Cardiac mass in a patient with HIV infection: A case report and literature review

ADRIAN GRADINARU[1], ANDREI COLITA[2], IONELA MURARETU[1], MIRCEA BAJDECHI[3], ANCA MIHAILESCU[1], CARMEN SAGUNA[2], RADOI ROXANA[4] and ADRIANA-LUMINITA GURGHEAN[1]

[1]Department of Medical Semiology, Coltea Clinical Hospital, Faculty of Medicine, Carol Davila University of Medicine and Pharmacy, Bucharest 050474, Romania; [2]Department of Hematology, Coltea Clinical Hospital, Faculty of Medicine, Carol Davila University of Medicine and Pharmacy, Bucharest 050474, Romania; [3]Faculty of Medicine, Ovidius University of Constanta, Constanta 900470, Romania; [4]First Department, Infectious Diseases, Dr. Victor Babes Clinical Hospital of Infectious and Tropical Diseases, Bucharest 030303, Romania

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Abstract. Hematological malignancies have multiple complications depending on their type and treatment, ranging from thromboembolic events to myocardial infarction and cardiac metastases. Cardiac masses can be benign or malignant (primary or secondary). When diagnosed, malignant cardiac tumors are usually metastatic in nature and are observed in patients with advanced neoplasia or disseminated disease. Depending on the subgroup of patients, some hematological malignancies have a higher incidence compared with the general population, such as patients living with HIV infection (PLWHIV). In this subgroup of patients, lymphomas, mainly the non-Hodgkin lymphoma subtypes, including large B-cell lymphoma and Burkitt lymphoma (BL), are the most frequent malignant tumors identified. These tumors may occur even in PLWHIV with a normal CD4$^+$ cell count and are highly aggressive tumors that, even though not frequently, may lead to cardiac metastases, highlighting the need for a high degree of suspicion, rapid diagnosis and treatment initiation in these patients. The present study encompasses a literature review on cardiac BL and a representative case of a 57-year-old man with a history of HIV infection, with a preserved CD4$^+$ cell count, who was diagnosed with BL with secondary cardiac involvement. The rapid growth of the tumor was outlined in sequential echocardiographic evaluations and a multi-agent chemotherapy regimen was initiated. The treatment was well tolerated, with a notable reduction in the cardiac mass and no cardiovascular complications associated with treatment during monitoring of the patient. A literature review was conducted to identify all the documented cases of adult cardiac BL of the past 15 years, outlining the low prevalence and high risk of these lymphoid tumors and main diagnostic methods, highlighting the importance of early imaging and multidisciplinary management of these rare but life-threatening cases.

Correspondence to: Dr Ionela Muraretu, Department of Medical Semiology, Coltea Clinical Hospital, Faculty of Medicine, Carol Davila University of Medicine and Pharmacy, 8-T Eroii Sanitari Boulevard, Bucharest 050474, Romania
E-mail: ionela-raluca.craciun@drd.umfcd.ro

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Introduction

Hematological neoplasms lead to multiple cardiovascular complications depending on their type and mechanism. In patients with myeloproliferative malignancies, the outcome is influenced not only by the disease itself, but also by severe cardiovascular complications. The most frequent complications are thrombotic events, pulmonary hypertension, accelerated atherosclerosis leading to ischemic heart disease and heart failure (1). Patients with lymphoproliferative malignancies have a greater risk of chemotherapy-associated cardiomyopathy, heart failure and arrhythmia compared with the general population, effects that are related either to the primary malignancy or to the specific treatment (anthracyclines, tyrosine kinase inhibitors, Bruton's tyrosine kinase inhibitors, stem cell transplantation and radiation therapy) (2). Venous and arterial thrombosis, as well as intracardiac thrombosis, are usually found in myeloproliferative neoplasms, mainly in polycythemia vera (PV) (3), with thrombosis being the leading cause of morbidity and mortality globally in PV and essential thrombocythemia (4), while cardiac metastases are usually identified in hematological tumors of the lymphoid line (that is lymphoproliferative neoplasms, mainly lymphomas) (5).

Cardiac neoplasms can be either primary or secondary, with the most common tumors identified being metastatic (6). Among primary tumors, ~90% are benign (including atrial myxomas and lipomas) (7). Secondary cardiac tumors are of various histological types, with lung adenocarcinoma being the most frequent cardiac metastasis identified in autopsy studies (14.6%), followed by poorly differentiated lung carcinoma (12.4%), squamous cell lung carcinoma (11.8%) and lymphomas/leukemias (10.1%) (8).

Secondary cardiac involvement in lymphomas accounts for ~10% of cardiac metastases (8), although the percentages vary (between 8.7 and 20%), as identified by previous autopsy

studies (8,9). Secondary cardiac lymphomas are more frequent in patients with disseminated disease, rarely presenting as primary cardiac tumors (10,11). Of these, the most frequent lymphomas identified are B-cell lymphomas, with the diffuse large B-cell lymphoma (DLBCL) subtype of non-Hodgkin lymphoma (NHL) being the most frequent (11,12). Burkitt lymphoma (BL), another subtype of NHL, is a rare and aggressive B-cell lymphoma, characterized by rapid cell proliferation (12), with a relatively low prevalence in the general population (~0.8% of all adult B-cell lymphomas) (5). However, their prevalence increases markedly in patients who are HIV-positive, with BL being the second most common type of NHL after DLBCL in this subgroup of patients (13).

Patients living with HIV infection (PLWHIV) are at an increased cardiovascular risk compared with the general population. A high prevalence of traditional and specific risk factors (chronic inflammation, endothelial dysfunction, immune dysregulation and antiretroviral therapy) can lead to accelerated atherosclerosis and, consequently, to ischemic heart disease (14). The symptomatology of acute coronary syndromes in these patients can be atypical, creating diagnostic issues. PLWHIV can develop other cardiac conditions, such as pericardial disease, endocarditis, dilated cardiomyopathy and pulmonary hypertension (15,16). The increased incidence of cardiovascular disease in this population outlines the need for a higher degree of suspicion and cardiovascular screening in these patients, especially concerning the early detection and treatment of conditions such as myocardial infarction, heart failure, hypertension or arrhythmia, all highly prevalent in this population group (16). Additionally, lifestyle optimization and the primary prevention of cardiovascular disease in PLWHIV need to be considered to prevent these secondary outcomes (16).

