

HIV-Associated Malignancies: Unraveling Cancer Pathogenesis Through Medical Imaging, Biomarkers, Precision Medicine, and Vaccine Innovation

Rikza Saleem

Department of Radiology and Imaging Technology, Government College University
Faisalabad

rikzasaleem18@gmail.com

Nawal Shazif

Department of Biological Sciences, Virtual University of Pakistan

nawalshazifofficial@gmail.com

Aqsa Hussain (Corresponding Author)

Department of Physiotherapy, Ziauddin University

aqsahussain041@gmail.com

Rida Abbas

Department of Radiology and Imaging Technology, Government College University
Faisalabad

ridaabbas980@gmail.com

Malahat Choudhary

Department of Human Nutrition & Dietetics, Bahauddin Zakariya University, Multan

choudharymalahat@gmail.com

Areeha Rashad

Department of Radiology and Imaging Technology, Government College University
Faisalabad

areeharashad447@gmail.com

Author Details

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Corresponding E-mail & Author*:

Aqsa Hussain: Department of Physiotherapy, Ziauddin University
aqsahussain041@gmail.com

Abstract

Background: The introduction of effective antiretroviral therapy (ART) has transformed HIV infection into a chronic condition for many people living with HIV (PLWH), yet cancer remains an important source of morbidity and mortality. The malignancy spectrum is heterogeneous: risk is particularly elevated for tumors linked to oncogenic viruses and immune dysfunction, while selected non-AIDS-defining cancers also contribute to the contemporary burden. HIV-associated carcinogenesis is not explained by CD4 depletion alone; persistent viral co-infections, chronic inflammation, immune exhaustion, altered cytokine networks, and tumor-microenvironment effects interact over time. **Objective:** This narrative review integrates current evidence on HIV-associated malignancy biology with the clinical roles and limitations of medical imaging, tissue and circulating biomarkers, precision oncology, and vaccine-based prevention or treatment. It emphasizes the distinction

between established clinical approaches and investigational strategies. **Methods:** A focused narrative synthesis was developed from peer-reviewed epidemiologic studies, mechanistic investigations, clinical trials, and authoritative scholarly reviews. Core themes included cancer-risk patterns in the ART era; KSHV-, EBV-, and HPV-associated oncogenesis; imaging for staging and response assessment; biomarker translation; treatment adaptation; and vaccine innovation. This is not a systematic review, and no formal risk-of-bias assessment or pooled analysis was performed. **Results:** Kaposi sarcoma, non-Hodgkin lymphoma, Hodgkin lymphoma, and HPV-associated anogenital cancers remain central to the HIV-cancer interface. Modern ART reduces several AIDS-defining cancer risks but does not eliminate risk, particularly when immune recovery is incomplete or oncogenic co-infections persist. FDG-PET/CT is useful in selected lymphoma contexts but is not cancer-specific; inflammatory and infectious processes may produce false-positive findings. Histopathology remains essential for diagnosis. Molecular characterization, circulating tumor DNA, immune profiling, and biomarker-guided treatment are promising, but validation and implementation remain uneven. Cancer vaccines have a clear preventive role for HPV and hepatitis B virus where indicated, while therapeutic cancer vaccines and HIV-directed immunization approaches remain investigational. **Conclusion:** Better outcomes require integrated HIV and oncology care, timely ART, prevention and screening, accurate tissue diagnosis, context-aware imaging, and evidence-calibrated use of molecular tools. Future research should prioritize prospective biomarker validation, equitable access to screening and treatment, representation of PLWH in trials, and mechanistically informed vaccine and immunotherapy studies.

1. Introduction

The relationship between HIV infection and malignancy has evolved substantially since the early AIDS epidemic. Severe cellular immunodeficiency initially brought Kaposi sarcoma (KS), aggressive non-Hodgkin lymphoma (NHL), and invasive cervical cancer into clinical focus. ART has changed the natural history of HIV and reduced the incidence of several AIDS-defining cancers, but the cancer burden has not disappeared. With longer survival, clinicians increasingly encounter malignancies arising through a combination of persistent immune dysfunction, oncogenic co-infections, age-associated exposures, and conventional cancer risk factors [1–4].

The term HIV-associated malignancy therefore describes a clinically diverse group rather than one unified disease. For some tumors, immune suppression is a dominant determinant; for others, the persistence of a carcinogenic virus and local immune dysregulation may remain important despite virologic suppression. Epidemiologic comparisons are also affected by competing risks, smoking and alcohol exposure, access to screening, ART timing, and regional prevalence of oncogenic viruses [2–5]. A useful review must distinguish association from causation and avoid attributing every excess cancer risk directly to HIV.

