and typically presents with more limited disease with an indolent course resembling classic KS compared with the more aggressive HIV-KS occurring in the setting of advanced immunosuppression and uncontrolled HIV viremia.²⁸

The differential diagnosis for KS is broad and depends on lesion morphology. It includes pyogenic granuloma, bacillary angiomatosis, dermatofibroma, cutaneous lymphoma and pseudolymphoma, spindle-cell hemangioma, other vascular neoplasms or malformations, nonmelanoma skin cancers, vasculitis or other causes of purpura, and inflammatory dermatoses such as lichen planus.⁴

By omitting a skin biopsy, the clinician runs the risk of inaccurately identifying KS. In a study of 1,106 biopsies from two large HIV care sites in Kenya and Uganda, clinical suspicion for KS had a positive predictive value of only 77% compared with US-based dermatopathological review as a reference standard.²⁹ Per the US National Comprehensive Cancer Network (NCCN) guidelines, the diagnosis of KS should be confirmed by biopsy with histopathologic review, including immunohistochemistry for HHV-8/KSHV LANA-1 when

available.³⁰ Initial evaluation should include a comprehensive history and physical examination, with complete skin, oral, and lymph node assessment, HIV testing, complete blood count, comprehensive metabolic panel, and photographic documentation of lesions. Additional evaluation should be guided by symptoms and disease extent: chest imaging and bronchoscopy should be considered for suspected pulmonary involvement, while abdominal/pelvic computerized tomography (CT) or magnetic resonance imaging (MRI) and endoscopic evaluation are appropriate for gastrointestinal symptoms or positive fecal occult blood testing. In cases with concern for KICS, MCD or HHV-8-associated lymphoma, further evaluation may include advanced imaging (e.g., fluorodeoxyglucose positron emission computerized tomography [FDG-PET-CT]), and laboratory studies such as C-reactive protein, HHV-8 viral load, serum protein electrophoresis, and cytokine markers (e.g., IL-6, IL-10). As per NCCN Harmonized Guidelines for SSA, evaluation should be adapted to resource availability: access to immunohistochemistry may be limited, tuberculosis evaluation is important in endemic settings, and advanced diagnostic modalities such as endoscopy or FDG-PET-CT may not be readily available.³¹

Currently there is no standardized staging system for KS within the American Joint Committee on Cancer (AJCC) Tumor Node Metastases (TNM) framework. Instead, criteria developed by the AIDS Clinical Trials Group (ACTG) are commonly used to stage HIV-associated KS and assess response to therapy (Tables 1 and 2).^{30,32,33}

4. Treatment

4.1 Antiretroviral Therapy

ART is foundational in HIV-associated KS therapy and all PLHIV should be offered ART. Integrase strand inhibitor-based ART remains the preferred choice for initial treatments per current WHO, European AIDS Clinical Society and International Antiviral Society guidelines.³⁴⁻³⁶ Tumor regression or resolution occurs over several months in most patients with HIV-associated KS receiving ART, particularly those with limited disease and who are ART-naïve.^{15,37} In PLHIV with limited cutaneous KS, when lesions are asymptomatic and cosmetically acceptable, ART alone may suffice as first-line therapy.

In some patients, KS may initially develop or worsen within weeks to months after ART initiation due to immune reconstitution inflammatory syndrome (IRIS). Therefore, systemic KS-directed therapy should be initiated as soon as possible in PLHIV with symptomatic or advanced KS, along with ART.³⁰ The risk of IRIS is higher in PLHIV with low CD4+ cell counts and high HIV viral loads. ART should not be delayed or discontinued unless life-threatening IRIS develops. Glucocorticoids, commonly used for other forms of IRIS, may exacerbate KS and should be avoided when possible, except in life-threatening conditions such as anaphylaxis.³⁸

4.2 Local Therapy

For limited cutaneous KS that is symptomatic or cosmetically bothersome, lesion-directed local therapy may be considered for disease control. Options include topical agents such as alitretinoin 0.1% gel or imiquimod 5% cream, radiation therapy (RT), cryotherapy, local excision, and intralesional chemotherapy.³⁰ KS is a radiosensitive tumor, but given the sensitivity of mucous membranes, irradiation of the conjunctiva and the upper aerodigestive tract should be performed with caution. The lowest effective dose of RT is employed to minimize long-term complications, including worsening lymphedema, delayed wound healing, fibrosis, and, rarely, secondary malignancy.³⁹ In advanced cutaneous KS, radiation is generally reserved for palliation or short-term local disease control when systemic therapy is not feasible or while awaiting initiation of systemic therapy.