HIV infection, in addition to the aforementioned particular risk factors and secondary cardiovascular effects, affects the development of hematological and non-hematological neoplasms, with >40% of HIV-positive individuals developing acquired immunodeficiency syndrome (AIDS)-associated lymphomas (13). Even though the incidence of malignant tumors is higher in HIV-positive individuals, some [such as Kaposi sarcoma, a multicentric tumor caused by infection with the oncovirus Kaposi sarcoma-associated herpesvirus (KSHV)], have a risk inversely related to the $CD4^+$ count, while others (such as BL), even though slightly more prevalent in the AIDS stage of the infection, frequently appear in patients with a normal $CD4^+$ cell count and are not associated with specific oncoviruses (13,17,18). However, lymphoproliferative disorders, mainly lymphomas, are the most common malignancies in PLWHIV, who exhibit either a high or low T-helper cell count (17).

Hodgkin lymphomas (HLs) and NHL are the main types of HIV-associated malignancies, with NHL being the most common and the leading cause of HIV-associated mortality globally (13). In PLWHIV, 25-30% of NHL are BL or Burkitt-like lymphomas (BLLs), being the second most common subtype of NHL after DLBCL (13). BL and BLL are also frequently identified and should be taken into consideration in patients with a normal $CD4^+$ cell count, with the risk of developing BL in PLWHIV being more strongly associated with cumulative HIV viremia (19,20), outlining the need for a high degree of suspicion even in patients who are not in the AIDS stage of HIV infection (13,18). Oncoviruses, as aforementioned, also have an important role in the development of HIV-associated lymphomas, with Epstein-Barr virus (EBV) and KSHV being responsible for different subtypes of lymphomas in PLWHIV (20). In these patients, the uncontrolled proliferation of virus-infected cells and expression of viral oncogenic proteins is induced by HIV-triggered immunosuppression (20). While EBV latency type II [expressing EBV nuclear antigen 1 (EBNA1) and latent membrane proteins (LMP)-1 and -2] leads to an increased risk of development of HL in HIV-infected individuals, other oncoviruses such as KSHV/human herpesvirus 8 (HHV8) are linked to NHL, particularly DLBCL or primary effusion lymphoma (PEL) (20). BLs are not linked with these and often appear sporadically in PLWHIV, more likely secondary to cumulative HIV viremia, as aforementioned or occasionally in association with EBV latency type I (expressing only EBNA1, without expressing LMP1 or LMP2) (20).

Despite being one of the most common lymphomas diagnosed in PLWHIV, BL rarely represents a cause for intracardiac masses, with limited cases presented in the literature and with the disseminated form of the disease being more common than the primary cardiac form (21-23). While there is no clear or specific molecular mechanism identified yet underlying cardiac metastasizing in BLs found in PLWHIV, cardiac involvement potentially arises secondary to numerous mechanisms. Firstly, early hematogenous spread appears secondary to the high circulating tumor burden in BLs, caused by MYC dysregulation, which drives rapid proliferation in tumor cells (reflected by a markedly high Ki-67 index of >95%) and by the loss of immune surveillance found especially in PLWHIV; and, secondly, through transvenous extension from caval or mediastinal disease, explaining the preference for the right atrium when it comes to secondary cardiac masses in these patients (5,13,21,24).

The present report encompasses an illustrative case of cardiac involvement in a patient with BL and HIV infection, highlighting the diagnostic challenges, management and clinical course of such cases.

Case report

The present study describes the case of a 57 year-old man, known to have immunological class B2 HIV infection diagnosed 1.5 years prior to the current presentation, who has been treated since diagnosis with antiretroviral therapy (ART) consisting of a combination of doravirine, lamivudine and tenofovir disoproxil, with a $CD4^+$ count of 236 cells/mm^3 (13.11%) (normal range, 500-1,500 cells/mm^3), $CD8^+$ count of 1,175 cells/mm^3 (65.30%) (normal range, 170-1,000 cells/mm^3) and a $CD4^+/CD8^+$ fraction of 0.20 (normal range, 1-3), 1 month prior to presentation, suggestive of moderate-severe immunosuppression. The HIV infection history of the patient was notable due to a $CD4^+$ count of 349 cells/mm^3 and a $CD4^+/CD8^+$ fraction of 0.20, with a HIV viremia of 117 viruses/ml shortly after ART initiation, suggestive of moderate immunosuppression. The patient presented in April 2025 to the emergency room of Coltea Clinical Hospital (Bucharest, Romania) with rapidly growing (~3 months), painful, round, soft and adherent

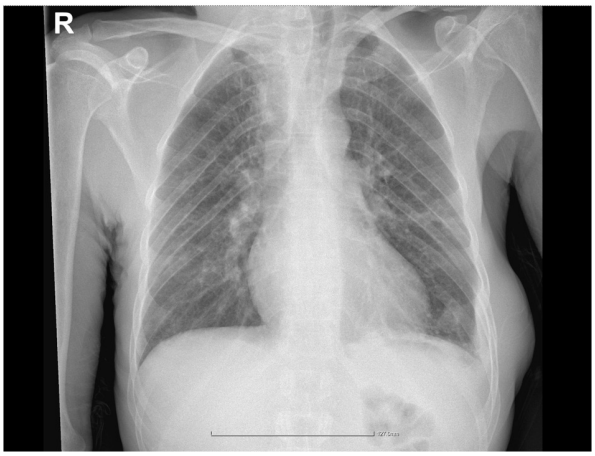

Figure 1. Chest X-ray showing left basal pleural effusion and right upper mediastinum enlargement with tracheal compression. Scale (horizontal bar), 127.6 mm. R, right side.