This article links mechanisms to practical diagnostic and translational questions. Imaging can map disease distribution and support response assessment, but it cannot reliably distinguish malignancy from infection in every immunocompromised patient. Biomarkers may refine prognosis or guide therapy, yet a marker should not be described as clinically actionable unless analytical validity, clinical validity, and utility are established. Precision treatment requires coordination of antineoplastic therapy with ART, comorbidity management, and drug-interaction review. Vaccine strategies span established prophylaxis against oncogenic infections and experimental therapeutic platforms [6–10].

2. Review approach and scope

This is a narrative review intended to integrate representative, high-quality evidence rather than estimate a pooled effect. The evidence base includes population-based registry studies, cohort and case-control studies, mechanistic research, clinical trials, and peer-reviewed reviews in oncology, infectious diseases, pathology, and radiology. Priority was given to publications with clear methods, established scholarly publication records, and direct relevance to the review's central themes. The reference list below is numbered in order of first citation; each bracketed citation in the text maps to a complete bibliographic entry. The narrative does not claim exhaustive database coverage, and emerging technologies are discussed with explicit caution where clinical validation is limited.

3. Epidemiology and the changing cancer spectrum

Population-based studies demonstrate that PLWH experience a higher incidence of several cancers than the general population, but the magnitude varies markedly by tumor type. KS and selected lymphomas show particularly large relative elevations, while anal cancer and Hodgkin lymphoma also occur more frequently. Some cancers not classically categorized as AIDS-defining—including lung and liver cancers—contribute to excess burden, although causal pathways may include smoking, viral hepatitis, chronic inflammation, and social determinants rather than a direct oncogenic effect of HIV [1–5].

ART-era trends should be interpreted carefully. Incidence reductions in KS and some NHL subtypes are consistent with improved immune function and viral suppression. However, residual risk persists, and trends differ across lymphoma subtypes. Diffuse large B-cell lymphoma remains a major HIV-associated lymphoma; Burkitt lymphoma, primary effusion lymphoma, and plasmablastic lymphoma have distinct biology and may not follow the same trend. Hodgkin lymphoma risk remains elevated and is often linked to EBV biology and immune dysregulation [6,7]. These patterns argue against a single aggregate “HIV cancer risk” measure as sufficient for clinical planning.

Geography changes the relative importance of malignancies. In regions with high KSHV prevalence, KS is prominent; where HPV exposure and screening gaps are substantial, cervical and other HPV-related cancers are major concerns. A 2025 global incidence analysis estimated that 81,300 cancer cases worldwide in 2022 were attributable to HIV across nine cancer sites, with the largest contributions from cervical cancer, KS, and NHL and a substantial concentration in eastern and southern Africa [5]. These estimates depend on population-attributable-fraction assumptions and underlying cancer-registration quality; they should be read as modeled burden, not

individually observed causal attribution.

Table 1. Major malignancy groups associated with HIV, key biologic links, and clinical implications

Malignancy group	Principal biologic association	Clinical pattern / concern	Evidence-linked implications
Kaposi sarcoma (KS)	KSHV/HHV-8; immune dysfunction; cytokine and angiogenic signaling	Cutaneous, mucosal, nodal or visceral disease; may flare with immune reconstitution	Confirm histologically when needed; ART is foundational; systemic therapy for advanced disease [8,9].
HIV-associated NHL	EBV/KSHV in selected subtypes; B-cell activation; immune surveillance failure	DLBCL, Burkitt lymphoma, primary CNS lymphoma, primary effusion lymphoma and plasmablastic lymphoma are biologically distinct	Subtype by pathology; stage and response assessment; coordinate ART and chemotherapy [6,7].
Hodgkin lymphoma	Often EBV-associated; chronic immune dysregulation	Risk remains elevated in ART era; presentation may be advanced	Histologic diagnosis and standard lymphoma staging; tailor treatment to patient and disease [6,7].
Cervical / anal / other HPV-related cancers	Persistent high-risk HPV; impaired viral clearance and local immune control	Cervical precancer/cancer; anal and other anogenital disease	Vaccination, screening and timely treatment of precursor lesions are central; screening policies vary by population [10,11].
Selected non-AIDS-defining cancers	Mixed factors: aging, smoking, HBV/HCV, inflammation and other exposures	Lung, liver and other cancers contribute to overall burden	Avoid simplistic HIV-only attribution; address modifiable risks and follow standard evidence-based screening [2–4].