Cryotherapy and local excision may be useful for selected small lesions but require appropriate procedural expertise and may be less feasible in resource-limited settings.³¹ Intralesional vinblastine or bleomycin may also be considered for small symptomatic lesions in selected patients. A recent phase I trial found intralesional nivolumab, an immune checkpoint inhibitor, to be safe and well tolerated in limited cutaneous KS, with preliminary evidence of tumor regression.⁴⁰

4.3 Systemic Therapy

Systemic therapy, in combination with ART, is a mainstay approach for people with advanced HIV-associated KS.³⁰ It is also appropriate for patients with limited but symptomatic or cosmetically un-

acceptable cutaneous disease that is refractory to local therapy.

The preferred first-line systemic therapy is pegylated liposomal doxorubicin (PLD).³⁰ In a randomized study comparing PLD with bleomycin and vincristine (BV), PLD demonstrated superior response rates: 58.7% versus 23.3%.⁴¹ BV was associated with a higher incidence of peripheral neuropathy, and patients receiving BV were more likely to discontinue treatment early because of adverse events. PLD was more commonly associated with neutropenia. In a prospective, single-arm observational study in Mozambique, PLD was evaluated as first-line chemotherapy in 116 participants with HIV-associated KS. The overall response rate was 80%, including 13% who achieved complete remission.⁴²

An alternative first-line systemic therapy is paclitaxel.³⁰ In a randomized trial comparing paclitaxel with PLD, response rates and overall clinical outcomes were similar between the two groups. However, the overall incidence of grade 3 or higher toxicity was somewhat greater in the paclitaxel arm, particularly neutropenia. Alopecia and sensory neuropathy were also more common with paclitaxel compared with PLD.⁴³

The US National Cancer Institute sponsored Consortium for Advancing Management and Prevention of Cancer in People with HIV (AMC) enrolled 334 participants with advanced KS between 2013 and 2018 to receive BV, oral etoposide, or intravenous paclitaxel, each given with ART.⁴⁴ Paclitaxel plus ART was superior to both comparator arms, supporting its use for advanced HIV-associated KS in resource-limited settings. The study had initially intended to use PLD as the active control, but this was not pursued because of a worldwide shortage. At the time, paclitaxel—not PLD—was included in the WHO Model List of Essential Medicines. In 2023, PLD was added to the WHO list, given its efficacy in treating KS.⁴⁵

A subsequent cost-effectiveness analysis from Kenya found that paclitaxel would substantially increase life expectancy compared with BV and would be cost-effective for advanced KS. PLD would further improve survival by an estimated 0.6 years per person compared with paclitaxel but would be cost-effective only if its price was reduced by 44%.⁴⁶

When preferred regimens are unavailable, other recommended systemic therapy options for KS in SSA include BV, doxorubicin plus BV (ABV), and vincristine alone.³¹

For relapsed or refractory KS, treatment options include repeating the first-line regimen if it was previously well tolerated and produced a durable response or switching to an alternative first-line systemic therapy. For patients with further progression despite paclitaxel or PLD, NCCN guidelines recommend immunotherapy agents such as nivolumab or pembrolizumab, or immunomodulatory agents such as pomalidomide.³⁰ Other therapies that may be considered for treatment of refractory KS include bortezomib, etoposide, gemcitabine, imatinib, lenalidomide, and vinorelbine. In select patients, best supportive care may also be appropriate.

Immune checkpoint inhibitors are safe when employed for treatment of advanced cancers in PLHIV.⁴⁷ A meta-analysis of five studies evaluating PD-1/PD-L1 inhibitors specifically in KS reported a pooled objective response rate of 61%, with 17% of participants achieving a complete response. The three most common adverse events across studies were pruritus, fatigue, and arthralgia.⁴⁸ However, high costs and infrastructure limitations are barriers to using such therapies in resource constrained settings. The AMC is exploring the promising role of systemic low-dose immunotherapy for advanced KS in LMICs.

Abemaciclib, an oral cyclin-dependent kinase 4/6 inhibitor currently approved by the US Food and Drug Administration for breast cancer, has shown promising activity in a phase I/II trial in KS. Among 31 evaluable participants who received more than two cycles, 77% achieved a partial response.⁴⁹ Common adverse events included diarrhea, increased serum creatinine, and neutropenia. If made available through compassionate-use or expanded access programs, abemaciclib may represent a potential therapeutic option for patients with KS who have limited treatment options.