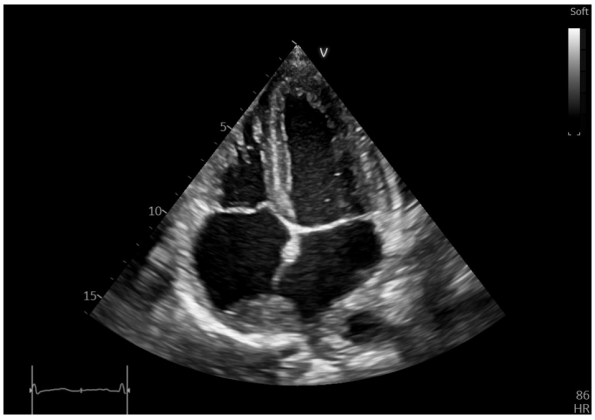

Figure 2. Echocardiography at the initial evaluation (apical four chamber view) revealing an atrial mass that infiltrates the roof of the right and left atrium as well as the right upper pulmonary vein. HR, heart rate.

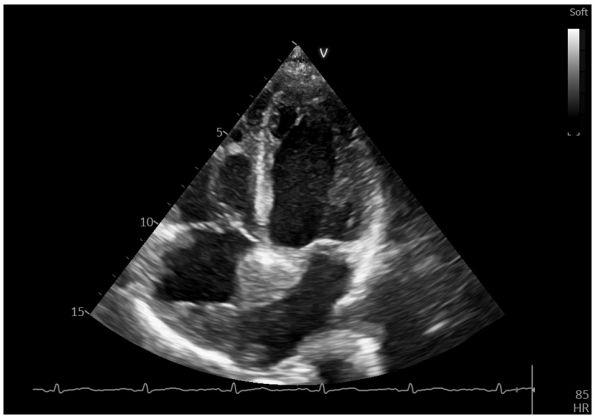

Figure 3. Echocardiography at the initial evaluation (modified apical four chamber view) revealing an atrial mass that occupies 1/3 of the right atrial area and interatrial septum. HR, heart rate.

left latero-thoracic and right latero-cervical subcutaneous masses, measuring 12x7 cm (latero-thoracic) and 9x5 cm (latero-cervical) in size. Clinical examination revealed

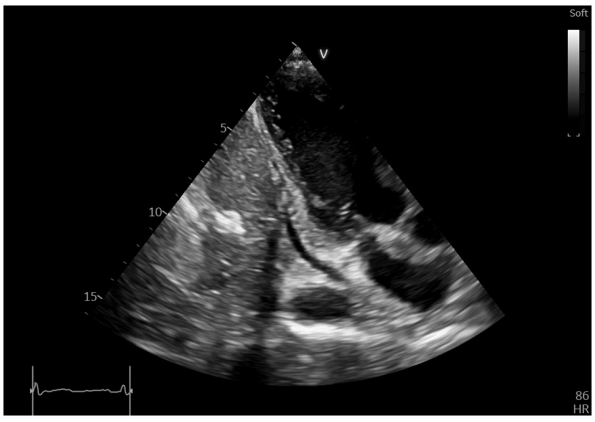

Figure 4. Echocardiography at the initial evaluation (apical three chamber view) showing the atrial mass extension to the anterior wall of the left atrium, with a noticeable thickened appearance. HR, heart rate.

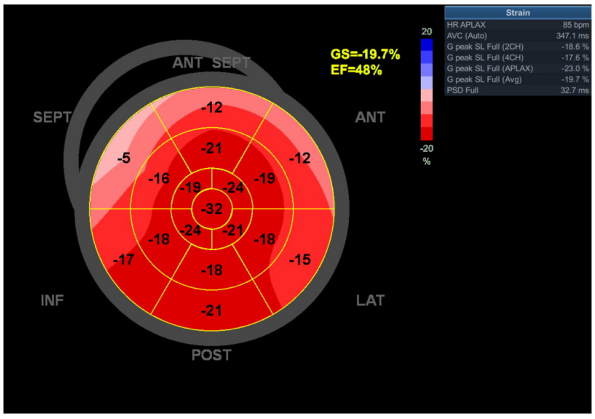

Figure 5. Echocardiography bull's-eye plot at the initial evaluation showing a slightly reduced global strain and ejection fraction, localized at the level of the basal segments of the antero-lateral, antero-septal and septal walls and interventricular septum. SEPT, septal wall; ANT, anterior; LAT, lateral wall; POST, posterior; INF, inferior; GS, global longitudinal strain; EF, ejection fraction; AVC, aortic valve closure; HR, heart rate; APLAX, apical long-axis; PSD, peak strain dispersion; G peak SL, global peak longitudinal strain.

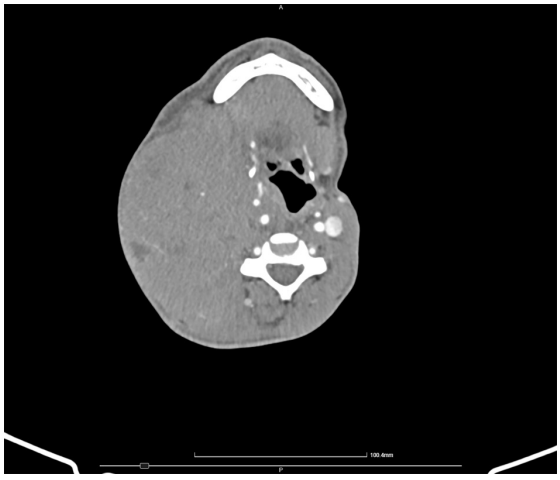

Figure 6. CT scan showing a cervical mass with laryngeal compression, with compression and stenosis of the right (left side of the image) common carotid artery, internal carotid artery and external carotid artery and apparent extrinsic obstruction of the right internal jugular vein. Scale (horizontal bar), 100.4 mm.