4. Mechanistic foundations of HIV-associated carcinogenesis

4.1 Immune surveillance and chronic immune activation

HIV preferentially infects and depletes CD4⁺ T cells, disrupting coordinated antiviral and antitumor immunity. Loss of helper function impairs cytotoxic T-cell responses, B-cell regulation, and immune memory. Even with suppressive ART, immune reconstitution may be incomplete; persistent immune activation, altered T-cell phenotypes, and exhaustion-associated pathways can remain detectable. These changes can weaken control of oncogenic viruses and tumor cells, although their contribution

differs across cancers and cannot be inferred from CD4 count alone [6,7,12].

The cancer relationship is not a simple linear function of current CD4 count. Cumulative immunosuppression, nadir CD4 count, duration of uncontrolled viremia, age, coinfections, and treatment history may all matter. Some elevated cancer risks persist in people with virologic suppression and relatively preserved CD4 counts, especially for tumors associated with chronic viral infection or local mucosal immune abnormalities [3,4,11,12]. This makes longitudinal HIV history more informative than a single laboratory measurement.

4.2 Oncogenic co-infections and viral mechanisms

KSHV is the etiologic agent of KS and is implicated in primary effusion lymphoma and KSHV-associated multicentric Castleman disease. Latent and lytic viral programs encode proteins that promote survival, angiogenesis, inflammatory signaling, and immune evasion. KS lesions contain spindle-shaped endothelial-lineage cells within a cytokine-rich, vascular microenvironment; the tumor phenotype reflects both viral gene expression and host responses. This biology explains why immune restoration can lead to regression in some cases, while advanced disease often requires systemic antineoplastic treatment [8,9].

EBV contributes to several HIV-associated lymphoid malignancies. Loss of effective EBV-specific immune control permits expansion of infected B cells, while viral latency proteins and host signaling pathways support proliferation and survival. Yet EBV status and molecular phenotype vary by lymphoma subtype; EBV positivity should be interpreted within histopathologic context rather than treated as a universal marker of HIV-related lymphoma [6,7,13].

High-risk HPV drives cervical, anal, and other anogenital carcinogenesis through persistent infection and expression of E6 and E7 oncoproteins, which interfere with p53- and retinoblastoma-related tumor suppressor pathways. HIV-associated immune dysfunction may impair HPV clearance, sustain high-risk HPV infection, and contribute to dysplastic progression. Local immune effects, including altered cytotoxic T-cell infiltration and checkpoint signaling, have been described in anal carcinogenesis, providing mechanistic rationale for prevention and immunotherapy research [10,11].

4.3 Inflammation, cytokines, and tumor microenvironment

Chronic immune activation can alter cytokine and chemokine networks, promote B-cell hyperactivation, and support angiogenic or anti-apoptotic signals. These processes may contribute to lymphomagenesis and the persistence of virus-associated lesions. The tumor microenvironment is therefore not a passive backdrop: immune cells, endothelial cells, fibroblasts, cytokines, and viral products interact to shape growth, immune escape, and response to treatment [8,9,12]. However, many mechanistic observations arise from experimental systems or selected tumor cohorts; translation to patient-level prognostic markers requires prospective validation.

4.4 ART, immune reconstitution, and immune reconstitution inflammatory syndrome

ART is a core component of cancer prevention and treatment in PLWH, but immune reconstitution can complicate the timing and interpretation of cancer-associated inflammation. KS-IRIS can present with worsening lesions or edema after ART initiation, and lymphoma or infection may also become clinically apparent during immune recovery. These phenomena require a differential diagnosis that considers tumor progression, opportunistic infection, drug toxicity, and immune reconstitution. ART should not be reflexively interrupted; decisions require multidisciplinary assessment and attention to the malignancy, immune status, and interaction profile [8,9,14].

5. Medical imaging: strengths, limits, and integration with pathology

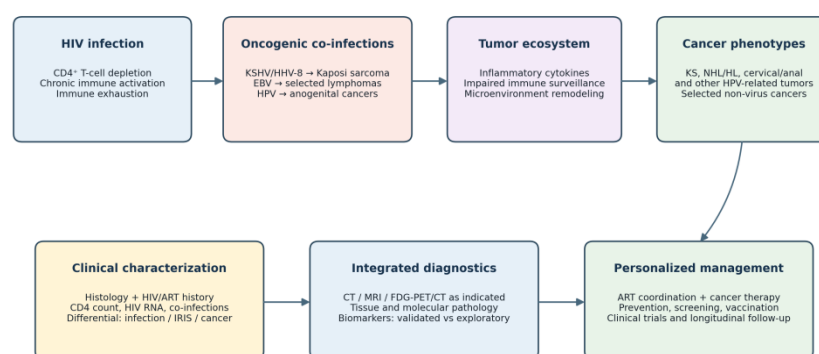
Imaging supports detection, staging, biopsy planning, assessment of disease extent, and evaluation of response. The modality should be selected according to tumor

