SAS Journal of Medicine
Abbreviated Key Title: SAS J Med
ISSN 2454-5112
Journal homepage: https://saspublishers.com

OPEN ACCESS

Infectious Diseases

Burkitt Lymphoma Unmasking HIV Infection Amid Multiple Life-Threatening Opportunistic Infections: A Case Report

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DOI: https://doi.org/10.36347/sasjm.2026.v12i06.015 | **Received:** 05.05.2026 | **Accepted:** 18.06.2026 | **Published:** 20.06.2026

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Abstract **Case Report**

Background: Burkitt lymphoma (BL) is an aggressive AIDS-defining B-cell non-Hodgkin lymphoma. Its simultaneous occurrence with severe opportunistic infections at HIV diagnosis represents an exceptional and life-threatening scenario. ***Case Presentation:*** A 36-year-old male with newly diagnosed HIV infection (viral load >10,000,000 copies/mL) presented simultaneously with invasive pulmonary aspergillosis, oropharyngeal-esophageal candidiasis, marked hepatic impairment, and a multinodular hepatic pattern with a suspicious pancreatic lesion on MRI. Inguinal lymph node biopsy confirmed Burkitt lymphoma with a starry-sky histological pattern. Liposomal amphotericin B was initiated first, followed by TDF/3TC/DTG antiretroviral therapy. After emergency transfer to hematology, the patient died approximately three weeks after chemotherapy initiation. ***Conclusion:*** This case highlights the exceptional prognostic severity of BL diagnosed concurrently with AIDS-defining opportunistic infections, and underscores the need for early HIV diagnosis, systematic lymph node biopsy in immunocompromised patients, and multidisciplinary management.

Keywords: Burkitt lymphoma; HIV/AIDS; invasive aspergillosis; AIDS-defining malignancy; antiretroviral therapy.

INTRODUCTION

Burkitt lymphoma (BL) is classified by the WHO as a highly aggressive mature B-cell non-Hodgkin lymphoma characterized by MYC oncogene translocation — most commonly t(8;14) — and a near-100% Ki-67 proliferative index. Its immunodeficiency-associated variant is a well-established AIDS-defining malignancy, occurring approximately 1,000 times more frequently in HIV-infected individuals than in the general population. Unlike other HIV-associated lymphomas that typically arise at very low CD4 counts, BL may develop at relatively preserved CD4 levels, driven by HIV-induced chronic B-cell stimulation, EBV co-infection, and dysregulated immune surveillance. The simultaneous presentation of BL with multiple severe opportunistic infections in a newly diagnosed HIV patient with extreme viremia is exceptional. Each therapeutic intervention — antifungal treatment, ART initiation, and chemotherapy — carries additive toxicity risks, making clinical decision-making particularly challenging. We report this case to highlight the diagnostic complexity, the required therapeutic sequencing, and the fatal prognosis of this combined presentation.

CASE OBSERVATION

A 36-year-old single male with no significant medical or surgical history with a 10-day history of general deterioration, dyspnea, and vomiting. He reported active tobacco and cannabis use since age 17, alcohol consumption for approximately 7 years, and unprotected sexual intercourse. Physical examination revealed oropharyngeal-esophageal candidiasis treated with fluconazole, and a painless left inguinal lymphadenopathy. Initial workup confirmed positive HIV serology. At follow-up, the inguinal adenopathy measured 5 cm on imaging, CT scan revealed multiple deep lymphadenopathies, and hepatic cytolysis was noted. The patient was hospitalized with a viral load exceeding 10,000,000 copies/mL. During hospitalization, sputum examination identified *Aspergillus spp.* filaments confirmed by chest CT as invasive pulmonary aspergillosis (IPA), and hepatic workup showed a mixed cytolysis-cholestasis pattern (cytolysis ~10× normal). Liposomal amphotericin B was initiated at 200 mg/day. Hepatic MRI revealed a heterogeneous multinodular liver with multiple T1-hypo/T2-hyper/diffusion-restricted enhancing nodules grouped in the left lobe (largest 36 mm at segment VI), hilar lymphadenopathies of secondary appearance

(largest 15 mm), and a well-defined 29 × 21 mm unilocular pancreatic body lesion with thin wall and peripheral enhancement — in contact with the splenic vein without vascular invasion — raising suspicion of a primary pancreatic process. The inguinal lymph node biopsy of a 9g lymph node showed diffuse disruption of nodal architecture by a monomorphic atypical lymphoid infiltrate with a characteristic starry-sky pattern: scattered tingible-body macrophages engulfing apoptotic debris within a sea of medium-sized B cells showing prominent nucleoli, scant basophilic cytoplasm, and numerous mitoses, concluding to Burkitt lymphoma (+++), with immunohistochemistry recommended. Key biological findings included: LDH 1,945 UI/L (~8× normal), uric acid 109.51 mg/L (suggesting spontaneous tumor lysis), platelets 67,000/mm³, albumin 24.54 g/L, lymphocytes 520/mm³, and CRP 51.8 mg/L — together reflecting extreme tumor burden, profound immunodepression, and systemic depletion. CMV and toxoplasma IgG were positive with negative IgM (past infections); HIV positive; HBsAg, HCV, TPHA/VDRL negative. ART was deferred two weeks after antifungal initiation to reduce IRIS risk, then started on January 8, 2026 with TDF/3TC/DTG (TLD). Supportive care included IV rehydration, parenteral nutrition, potassium supplementation, cotrimoxazole prophylaxis, ondansetron, and proton pump inhibitor. Following BL confirmation, the patient was urgently transferred to hematology for chemotherapy; he died approximately three weeks after initiation, illustrating the catastrophic convergence of extreme tumor burden, profound immunosuppression, concurrent opportunistic infections, organ dysfunction, and malnutrition.