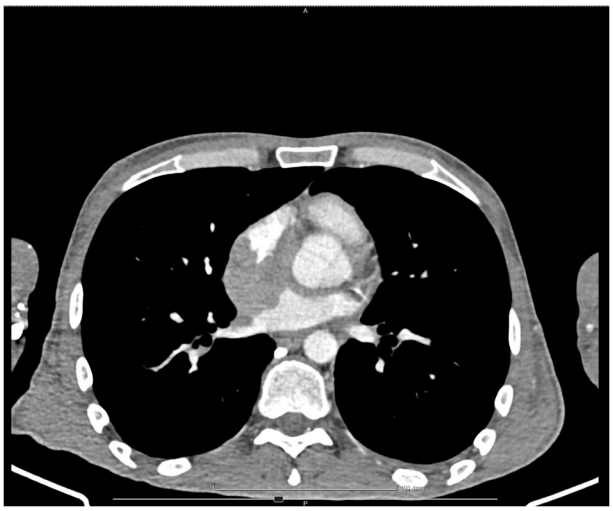

Figure 7. CT scan showing a right atrial mass that infiltrates the left atrium, interatrial septum and right upper pulmonary vein, surrounding the aortic root. Scale (horizontal bar), 100.4 mm.

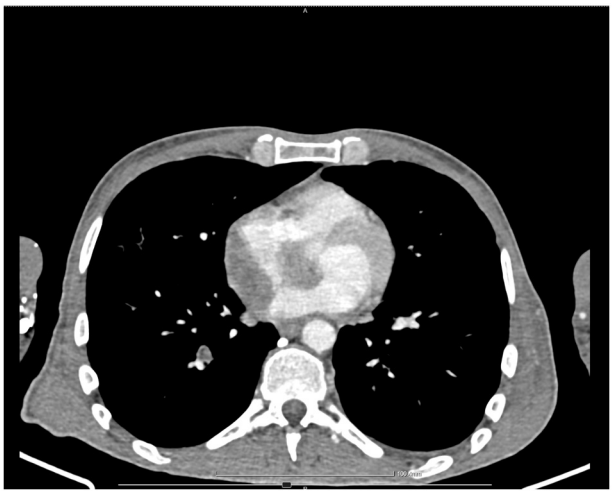

Figure 8. CT scan showing that the right atrial mass infiltrates the interatrial septum, roof of the right atrium and upper right pulmonary vein. Scale (horizontal bar), 100.4 mm.

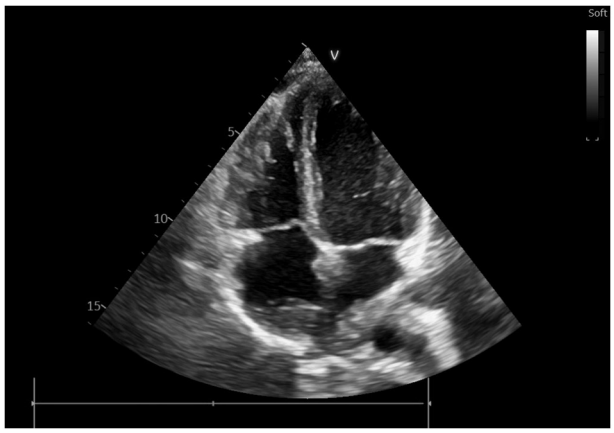

Figure 9. Echocardiography at 1 week after the initial evaluation (apical four chamber view) revealing that the atrial mass exhibits an increase in size, infiltrating the roof of the right atrium, interatrial septum, left atrium and the right upper pulmonary vein.

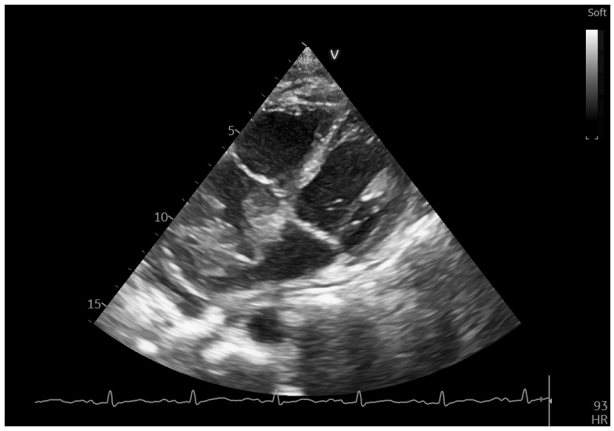

Figure 10. Echocardiography at 3 weeks after the initial evaluation (modified apical four chamber view) revealing growth of the tumoral mass, infiltrating the right atrial area, the left atrium and interatrial septum and extending to the base of the left and right ventricles. HR, heart rate.

tachycardia with an irregular heart rate (HR) of 140 bpm, skin pallor, nail clubbing, absent lung sounds in the left pulmonary base, no crackles on pulmonary auscultation, normal blood pressure, blood oxygen saturation of 96%, normal respiratory rate, light exertional dyspnea and no palpitations, dizziness or other complaints at the moment of presentation. The patient also mentioned an unintentional weight loss of ~10% in the past 2 years as well as night sweats.

Considering the clinical presentation and patient history, laboratory tests were ordered, along with an electrocardiogram (ECG) and a chest X-ray. The laboratory tests revealed a normal white blood cell count [5,720 cells/μl (4,000-11,000 cells/μl normal range), including 35% lymphocytes (20-45% normal range) and 56% neutrophiles (43-65% normal range)], mild microcytic anemia [hemoglobin, 10.3 g/dl (14-17 g/dl normal range); mean corpuscular hemoglobin, 22.5 pg (27-33 pg normal range); and mean corpuscular volume, 74.3 fl (80-96 fl normal range)] with no iron deficit [decreased total iron binding capacity of 219 μmol/dl (250-450 μmol/dl normal range) with increased iron saturation of 40% (16-25% normal range) and increased ferritin], mild thrombocytosis [platelet count, 454,000 cells/μl (150,000-400,000 cells/μl normal value)], inflammatory syndrome with an increase in all inflammatory biomarkers measured [C-reactive protein (CRP), 9.4 mg/dl (normal value <0.3 mg/dl); erythrocyte sedimentation rate, 86 mm/h (3-8 mm/h normal range); fibrinogen, 596 mg/dl (150-400 mg/dl normal range); and ferritin, 870 ng/ml (24-260 ng/ml normal range)] and an elevated N-terminal pro B-type natriuretic peptide (NT-proBNP) value, suggestive of heart failure [2,260 pg/ml (0-300 pg/ml normal range)]. An ECG revealed atrial fibrillation with a rapid ventricular response [heart rate (HR), 140 bpm], with a QRS complex duration of <100 msec, diffuse QRS microvoltage and no notable repolarization abnormalities nor pathological Q waves.