type and clinical question. CT is widely used for thoracic, abdominal, and nodal assessment; MRI provides high soft-tissue contrast and is important in central nervous system disease; FDG-PET/CT can characterize metabolically active lymphoma and assist staging or response evaluation. In KS, imaging may be used to assess suspected visceral involvement, while clinical examination and tissue diagnosis remain important for cutaneous disease [15–18].

FDG uptake is not specific for cancer. HIV-associated lymphadenopathy, opportunistic infections, inflammatory conditions, and immune reconstitution may be metabolically active. A positive PET finding therefore requires interpretation alongside symptoms, CD4 count, viral suppression, timing of ART, anatomic pattern, and—when feasible—histology or microbiology. PET/CT can be particularly useful in HIV-associated lymphoma, but published evidence is often based on relatively small cohorts and should not be generalized to every malignancy or every clinical scenario [16,17].

For suspected primary CNS lymphoma, MRI characterizes lesion location, enhancement, edema, and multiplicity, but imaging overlap with toxoplasmosis and other infections remains a recognized challenge. FDG-PET may provide additional information in selected settings, yet no imaging test universally replaces tissue diagnosis or appropriate microbiologic evaluation. Imaging algorithms must also account for renal function, contrast risks, radiation exposure, resource availability, and the need to avoid unnecessary repeated scans [15,18].

Figure 1. Integrated conceptual model linking HIV-related immune and viral mechanisms to diagnostic and translational care.



Conceptual synthesis: multiple interacting mechanisms; no single biomarker or imaging test replaces tissue diagnosis when indicated.

Figure legend: HIV-associated malignancy emerges through interacting processes rather than a single pathway. HIV-related immune dysfunction and oncogenic co-infections shape tumor biology and the microenvironment. Clinical evaluation integrates histopathology, HIV history, imaging, and molecular tests; management combines ART, tumor-directed therapy, prevention, and longitudinal monitoring. The diagram is a conceptual synthesis and not a validated clinical decision algorithm.

6. Biomarkers: clinical utility versus emerging promise

Biomarkers in HIV-associated cancer span several categories: clinical and immunologic markers (CD4 count, HIV RNA, immune history), pathogen-associated measures (EBV or KSHV detection in appropriate contexts, HPV testing), tissue pathology and immunophenotyping, genomic alterations, and circulating analytes such as cell-free DNA. Their purposes differ: screening, diagnosis, subtype classification, prognosis, response monitoring, or selection of therapy. A marker useful for one purpose is not automatically appropriate for another [6,7,19].

CD4 count and HIV viral load are clinically important contextual variables but are not cancer-specific diagnostic biomarkers. A low CD4 count may signal vulnerability to selected malignancies and infections, while a suppressed viral load

reflects ART effectiveness; neither establishes a tumor diagnosis. Similarly, EBV-associated assays can be informative in selected lymphoproliferative disorders but require interpretation based on tumor subtype, sample type, and clinical setting [6,7,13].

Tissue biomarkers—including immunohistochemistry, in-situ hybridization, and targeted molecular testing—are central to classifying lymphoma and other tumors. They can distinguish entities with different prognosis and treatment, identify actionable alterations, and clarify whether a lesion is malignant or reactive. In practice, adequate sampling and expert pathology review are often more consequential than adding an unvalidated broad biomarker panel. When molecular testing is used, the report should specify assay, specimen, tumor fraction, and limitations [6,7,19].

Circulating tumor DNA and other liquid-biopsy approaches may offer minimally invasive monitoring of tumor burden, clonal evolution, and treatment response. However, HIV-related inflammation, infection, clonal hematopoiesis, and tumor heterogeneity can complicate interpretation. Analytical performance, thresholds, and clinical utility need validation for each disease and intended use. At present, liquid biopsy should not be portrayed as a universal replacement for tissue diagnosis or standard response criteria [19,20].