5. Barriers to Care in LMICs

Barriers to KS care in LMICs are multifactorial and extend beyond chemotherapy availability alone. Delayed HIV diagnosis, limited pathology access, workforce shortages, fragmented referral systems,

transportation barriers, stigma, and dependence on external funding mechanisms collectively contribute to delayed diagnosis and poor outcomes.

5.1 Suboptimal ART Coverage and Adherence

Of the estimated 40.8 million PLHIV globally in 2024, 13% remained unaware of their HIV status and 23% were not receiving ART. In the WHO African Region, of the approximately 26.3 PLHIV, 21.7 million were receiving ART, leaving nearly 4.6 million untreated. Among all PLHIV in the African Region, more than one-fifth remained without viral suppression and therefore vulnerable to KS and other HIV-related complications.⁵⁰ Barriers to ART adherence in SSA are multifactorial, including individual factors such as mental health conditions and substance use disorders, social barriers including stigma and intimate partner violence, and economic constraints such as food insecurity, transportation costs and income instability. Structural barriers, including fragmented health systems, limited clinic accessibility, and drug stockouts, are common and were often worsened by the COVID-19 pandemic, which disrupted service delivery and exacerbated socioeconomic vulnerabilities.⁵¹ These challenges are compounded by emerging issues such as HIV drug resistance and declining political commitment, which risk reversing prior gains.⁵²

5.2 Diagnostic Limitations

Given the morphologic variants of KS and the many conditions that can mimic these lesions, visual impression alone cannot be relied upon for diagnosis. In an East African study, clinical suspicion alone had only a 77% positive predictive value for KS diagnosis, and alternative diagnoses included warts, dermatofibroma, squamous cell carcinoma, lymphoma, cryptococcosis, bacillary angiomatosis, psoriasis, and sarcoidosis.²⁹ It is essential for the clinician to biopsy a representative lesion and obtain histopathologic confirmation when contemplating treatment of KS.

In resource-limited settings, task-shifting of punch biopsies from surgeons to nurses, other physicians, clinical officers and technicians is feasible, although shortages of trained dermatopathologists and limited availability of LANA-1 immunohistochemical staining continue to pose major diagnostic challenges.⁵³ A qualitative study on diagnostic delays

for KS in Kenya identified patient-level factors such as limited KS awareness, lack of strong social supports, and the use of alternative folk treatments as significant barriers to care. Additional delays in care were attributed to patient fear of cancer, biopsy, or amputation, and fear that others may learn of their HIV diagnosis. Also highlighted in this study were patients who experienced health-system barriers including prior negative healthcare encounters, misdiagnosis, incomplete physical examination, delayed referral, and limited biopsy availability, and financial constraints.⁵⁴

5.3 Cancer Care Infrastructure

Cancer infrastructure deficits in LMICs create barriers to KS care across the continuum, from diagnosis to treatment, contributing to late-stage presentation, limited access to effective chemotherapy, loss to follow-up, and poor survival. Cancer registry coverage is often limited, and shortages of healthcare personnel—including oncologists, pathologists, and lymphedema specialists—as well as limited diagnostic imaging, RT, cancer facilities, and infusion capacity further constrain care delivery.^{55,56} A 2018 global survey of the clinical oncology workforce found severe oncology workforce shortages in Africa. An extreme shortage, defined as more than 1,000 incident cancers per clinical oncologist, was present in 25 African countries. In addition, seven African countries had no clinical oncologist available to care for patients with cancer.⁵⁷ Safe storage, preparation, and administration of chemotherapy agents such as paclitaxel for KS require trained personnel, equipment, monitoring, and supportive medications, including hypersensitivity premedication, which may not be reliably available in routine LMIC clinical practice.⁴⁴ In a community-based sample of patients diagnosed with KS across five SSA countries, almost half were lost to follow-up by two years.⁵⁸ In Kenya, nearly half of patients meeting criteria for systemic therapy had not received chemotherapy by one estimation.⁵⁹ In a cross-sectional survey of patients and caregivers across nine SSA countries, major barriers to chemotherapy included accessibility barriers, particularly transportation costs; affordability barriers, including treatment costs and patient time away from home; and acceptability barriers, including limited knowledge or awareness and fear of treatment.⁶⁰