Furthermore, a chest X-ray revealed left basal pleural effusion and right upper mediastinum enlargement with secondary tracheal compression (Fig. 1).

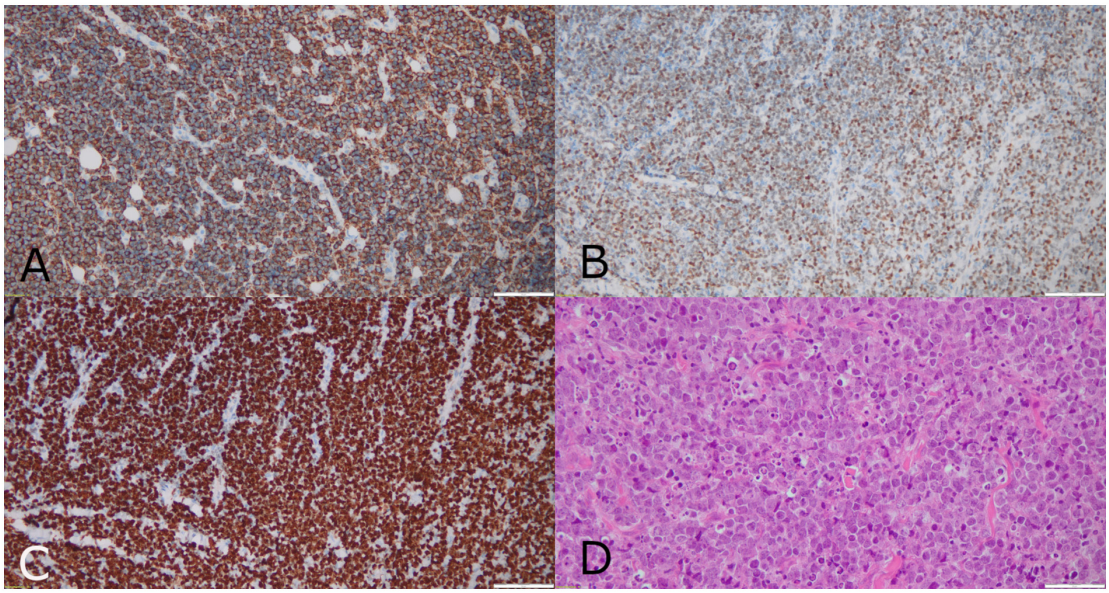

Figure 11. Representative histopathology and immunophenotype supporting the diagnosis of Burkitt lymphoma. (A) Ki-67 proliferation index of ~98%. (B) CD20 highlights diffuse B-cell phenotype. (C) Nuclear c-Myc expression in ~70% of tumor cells(D) Standard H&E staining showing diffuse B-cell infiltrate with strong basophilia and multiple prominent basophilic nucleoli, with multiple mitoses [(A-C) magnification, x20; scale bar, 100 μm; (D) magnification, x40; scale bar, 50 μm].

Considering these findings (newly diagnosed atrial fibrillation, diffuse QRS microvoltage on the ECG and an increased NT-proBNP value) echocardiography was performed, which revealed an irregular, diffusely hyperechoic, immobile upper atrial mass, which occupied ~1/3 of the right atrial (RA) area (4.2 cm^2, with an RA area of 16.9 cm^2) and infiltrated the interatrial septum, extending to the anterior wall and roof of the left atrium (LA), also involving the right upper pulmonary vein (Figs. 2-4), a small pericardial effusion and mild tricuspid regurgitation, with no apparent flow obstruction at the level of the tricuspid valve.

Functionally, the left ventricle demonstrated normal filling pressures, but a left ventricular global longitudinal strain (GLS) of -19.7% with a slightly reduced ejection fraction (EF) of 48% (Fig. 5). The slight reduction in GLS and EF, without any specific segmentary distribution, suggesting an ischemic etiology, was suspected to be due to tumoral infiltration. As a consequence, a CT scan was ordered to further understand the tumoral infiltration extent and distribution.

The whole-body CT showed multiple large masses, including: i) A right lateral cervical mass of ~8.3x12x18 cm, with a diffuse iodine uptake, necrosis areas and pharyngeal, laryngeal, tracheal and thyroid compression, alongside compression and stenosis of the right common, internal and external carotid arteries and apparent extrinsic obstruction of the internal jugular vein (Fig. 6); ii) a left latero-thoracic mass of 8.4x4x11 cm with diffuse iodine uptake and necrosis areas; and iii) an RA mass, which infiltrated the LA and the right upper pulmonary vein, extending to the interatrial septum, surrounding the aortic root and further extending to the base of the interventricular septum and right ventricle (Figs. 7 and 8). No central nervous system (CNS) involvement was identified, and neither any other notable findings on the CT scan. Considering the cardiac infiltration observed on the CT scan, which extended to the base of the left and right ventricles, the slight reduction in EF and the reduced GLS observed at the level of the basal segments of the antero-lateral, antero-septal and septal walls as well as the interventricular septum was interpreted in the context of tumoral infiltration.

Considering these findings, anticoagulant therapy was initiated with a direct oral coagulant, due to the high thromboembolic risk in the context of atrial fibrillation (apixaban, 5 mg twice daily, with no interactions with ART) and a β-blocker (metoprolol) for rate control. Additionally, a discussion was held regarding the addition of a sodium-glucose cotransporter-2 [inhibitor to the treatment regimen (dapagliflozin or empagliflozin, sodium-glucose cotransporters used in treating type 2 diabetes and heart failure)] according to the current guideline recommendations and considering the presence of heart failure (25); however, after careful consideration of the potential benefits (a reduction in heart failure mortality and hospitalization) and risks (increased risk of urinary tract infections in the context of HIV co-infection with moderate-severe immunosuppression), it was decided, in accordance with the wishes of the patient, to temporarily withhold the addition of dapagliflozin or empagliflozin to the treatment regimen until an improved immune control was obtained with ART (changed from doravirine, lamivudine and tenofovir disoproxil to bictegravirum/emtricitabine/tenofovir alafenamide after hematological diagnosis and chemotherapy initiation).