Table 2. Biomarker classes, intended roles, and evidence cautions

Biomarker class	Potential role	Current clinical footing	Important caveat
CD4 count / HIV RNA	Immune context; longitudinal risk and ART response	Routine HIV care measures	Not tumor-specific; do not diagnose malignancy from these values alone [6,12].
Histology / immunophenotype	Tumor diagnosis, classification, prognostic grouping	Established and often indispensable	Requires adequate specimen and interpretation in clinical context [6,7].
EBV/KSHV/HPV-related assays	Support etiologic characterization or disease-specific evaluation	Use depends on cancer subtype and assay	Detection alone may not prove causation in an individual lesion [8,10,13].
Tumor genomics / actionable alterations	Treatment selection; disease classification	Established for selected tumors/alterations	Do not assume every detected variant is actionable or validated in HIV cohorts [19].
ctDNA / liquid biopsy	Potential monitoring, residual disease, evolution	Emerging; context- and disease-specific	Validation and clinical utility remain variable; confounding is possible [20].
Immune/checkpoint inhibitor profiling	Mechanistic insight;	Primarily translational or	Biologic plausibility does

	potential treatment stratification	selected clinical settings	not establish a validated clinical test [11,19].
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7. Precision medicine and treatment adaptation

Precision oncology seeks to match treatment to tumor biology, host context, and patient preferences. In HIV-associated malignancies, the relevant precision framework includes histologic subtype, stage, molecular features, performance status, organ function, prior ART exposure, CD4 count, HIV RNA, opportunistic infection risk, and drug–drug interactions. This is not a rationale to withhold standard cancer treatment solely because a patient has HIV. Rather, the goal is to deliver effective oncologic care while maintaining HIV control and minimizing avoidable toxicity [6,7,21].

For HIV-associated lymphoma, contemporary management generally follows disease-specific oncology principles, with ART continued or optimized in coordination with the HIV team. Regimen choice and supportive care require careful review of interactions between antiretrovirals, cytotoxic drugs, corticosteroids, antiemetics, antimicrobials, and anticoagulants. The risk of infection, marrow suppression, hepatic or renal dysfunction, and treatment interruption must be addressed individually. The evidence base supports multidisciplinary care; it does not support a one-size-fits-all dose modification based only on HIV status [6,7,21].

KS treatment illustrates the importance of biologically informed staging. ART can be sufficient for selected limited disease, whereas advanced, symptomatic, or visceral KS often requires systemic chemotherapy alongside ART. Treatment decisions incorporate lesion burden, edema, oral or visceral involvement, immune status, and potential KS-IRIS. Mechanism-directed approaches and immunotherapies are being studied, but established therapies should remain distinguished from trial-based interventions [8,9].

Immune checkpoint inhibitors have generated interest because they can reinvigorate antitumor T-cell activity. PLWH were historically excluded from many oncology trials, leaving gaps in evidence. Prospective studies and clinical experience have increasingly examined safety and activity in people with controlled HIV, but selection criteria, HIV monitoring, immune-related adverse events, and tumor-specific efficacy remain important. Results in one cancer type or a carefully selected cohort should not be generalized automatically to all PLWH or all checkpoint inhibitors [21,22].

Molecularly targeted agents and cellular therapies may be appropriate when tumor-specific indications are present, but HIV-specific pharmacology and immune context should be considered. Precision medicine must be clinically actionable, not merely descriptive: the molecular finding should have evidence supporting a treatment decision, and access to testing and matched therapy should be feasible. Trial participation is particularly valuable where HIV-associated subgroups remain underrepresented [19,21,22].

8. Vaccine innovation and cancer prevention

Vaccine strategies are best separated into prevention of oncogenic infections, therapeutic cancer vaccination, and HIV-directed vaccines. Prophylactic HPV vaccination prevents infection with vaccine-covered HPV types and is a cancer-prevention intervention; vaccination does not treat established HPV-related lesions. HBV vaccination can reduce the risk of HBV-associated liver cancer through prevention of chronic infection. Vaccine schedules and eligibility should follow current national guidance and account for immune status [10,23].

For PLWH, prevention also includes cervical screening and management of

precancerous lesions, risk-adapted anal cancer evaluation where recommended, smoking cessation, viral hepatitis diagnosis and treatment, and sustained ART. Screening recommendations differ among jurisdictions and populations because evidence, access, and benefit–harm balances are not identical. Programs should therefore state the guideline framework and population rather than imply one universal screening schedule [10,11,23].

Therapeutic cancer vaccines aim to induce or amplify tumor-specific T-cell responses, including responses to viral antigens such as HPV E6/E7 or EBV-associated targets. Platforms include peptide, protein, nucleic-acid, viral-vector, and cell-based approaches. Their rationale is strong in virus-associated cancers, but therapeutic vaccination remains an investigational strategy for most established HIV-associated malignancies; efficacy must be demonstrated in appropriately designed trials, often in combination with standard therapy or immune modulation [22,24].