5.4 Stigma

Stigma remains a major barrier to timely diagnosis, treatment engagement, and retention in care for patients with HIV-associated KS in SSA.⁶¹ Patients with HIV-associated KS may experience particularly severe stigmatization because the disease combines three highly visible and stigmatized conditions: HIV, cancer, and disfiguring skin lesions. Intersectional stigma may result in labeling, stereotyping, social isolation, fear, and discrimination within both communities and healthcare settings, contributing to delays in presentation and interruptions in care.⁶²

5.5 Threatened International Funding

Substantial progress in the global HIV response has been driven by donor financing, with many high-HIV-burden countries relying on international funding for essential HIV prevention, testing, and treatment services. According to UNAIDS Financial Dashboards, approximately 40% of all HIV funding in LMICs has come from donors since 2015. The US has historically provided the largest share of global HIV financing, with 92% of US contributions delivered bilaterally through the President's Emergency Plan for AIDS Relief (PEPFAR). Since its creation in 2003 through 2024, PEPFAR has saved more than 26 million lives and averted almost 5 million new HIV infections by investing in critical HIV prevention, treatment, care, and support programs in 55 countries.⁶³

In January 2025, the US government issued an executive order pausing foreign development assistance for a 90-day review, with subsequent stop-work orders affecting many foreign aid programs, including HIV-related services. USAID, founded in 1961 and the world's largest funding agency for humanitarian and development aid, was effectively dismantled, with remaining foreign assistance functions transferred to the State Department as of July 2025. The US Department of Health and Human Services announced that the country finalized its withdrawal from the WHO in January 2026.⁶⁴

A modeling study estimated that unmitigated international funding reductions, including discontinued PEPFAR support, could result in an additional 4.43–10.75 million new HIV infections and 0.77–2.93 million HIV-related deaths between 2025 and 2030 compared with the status quo.⁶⁵ Another

modeling study evaluating steep USAID funding cuts and the potential dissolution of the agency predicted more than 14 million additional deaths by 2030, averaging more than 2.4 million deaths annually.⁶⁶ The US is the largest financial contributor to WHO, and withdrawal of US support will negatively affect cancer care in Africa, including access to medicines, emotional support programs, and cancer prevention initiatives.⁶⁷

The US State Department released the *America First Global Health Strategy* in September 2025, reaffirming its commitment to HIV care through PEPFAR while expressing concerns that global health programs had become inefficient, wasteful, and overly dependent on external aid among recipient countries.⁶⁸ The full repercussions of this policy shift and funding disruptions remain uncertain and could be substantial unless funding gaps are offset through alternative international donors or increased domestic government investment.⁶⁹

A planned AMC clinical trial looking at PLD versus paclitaxel in resource constrained settings was recently suspended.⁷⁰ The pharmaceutical company that was supplying PLD abruptly indicated that they had a change in organizational plans and would no longer provide the AMC with free study drugs. The withdrawal of support for the trial underscores the vulnerability of research and clinical initiatives for PWHIV to fluctuations in external funding and industry support.

6. Emerging Tools and Technological Innovations

Emerging technologies including point-of-care (POC) diagnostics, artificial intelligence (AI)-assisted imaging and telemedicine could potentially offer promising avenues to address long-standing barriers in KS diagnosis and care in LMICs. While these innovations are unlikely to replace foundational investments in health systems, workforce, and drug availability, they may play a complementary role in improving access, timeliness of diagnosis, and care delivery.

6.1 POC Diagnostics and Molecular Testing

One of the most significant barriers to effective KS management in LMICs is limited access to timely histopathologic diagnosis. Conventional di-

agnostic workflows often require centralized pathology services, multiple patient visits, and prolonged turnaround times, contributing to advanced-stage presentation and poor outcomes. Molecular diagnostic approaches targeting KSHV offer a potential solution.