An incisional biopsy of the right latero-cervical mass was made and the fragment was sent for histopathological and immunohistochemical analyses. The patient was closely monitored clinically and biologically awaiting the results, with echocardiographic evaluations repeated weekly. After 1 week from the initial evaluation, the patient exhibited an increase in size and discomfort in the latero-thoracic and latero-cervical regions, with notable growth of the cardiac mass observed during echocardiographic re-evaluation, and no other morphological or functional changes observed compared with the

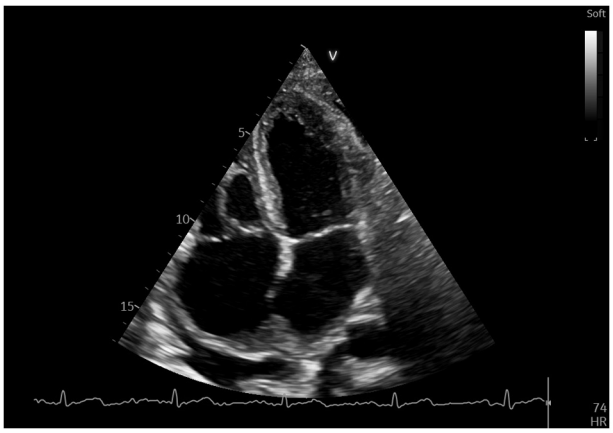

Figure 12. Echocardiographic apical four chamber view (5 days after chemotherapy initiation) revealing notable decrease in size of the tumoral mass is observed, which now occupies the roof of the right and left atrium and the upper portion of the interatrial septum. HR, heart rate.

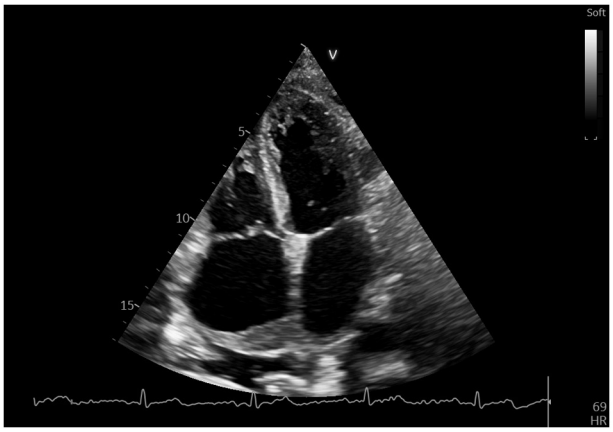

Figure 13. Echocardiographic slightly modified apical four chamber view made 5 days after chemotherapy initiation showing a remnant of the tumoral mass, observed at the base of the interatrial septum; however, it was much smaller in comparison with the evaluation before chemotherapy initiation. HR, heart rate.

initial evaluation (Fig. 9). The conversion to sinus rhythm was obtained spontaneously, without the need for rhythm control therapy, and sinus rhythm was maintained throughout subsequent evaluations, although certain changes in repolarization were observed on subsequent ECG evaluations, as the tumoral mass expanded in size (negative T waves in V1-V3), with no other repolarization abnormalities or pathological Q waves and a right bundle branch block (RBBB) QRS morphology (rsR' aspect of the QRS segment, suggestive of incomplete RBBB), with a QRS complex <100 msec and a slightly prolonged corrected QT (QTc) interval of 458 msec (corrected with the Fridericia formula: $QTc=QT/RR^{1/3}$). Additionally, a decrease in NT-proBNP levels shortly after spontaneous restoration of the sinus rhythm (786 pg/ml) was observed.

A subsequent echocardiographic follow-up evaluation at 3 weeks after the initial one, while awaiting the tissue immunohistochemical analysis results, revealed a marked increase in size of the RA mass, which now occupied >1/2 of the RA area (9.4 cm^2 with an RA area of 16.6 cm^2), infiltrating almost entirely the interatrial septum and more of the anterior wall, as well as the roof of the LA and right upper pulmonary vein (Fig. 10), demonstrating a notably rapid increase in the size of the cardiac tumor, but well tolerated at the time, with no apparent flow obstruction or further changes in cardiac function. However, the rapid growth of the latero-thoracic and latero-cervical masses made echocardiographic assessment difficult, and the aggressive growth of the cardiac mass highlighted the need for a quick, definitive diagnosis and the initiation of therapy.

Tissue immunohistochemical analysis was performed, using the following procedure: Sections with a thickness of 2.5 μm obtained from formalin-fixed paraffin-embedded (FFPE) tissue samples were cut and affixed to positively charged microscope slides. Subsequently, these slides were subjected to a heating process at a temperature of 60˚C for 1 h in a dry oven, thereby enhancing the adhesion of the tissue and softening the paraffin. The immunohistochemistry procedure was conducted as per established protocol using a Ventana BenchMark ULTRA autostainer (Roche Tissue Diagnostics). The sections went through steps of deparaffinisation, rehydration and antigen retrieval with Ventana's CC1 (prediluted, ph 8.0) solution (Roche Tissue Diagnostics). Primary antibodies were applied according to the manufacturer's dilution guidelines and permitted to interact with the sections. The primary antibodies utilized in this study included the following: Anti-c-MYC (cat. no. 790-4628) Rabbit Monoclonal Primary Antibody (ready-to-use; Roche Diagnostics GmbH), CONFIRM anti-bcl-2 (cat. no. 790-4464) Mouse Monoclonal Primary Antibody (ready-to-use; Roche Diagnostics GmbH), CONFIRM anti-Ki-67 (cat. no. 790-4286) Rabbit Monoclonal Primary Antibody (ready-to-use; Roche Diagnostics GmbH), CONFIRM anti-CD20 (cat. no. 760-2531) Primary Antibody (ready-to-use; Roche Diagnostics GmbH), CONFIRM anti-CD3 (cat. no. 790-4341) Rabbit Monoclonal Primary Antibody (ready-to-use; Roche Diagnostics GmbH), CD138/syndecan-1 (cat. no. 760-4248) Mouse Monoclonal Antibody (ready-to-use; Roche Diagnostics GmbH), VENTANA anti-CD10 (cat. no. 790-4506) Rabbit Monoclonal Primary Antibody (ready-to-use; Roche Diagnostics GmbH), bcl-6 (cat. no. 760-4241) Mouse Monoclonal Antibody (ready-to-use; Roche Diagnostics GmbH), MUM1 (cat. no. 760-6082) Rabbit Monoclonal Primary Antibody (ready-to-use; Roche Diagnostics GmbH), Epstein-Barr Virus (cat. no. 760-2640) Mouse Monoclonal Antibody (ready-to-use; Roche Diagnostics GmbH), HHV-8 (cat. no. 760-4260) Mouse Monoclonal Antibody (ready-to-use; Roche Diagnostics GmbH) and anti-CD30 (cat. no. 790-4858) Mouse Monoclonal Primary Antibody (ready-to-use; Roche Diagnostics GmbH). The incubation periods were established as 16 min for MYC and Ki67, and 32 min for BCL2, BCL6, CD10, CD20, CD3, MUM1, CD138, EBV-LMP1, HHV-8 and CD30. The visualization process was conducted using the Ultraview universal DAB IHC detection kit (cat. no. 760-500; Roche Diagnostics GmbH), followed by counterstaining utilizing hematoxylin II for 8 min and a bluing solution for 8 min at room temperature in the automated BenchMark ULTRA (cat. no. 750-600; Roche Diagnostics GmbH). The slides were meticulously cleaned and dehydrated through a graded series of ethanol and xylene. Finally, the slides were affixed with mounting media onto