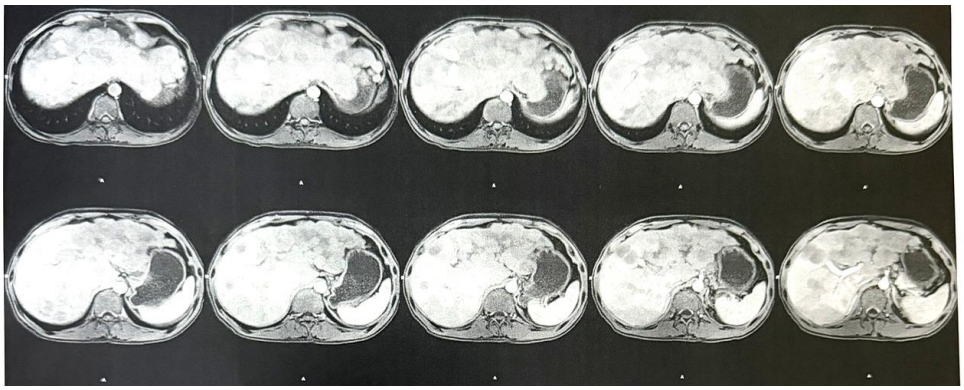

Figure 1: Hepatic MRI — Axial T2-weighted sequences. Multiple heterogeneous hepatic nodules in T2 hypersignal with lobulated confluent contours, predominantly left lobe, with diffusion restriction consistent with diffuse malignant (lymphomatous) hepatic infiltration

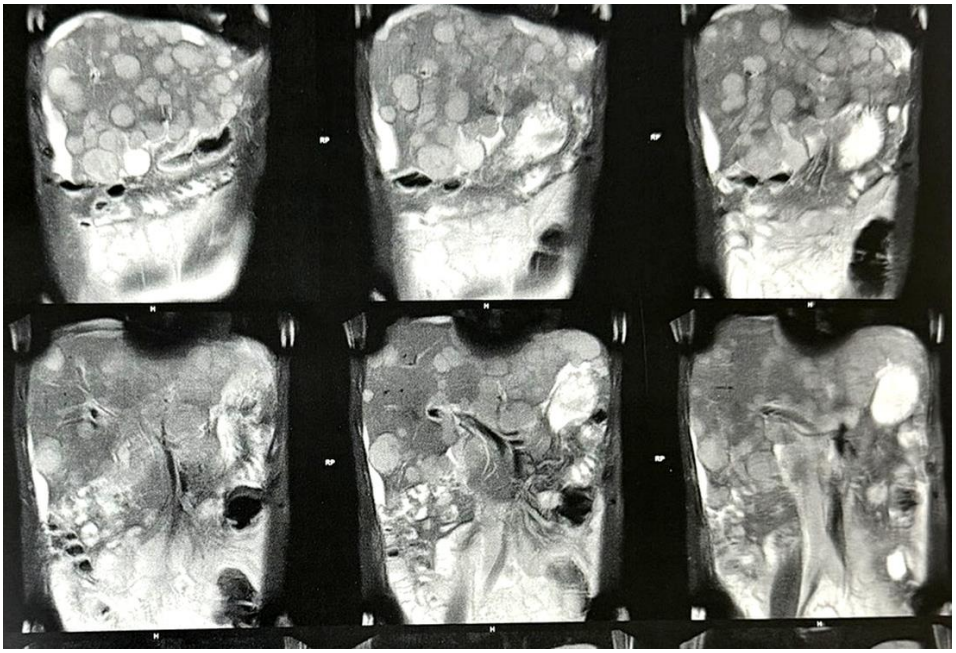

Figure 2: Hepatic MRI — Coronal post-contrast T1 VIBE sequences. Overview of the multinodular hepatic involvement, hilar lymphadenopathies (15 mm), and the 29 × 21 mm pancreatic body lesion with peripheral enhancement in contact with the splenic vein — without vascular invasion