Loop-mediated isothermal amplification (LAMP)-based assays for quantifying KSHV DNA from skin biopsies have demonstrated high diagnostic accuracy, with reported sensitivity of 97% and specificity of 92% compared with gold-standard US based pathology when tested in skin biopsies in over 500 patients from Uganda. The assays can be performed on Tiny Isothermal Nucleic acid quantification system (TINY) portable and energy-flexible POC device, compatible with the requirements with resource-limited settings.⁷¹ In an earlier field evaluation, unfixed punch-biopsy samples from suspected KS lesions underwent DNA extraction and subsequent analysis using TINY. Results were obtained approximately 2.5 hours after the start of the biopsy procedure, including approximately 85 minutes for DNA extraction, and the device could be heated using electricity, sunlight, or a flame.⁷² After training, local staff at the Ugandan clinics were able to operate the system autonomously during the 5-day field trial. Four months later, they independently performed DNA extraction and TINY testing using the instruction manuals, without assistance from the device developers. In a temporally distinct validation cohort of 421 additional KS biopsies from the same clinical sites, the assay maintained similarly high sensitivity (95%) and specificity (90%), supporting the robustness and reproducibility of the platform across time and clinical settings.⁷³ Recent advances in simplified DNA extraction techniques, such as the ColdSHOT method, further enable rapid, equipment-limited preparation of biopsy samples, reducing both processing time and infrastructure requirements.⁷⁴ Together, these innovations bring molecular KS diagnosis closer to true POC implementation and may help reduce diagnostic delays.

6.2 AI-Assisted Diagnostics and Pathology Augmentation

AI is increasingly being explored to augment both clinical and histopathologic diagnosis of KS. At the clinical level, AI-based interpretation of digital photographs of suspicious skin lesions has shown

moderate diagnostic accuracy in early studies. In a cohort of patients in Uganda, AI-based image analysis achieved a sensitivity of 89% and specificity of 51%, underscoring both the promise and current limitations of this approach. Although not yet suitable for stand-alone clinical use, such tools may serve as triage or screening aids, particularly in settings with limited dermatologic expertise.⁷⁵

At the histopathologic level, AI applications may have more immediate clinical utility. Automated detection of LANA has demonstrated excellent performance, with area under the curve values approaching 0.99 and high sensitivity and specificity. These systems can generate interpretable heatmaps and quantitative metrics, reducing interobserver variability and dependence on highly trained pathologists. In settings where pathology expertise is scarce, such approaches may offer a scalable means to improve diagnostic accuracy and workflow efficiency.⁷⁶

6.3 Telemedicine and Digital Health Expansion

Parallel to advances in diagnostics, the expansion of digital connectivity has created new opportunities for telemedicine. Individuals using the internet in SSA has increased from 3% in 2006 to 36% in 2025.⁷⁷ Telemedicine has shown potential to improve healthcare access, enhance efficiency, and bridge geographic disparities in SSA, particularly in underserved regions.⁷⁸ Its adoption accelerated during the COVID-19 pandemic, when healthcare systems increasingly relied on remote consultation and digital platforms to maintain continuity of care. Current telemedicine models include mobile health (mHealth) applications, video consultations, store-and-forward systems for image-based diagnosis, and remote patient monitoring. Although most evidence relates to general healthcare delivery rather than KS-specific care, these platforms may be adaptable to KS management, particularly for triage, specialist consultation, and longitudinal monitoring of skin lesions.

However, telemedicine adoption remains uneven, constrained by infrastructural limitations, regulatory gaps, and disparities in digital literacy. As such, while telemedicine may extend specialist expertise and support decentralized care models, its effectiveness will depend on parallel investments in

infrastructure, policy frameworks, and workforce training.

7. Discussion

The global epidemiology of HIV-associated KS illustrates the profound influence of structural inequities on cancer outcomes. Although KS incidence and mortality have declined substantially in HICs following widespread access to ART, the disease remains a major cause of cancer-related morbidity and mortality in SSA, where late presentation, limited oncology infrastructure, constrained pathology access, and persistent HIV burden continue to drive poor outcomes. Contemporary data demonstrating one-year mortality exceeding 40% in parts of East Africa despite the WHO "Treat All" era underscore that ART expansion alone has not fully translated into equitable cancer outcomes.¹⁸

The striking geographic variation in KS burden closely parallels differences in HIV prevalence, HHV-8 seroprevalence, and broader indicators of socioeconomic development and health-system capacity. Countries with lower Human Development Index (HDI) scores consistently demonstrate higher KS incidence and mortality rates. More broadly, a recent pan-cancer ecological study across 185 countries found that progress toward universal health coverage, higher healthcare expenditure, stronger clinical workforce capacity, improved gender equity, and greater national investment in health systems were associated with better cancer outcomes.⁷⁹ These findings reinforce that disparities in HIV-associated KS are not solely biologic, but are deeply linked to structural determinants of health, healthcare access, and economic development.