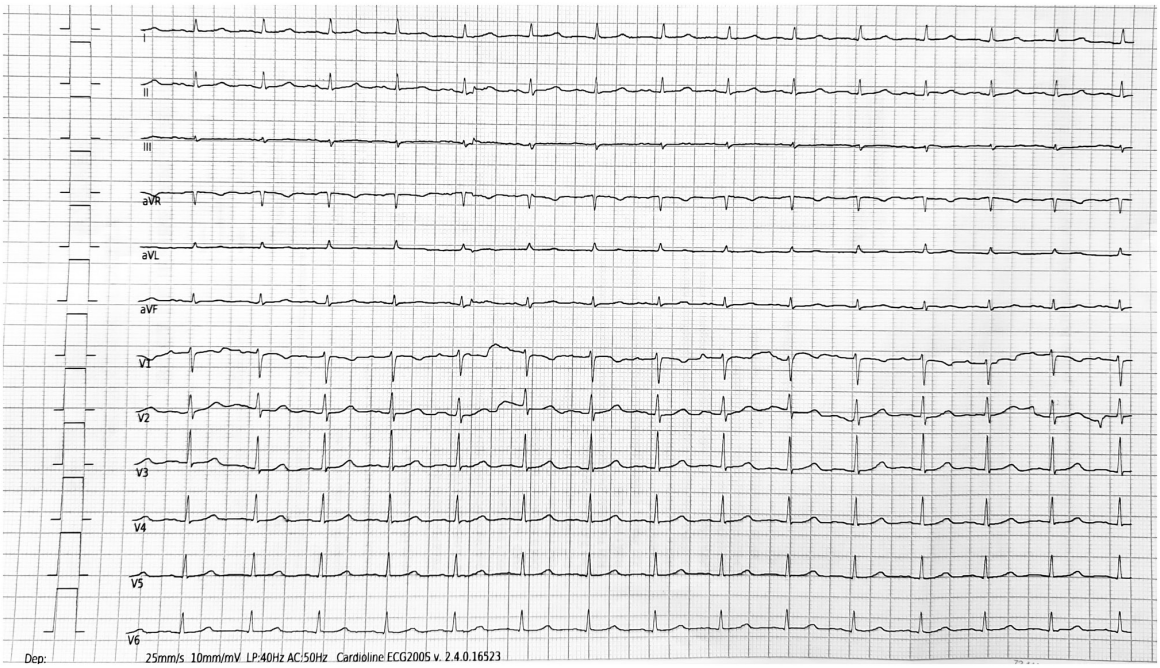

Figure 14. Post-chemotherapy electrocardiogram showing sinus rhythm, a heart rate of 90 bpm, remission of the right bundle branch block morphology, remission of the negative T waves in V2-V3, diffuse QRS microvoltage (showing in this case loss of electrical signal caused probably by tumoral infiltration) and normalization of the corrected QT interval (435 msec, Fridericia formula). V1-V6 refer to precordial leads. aVR, augmented vector right; aVL, augmented vector left; aVF, augmented vector foot; LP, low pass; AC, alternating current.

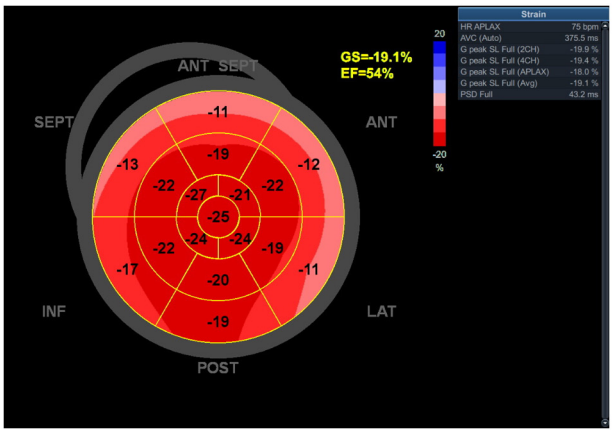

Figure 15. Echocardiography bull's-eye plot recorded after 3 cycles of chemotherapy showing slightly reduced GS, with minimal dyskinesia localized at the level of the basal segments of the antero-lateral, antero-septal, septal and lateral walls and preserved ejection fraction. SEPT, septal wall; ANT, anterior; LAT, lateral wall; POST, posterior; INF, inferior; GS, global longitudinal strain; EF, ejection fraction; AVC, aortic valve closure; HR, heart rate; APLAX, apical long-axis; PSD, peak strain dispersion; G peak SL, global peak longitudinal strain.

microscope slides. The subsequent analysis showed a CD20 diffusely positive, CD10 positive, B cell lymphoma 6 (BCL6) positive, multiple myeloma oncogene 1 (MUM1) negative, CD30 and BCL2 negative, and c-Myc 70% positive tissue (Fig. 11A and B). Positivity for CD3 was observed in frequent small intratumoral T cells, while CD138 was positive in rare intratumoral plasmacytes and negative in tumoral cells. The tissue was negative for EBV-LMP1 and HHV8 (thus excluding other oncovirus-driven hematological malignancies identified in PLWHIV, such as DLBCL, PEL or HL and supporting a diagnosis of BL), with a very high proliferation index Ki-67 of ~98%, helping differentiate BL from DLBCL, which has a lower index of proliferation (Fig. 11C) (26).