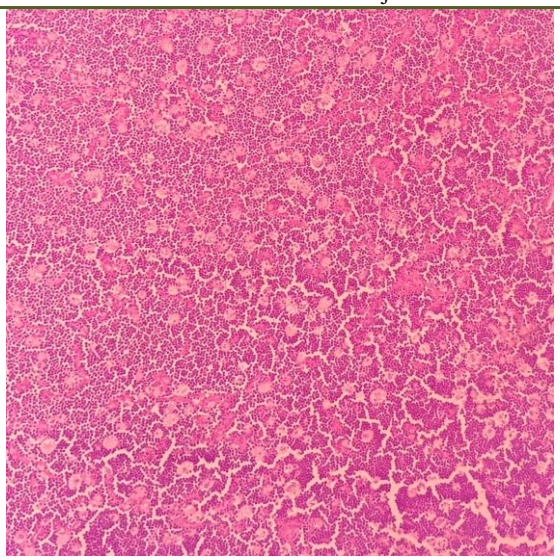

Figure 3: Inguinal lymph node biopsy (HE, low magnification). Diffuse effacement of nodal architecture with a starry-sky pattern: pale tingible-body macrophages scattered within densely packed neoplastic lymphoid cells — hallmark of Burkitt lymphoma

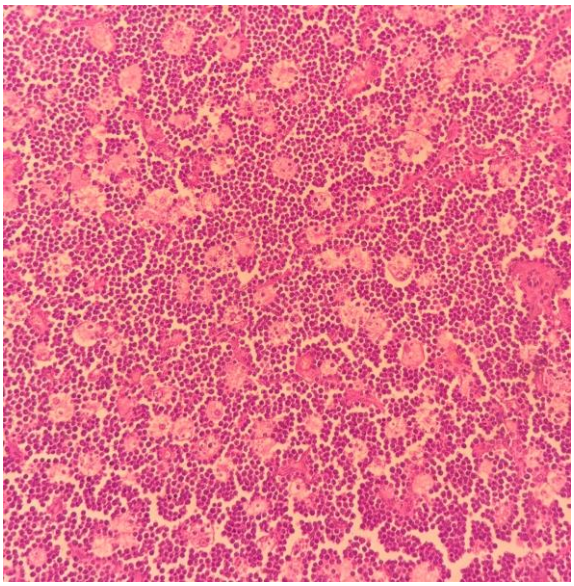

Figure 4: Inguinal lymph node biopsy (HE, high magnification). Monomorphic medium-sized lymphoid cells with prominent nucleoli, scant basophilic cytoplasm, and abundant mitotic figures, diagnostic of Burkitt lymphoma (Ki-67 ~100% expected on IHC)

DISCUSSION

This case exemplifies the catastrophic convergence of an AIDS-defining malignancy with multiple life-threatening opportunistic infections as the inaugural presentation of HIV, a clinical scenario that is exceptional and carries an extremely poor prognosis. Burkitt lymphoma in the HIV context arises from the interplay of chronic HIV-induced B-cell stimulation, EBV co-infection (serology pending at reporting), and immune surveillance failure. The characteristic histological pattern — diffuse monomorphic B-cell infiltrate with starry-sky appearance, high mitotic index, and apoptotic activity — combined with the biological profile (LDH 8× normal, hyperuricemia, thrombocytopenia, hypoalbuminemia) is consistent with extreme tumor burden and probable spontaneous tumor lysis syndrome, requiring aggressive preventive management before any cytotoxic treatment. The multinodular hepatic involvement on MRI most likely represents lymphomatous infiltration given confirmed BL and its known propensity for extra-nodal spread; the pancreatic lesion requires histological confirmation to distinguish lymphomatous infiltration from a primary pancreatic tumor. The concurrent invasive aspergillosis — a hallmark of profound cellular immunodepression — with a viral load exceeding 10 million copies/mL indicates prolonged undetected HIV infection with severe and durable immune collapse. The therapeutic sequencing followed current guidelines: antifungal-first to prevent IRIS before ART initiation, with a two-week

deferral consistent with WHO, DHHS, and EACS recommendations for invasive fungal co-infections. TDF/3TC/DTG (TLD) was selected as the first-line ART regimen per Moroccan national guidelines and WHO recommendations, with universal free access guaranteed by the Ministry of Health and co-financed by the Global Fund to Fight AIDS, Tuberculosis and Malaria. The fatal outcome three weeks after chemotherapy initiation reflects the cumulative burden of extreme tumor proliferation, immunological vulnerability, hepatic and renal involvement, nutritional depletion, and the inherent limitations of intensive chemotherapy infrastructure in a regional setting. This case advocates for systematic early HIV diagnosis — the only intervention capable of preventing such immune collapse — and for the routine consideration of lymphoma in any HIV-infected patient presenting with peripheral lymphadenopathy, elevated LDH, unexplained cytopenias, or B symptoms.

CONCLUSION

We report a fatal case of Burkitt lymphoma simultaneously unmasking HIV infection alongside invasive aspergillosis and candidiasis — a rare and catastrophic convergence of AIDS-defining conditions. This case reinforces three imperatives: systematic lymphoma screening in HIV-infected patients with lymphadenopathy or elevated LDH; structured therapeutic sequencing to minimize competing toxicities; and multidisciplinary oncology-infectious disease collaboration. Universal free ART access, supported by the Moroccan Ministry of Health and the Global Fund, is essential — but can only be effective when HIV diagnosis occurs early enough to prevent the irreversible immune collapse illustrated here.

Patient Consent

Patient data have been anonymized per ethical standards for case report publication (CARE guidelines).

Conflict of Interest

The author declares no conflict of interest.

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