The modern management of HIV-associated KS highlights both the successes and limitations of global HIV programs. PEPFAR and other international HIV initiatives have dramatically reduced HIV-related mortality and contributed to declining KS incidence in many regions. However, many LMIC health systems remain heavily dependent on external donor support for HIV prevention, ART delivery, pathology services, workforce training, and chemotherapy procurement. Recent disruptions in global health financing therefore raise concern that progress achieved over the past two decades may be vulnerable to reversal. Because KS is closely tied to HIV control, interruptions in

HIV testing, ART continuity, viral suppression, or oncology infrastructure may rapidly translate into increased KS incidence and mortality.

Prevention remains central to reducing the future burden of HIV-associated KS. Since no approved vaccine or functional cure currently exists for either HIV or HHV-8, reducing HIV transmission through established public health measures remains critically important. Expanding HIV testing, earlier ART initiation, viral suppression, harm-reduction strategies, and wider implementation of pre-exposure prophylaxis (PrEP) are likely to remain among the most effective strategies for preventing HIV-associated KS at the population level. Earlier diagnosis of HIV infection and improved retention in care may also reduce the frequency of severe immunosuppression and advanced disseminated KS presentations.

This review also highlights the importance of implementation-focused approaches to care delivery. Community-based interventions, patient navigators, decentralized HIV and oncology services, and task-shifting strategies may help mitigate workforce shortages and improve treatment engagement in resource-limited setting.^{53,80} Prior studies in SSA reveal that delayed diagnosis and treatment interruptions are often driven not only by infrastructure limitations, but also by transportation costs, stigma, limited health literacy, fear of cancer diagnosis, and fragmented referral pathways.⁵⁴ Addressing these barriers will require culturally informed and community-engaged approaches in addition to biomedical advances.

Emerging technologies may help partially bridge gaps in expertise and infrastructure. POC molecular diagnostics, AI-assisted pathology interpretation, digital imaging platforms, and telemedicine models show promise for improving diagnostic access and extending specialist expertise to underserved regions. However, these technologies should be viewed as complementary tools rather than substitutes for foundational investments in healthcare systems. Without reliable pathology services, medication supply chains, infusion infrastructure, trained personnel, and sustainable financing mechanisms, technological innovations alone are unlikely to substantially alter long-term outcomes.

Several limitations should also be acknowledged when interpreting contemporary epidemiologic

data on KS. Cancer registry coverage remains incomplete across many LMICs, particularly in SSA, raising the possibility of substantial underascertainment of both incidence and mortality. Variability in diagnostic confirmation, pathology availability, and death certification practices further complicates international comparisons. In addition, many studies evaluating treatment outcomes in LMICs are observational and may be influenced by loss to follow-up, referral bias, and heterogeneous treatment availability.

Ultimately, HIV-associated KS represents more than a virally driven malignancy; it serves as a visible marker of persistent global inequities in infectious disease control, cancer care access, and healthcare infrastructure. Continued progress will likely depend not only on advances in pharmacotherapy, but also on sustained investment in public health systems, oncology workforce development, HIV prevention and treatment programs, equitable access to diagnostics and chemotherapy, and broader social and economic development.

8. Conclusion

Despite major advances in HIV care and cancer therapy, HIV-associated KS remains an important cause of cancer-related morbidity and mortality globally, particularly in SSA. The epidemiology of KS reflects the intersection of HHV-8 prevalence, HIV burden, and structural inequities in healthcare access. ART remains the cornerstone of therapy, while systemic treatments such as PLD and paclitaxel have improved outcomes for patients with advanced disease. Emerging therapies, including immune checkpoint inhibitors and immunomodulatory agents, show promise but remain difficult to implement broadly in many LMICs because of cost and infrastructure limitations. Improving outcomes in HIV-associated KS will require more than advances in pharmacotherapy alone. Sustained progress depends on long-term investments in education, public health, poverty reduction, HIV programs, oncology and pathology capacity, access to essential medicines, stigma reduction, and stable domestic and international healthcare funding. Addressing these structural inequities will be essential to narrowing the global disparities that continue to define the modern burden of HIV-associated KS.

Conflict(s) of Interest

The authors declare no conflicts of interest.

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Not applicable. AI tools were not used to generate scientific content, interpret data, or influence the conclusions of this review.

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