An effective preventive HIV vaccine remains an unmet need. HIV therapeutic vaccines are being investigated to improve immune control or complement ART, but these approaches should not be conflated with approved cancer vaccines or routine cancer care. Vaccine innovation in this field will require careful attention to immune status, antigen selection, durability, safety, and representation of populations most affected by HIV [24,25].

9. Integrated clinical pathway and research priorities

A practical pathway begins with clinical suspicion and careful history: HIV diagnosis and ART timeline, adherence, viral suppression, current and nadir CD4 count when available, prior opportunistic infections, vaccination and screening history, tobacco and alcohol exposure, hepatitis status, and symptoms suggesting a specific malignancy. Examination and appropriate imaging can define disease extent and guide biopsy, but histopathology remains the anchor for diagnosis whenever feasible. In parallel, infection and inflammatory mimics should be considered, especially in advanced immunosuppression or following ART initiation [15–18].

After diagnosis, multidisciplinary planning should connect oncology, HIV/infectious diseases, radiology, pathology, pharmacy, and supportive care. The plan should specify tumor-directed treatment, ART continuation or adjustment, interaction review, prophylaxis where indicated, monitoring for immune reconstitution phenomena, and a response-assessment strategy. Shared decision-making should address treatment goals, fertility and sexual health when relevant, psychosocial needs, and access barriers. This coordinated model is especially important where cancer and HIV services are fragmented [6,7,21].

Research priorities include: (1) prospective, subtype-specific biomarker validation in PLWH; (2) imaging studies that distinguish tumor from infection and IRIS using reproducible criteria; (3) inclusion of PLWH in therapeutic trials with transparent eligibility and HIV-related outcomes; (4) real-world evaluation of treatment interactions and toxicity; (5) prevention implementation studies for HPV-associated cancers; and (6) equitable access to molecular diagnostics and high-quality cancer care. Studies should report ART status, CD4 measures, viral suppression, coinfections, and relevant social or geographic context to support interpretation and transportability [5,19,21,22].

10. A Multidisciplinary Translational Framework

HIV-associated malignancies arise through interactions among immune dysfunction, oncogenic co-infections, chronic inflammation, tumor biology, and broader social and healthcare determinants. Their investigation can therefore benefit from integrating oncology and infectious-disease expertise with pathology, medical imaging, molecular diagnostics, computational analysis, epidemiology, and implementation science. Findings from adjacent disciplines may provide transferable

methods and hypotheses, but should not be presented as direct evidence of HIV-associated cancer mechanisms unless validated in relevant populations.

A multimodal framework could combine HIV history, ART exposure, CD4 count, viral suppression, histopathology, imaging features, and selected molecular or circulating biomarkers. Computational methods may help identify patterns associated with tumor subtype, diagnostic uncertainty, or treatment response. However, clinical utility would require prospective validation, transparent reporting, and assessment across diverse healthcare settings [32,41,47,53,56,76,80,81].

10.1. One Health, Environmental Context, and Social Determinants

The One Health perspective emphasizes the interconnectedness of human health, environmental systems, and population-level conditions. Although HIV-associated malignancies are not simply environmental diseases, their burden is influenced by geographic variation in oncogenic-virus prevalence, access to ART, screening availability, diagnostic capacity, and treatment access. Research on environmental monitoring and population health can offer methodological perspectives for studying exposure and context [38,49,50,84]. These sources do not establish environmental causes of HIV-associated cancer; rather, they support a broader framework for examining determinants of health. Studies of migration, displacement, socioeconomic conditions, and healthcare access may also inform analyses of barriers to prevention and timely cancer care [54,55,70].

10.2. Biomarkers, Multi-Omics, and Liquid Biopsy

HIV-associated malignancies involve multiple interacting biological pathways, making integrated biomarker approaches potentially informative. Multi-omics and molecular-monitoring research in other systems provides methodological examples for combining complex biological datasets [39,49]. Work on nutritional biomarkers, host physiology, and inflammation may also inform hypothesis development [35,36,51,52,61,66,73,86], although such findings cannot independently validate a cancer biomarker in PLWH. Future studies could combine tissue classification with ctDNA, pathogen-associated markers, immune profiling, and longitudinal clinical data. Analytical validity, disease-specific performance, and clinical utility would need to be established, with attention to confounding from HIV-related inflammation, infection, and clonal hematopoiesis.

10.3. Imaging and Artificial Intelligence

Imaging abnormalities in PLWH may reflect malignancy, infection, inflammation, or immune reconstitution. AI-based imaging research offers a methodological basis for investigating whether radiological features can be interpreted more effectively when combined with clinical, pathological, and laboratory information [47,81]. Models developed in other cancers or populations should not be assumed to generalize to PLWH without independent validation. Relevant endpoints may include diagnostic uncertainty, biopsy targeting, response assessment, or discrimination of malignant lesions from infectious and inflammatory mimics.

10.4. Nutrition, Microbiome, and Immunometabolism

Research on functional foods, gut microbiome modulation, nutritional biomarkers, and chronic inflammation provides broader context for investigating host physiology [28,33,36,51,61,66,73]. These studies should not be interpreted as direct evidence that dietary interventions prevent or treat HIV-associated malignancies. A future research direction could assess whether nutritional status, microbiome features, or metabolic markers associate with immune recovery, treatment tolerance, or inflammatory profiles in PLWH with cancer. Any intervention would require disease-specific clinical testing and patient-centered outcomes.

10.5. Precision Oncology, Governance, and Responsible Data Integration

Precision oncology depends on more than identifying molecular alterations; it also requires validated tests, clinical interpretation, service coordination, and access to appropriate treatment. Research on governance, risk management, healthcare

workforce sustainability, and responsible AI implementation provides useful conceptual frameworks [42,55,57,80]. In PLWH, precision treatment should incorporate tumor biology alongside ART exposure, potential drug interactions, immune status, infection risk, organ function, and patient preferences. Data integration should support rather than replace multidisciplinary clinical judgment. Privacy, algorithmic bias, population representation, and unequal access to diagnostics must be considered before deployment.

11. Proposed Experimental Framework

11.1. Proposed Study Title

A Multimodal Clinical, Imaging, and Biomarker Framework for Characterizing HIV-Associated Malignancies: A Prospective Translational Study

11.2. Hypothesis

Integrating HIV-related clinical variables, medical imaging features, histopathological classification, and selected molecular or circulating biomarkers may provide more informative characterization of HIV-associated malignancies than clinical variables alone. This is a proposed hypothesis, not an established finding of this narrative review.

11.3. Study Design and Data Domains

A prospective observational study could recruit PLWH undergoing evaluation or treatment for suspected or confirmed malignancy. Participants could be characterized by tumor type, including Kaposi sarcoma, non-Hodgkin lymphoma, Hodgkin lymphoma, and HPV-associated anogenital malignancies. Data collection should be guided by clinical indication, ethics approval, available resources, and the specific malignancy; not every participant would require every test.

Data domain	Candidate variables	Analytical purpose
HIV-related clinical	CD4 count, HIV RNA, ART history, nadir CD4, opportunistic infections	Immune and treatment context
Medical imaging	CT, MRI, or FDG-PET/CT findings as clinically indicated	Disease distribution and imaging patterns
Histopathology	Tumor subtype, immunophenotype, pathogen-associated findings	Reference diagnostic classification
Molecular biomarkers	Tumor alterations or pathogen-associated markers where indicated	Disease-specific biological features
Circulating biomarkers	ctDNA or selected analytes where feasible	Exploratory monitoring or response signals
Immune/inflammatory	Selected cytokines or immune-profile features	Host-response patterns
Social/healthcare context	Screening history, access, geographic setting,	Implementation and equity factors

	diagnostic delays	
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11.4. Proposed Analytical Strategy

1. Clinical characterization: describe participants by malignancy subtype, ART history, immune status, and relevant clinical variables.
2. Single-domain analysis: examine clinical, imaging, pathological, and biomarker features against prespecified outcomes.
3. Multimodal integration: compare a clinical-variable model with models incorporating imaging, pathology, and biomarkers.
4. Validation: use an appropriate internal validation strategy and, where possible, an independent external cohort.

Potential endpoints include diagnostic classification, tumor subtype, treatment response, or time-to-event outcomes. Endpoints should be prespecified and compatible with the sample size and follow-up. Evaluation may include discrimination, calibration, sensitivity, specificity, and clinical utility. If the sample is limited, analysis should prioritize a small set of prespecified variables and avoid overfitting.

11.5. Conceptual Workflow

1. PLWH with suspected or confirmed malignancy
2. Clinical and HIV-related assessment
3. Imaging according to clinical indication
4. Histopathology and immunophenotyping
5. Selected molecular and circulating biomarkers
6. Multimodal data integration and statistical/computational analysis
7. Exploratory characterization or outcome prediction
8. Internal/external validation and multidisciplinary interpretation

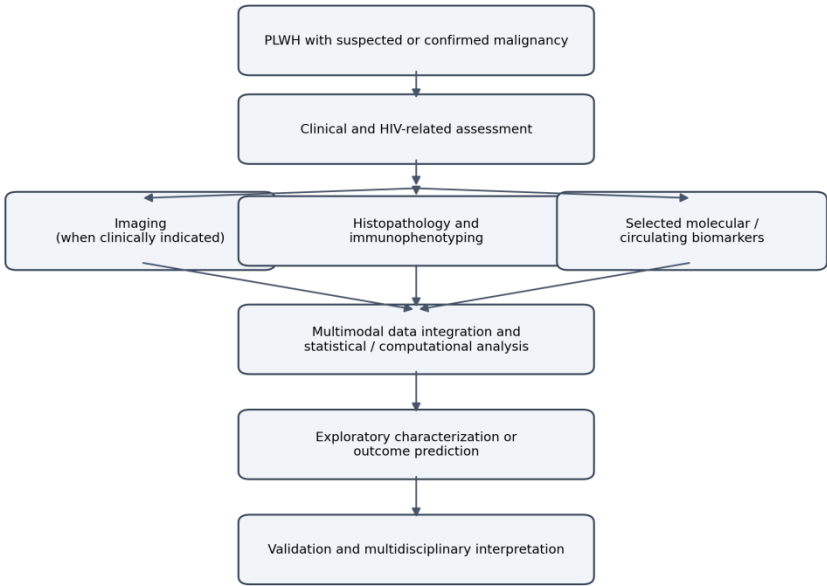


Figure 2 legend. Proposed multimodal research workflow for HIV-associated malignancies. The framework illustrates integration of clinical, imaging, pathological, and biomarker information in a prospective observational study. It is a research proposal, not a validated clinical algorithm.

11. Discussion

The evidence assembled here supports a model in which HIV-associated malignancy is produced by the intersection of immune impairment, persistent oncogenic infection, inflammatory signaling, and tumor-specific genetic events. ART has reduced the burden of several cancers by improving immune function, but it cannot reverse every carcinogenic process or guarantee clearance of established oncogenic viruses. The residual burden is shaped by tumor biology and by structural factors such

as screening availability, diagnostic delay, treatment access, and regional epidemiology [1–7].

This model has practical consequences. First, cancer risk should be interpreted by tumor subtype and exposure rather than as a uniform consequence of HIV. Second, imaging must be used as a decision-support tool, not a standalone diagnosis, because metabolically active infection and inflammation can resemble cancer. Third, biomarkers require purpose-specific validation: routine clinical measures, tissue classifiers, and emerging liquid biopsies occupy different levels of readiness. Fourth, precision oncology in PLWH means coordinating standard tumor treatment with HIV care—not withholding effective therapy or assuming that every patient requires an HIV-specific cancer regimen [6–8,15–22].

Vaccine innovation likewise spans different evidence tiers. HPV and HBV vaccination are preventive tools with established roles according to indication, whereas therapeutic cancer vaccines and HIV therapeutic vaccines remain areas of active investigation. The field should avoid language that blurs preventive efficacy, immunogenicity, and clinical tumor response. Translational progress will depend on endpoints that matter to patients, adequately powered studies, careful safety monitoring, and inclusion of populations often underrepresented in research [10,23–25].

Several limitations of this narrative review should be acknowledged. The review is not systematic; it does not provide a reproducible exhaustive search strategy, duplicate screening, formal quality appraisal, or meta-analysis. Evidence is heterogeneous across malignancy subtypes, countries, ART eras, and clinical settings. Some promising biomarker and immunotherapy concepts are supported primarily by early-phase trials or translational studies. Therefore, the conclusions are intended to organize evidence and identify research needs, not to replace disease-specific clinical guidelines or multidisciplinary decisions.

12. Conclusion

HIV-associated malignancies remain a clinically important and biologically diverse group of cancers despite major advances in ART. Their pathogenesis reflects interactions among immune dysfunction, oncogenic viruses, chronic inflammation, and tumor microenvironments. Imaging contributes to staging and response assessment but must be interpreted with awareness of infectious and inflammatory mimics; tissue pathology remains central. Biomarkers and precision approaches can improve classification and treatment selection when validated for the intended use, while liquid biopsy and immune profiling remain emerging in many settings. Cancer prevention through ART, vaccination, screening, and risk-factor modification should be integrated with timely, evidence-based oncology care. The next phase of progress depends on rigorous validation, clinical trial inclusion, multidisciplinary practice, and equitable access to prevention, diagnostics, and treatment.

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