The identified malignant cells also possessed strong basophilia and multiple prominent basophilic nucleoli and multiple mitoses observed under standard histological analysis, performed according to the following procedure by the Pathology Department: H&E staining was performed on 2.5-μm FFPE sections. Tissue was fixed in 10% neutral-buffered formalin, processed through graded ethanol and xylene and embedded in paraffin. Sections mounted on charged slides were dewaxed in xylene, rehydrated through descending alcohol concentrations and stained with hematoxylin (0.1% solution) for 5 min, followed by bluing in water, and counterstaining with eosin (0.5% solution) for 2 min, at room temperature. Slides were then dehydrated in 70, 95 and 100% ethanol, cleared in xylene and coverslipped. Histological evaluation was performed using a standard light microscope at x20 and x40 magnification (Fig. 11D). Thus, considering the presence of frequent basophilic monomorphic cells with prominent basophilic nucleoli on histological analysis, along with the diffusely positive CD20, positive BCL6 (Fig. S1A), intensely positive CD10 (Fig. S1B), negative MUM1 (Fig. S1C) and negative BCL2 (Fig. S1D), with a positive c-Myc of 70% and the high index of proliferation, close to 100% (Ki67 of 98%), a diagnosis of non-Hodgkin BL as BL international prognostic index 2 (27), high-risk secondary to HIV immunosuppression, was made (26). Chemotherapy with a hyperfractionated cyclophosphamide, doxorubicin, vincristine, methotrexate, cytarabine and dexamethasone (hyper CVAD) regimen was initiated considering the markedly high hematological risk of the patient according to the BL international prognostic index (namely, >40 years old, with a high index of tumoral

Table I. Documented cases of adult Burkitt cardiac lymphomas based on a PubMed search of the last 15 years (2010-2025).

First author, year	Age, years	Sex	Presenting symptoms	HIV status	Intracardiac mass location/s	Diagnostic modality	Treatment regimen	Outcome	(Refs.)
Tzachanis *et al*, 2014	44	F	Early satiety, abdominal bloating and epigastric discomfort	Negative	Right atrium	CT scan, TTE, pericardiocentesis and MRI	Cyclophosphamide, vincristine, doxorubicin and high-dose methotrexate; ifosfamide, etoposide and high-dose cytarabine; rituximab	Complete remission	(28)
Lazkani *et al*, 2015	38	M	Dyspnea, tachycardia and palpitations	Positive	Right atrium	CT scan, TTE and MRI	Rituximab, etoposide, prednisone, vincristine, cyclophosphamide and doxorubicin	Remission (6 weeks)	(29)
Chan *et al*, 2016	27	M	Palpitations and dizziness	Positive	Right atrium, right ventricular base and interatrial septum	CT scan, TTE and TEE	Modified cyclophosphamide, vincristine, doxorubicin, and methotrexate/ifosfamide; etoposide and cytarabine	Partial remission (12 weeks)	(21)
Vervloet *et al*, 2017	49	M	Dyspnea, chest pain and lower limb edema	Positive	Right atrium, left atrium and interatrial septum	X-ray, TTE and MRI	Methotrexate and fractionated high doses of ifosfamide; rituximab, cyclophosphamide, doxorubicin, vincristine and prednisone	Complete remission	(30)
Vilaça *et al*, 2017	64	M	Lipothymia, nausea and palpitations	Positive	Right atrium, right ventricle base and interventricular apical septum	CT scan, TTE and MRI	Prednisolone, vincristine, daunorubicin, L-asparaginase, rituximab and adaptive radiation therapy	Partial remission	(22)
Usry *et al*, 2020	80	F	Dyspnea	N/A	Right atrium, interatrial septum and inlet ventricular septum	TTE and CT scan	Rituximab, etoposide, prednisone, vincristine, cyclophosphamide and doxorubicin	Partial remission	(31)
Khalid *et al*, 2020	49	M	Dyspnea and palpitations	Positive	Right atrium and right ventricle base	TTE and CT scan	Rituximab, cyclophosphamide, vincristine, doxorubicin and methotrexate	Partial remission (complete response N/A)	(32)
Chen *et al*, 2020	68	M	Mild chest pain, nausea and wheezing	N/A	Right atrium	TTE, CT scan, MRI and PET-CT	Rituximab, etoposide, prednisone, vincristine, cyclophosphamide and doxorubicin	Complete remission	(33)

Table I. Continued.

First author, year	Age, years	Sex	Presenting symptoms	HIV status	Intracardiac mass location/s	Diagnostic modality	Treatment regimen	Outcome	(Refs.)
Martinez *et al*, 2021	77	F	N/A	N/A	Right atrium, left atrium and interatrial septum	PET-CT, EUS and CT scan	Rituximab, etoposide, prednisone, vincristine, cyclophosphamide and doxorubicin	Complete remission	(34)
Khaba *et al*, 2021	30	F	Dyspnea, non-productive cough and leg edema	Positive	Right atrium	X-ray and TTE	Modified cyclophosphamide, vincristine, doxorubicin, and methotrexate/ifosfamide; etoposide and cytarabine	Complete remission	(35)
Hérnandez Jimenez *et al*, 2021	65	F	Cough, fatigue, dyspnea and bilateral leg edema	Negative	Right atrium	TTE and MRI	None	Deceased (shortly after biopsy)	(36)
Schmiester *et al*, 2022	54	M	Paranasal sinus swelling, fatigue and dyspnea on exertion	Negative	Right ventricle and right atrium	CT scan, TTE and MRI	Cyclophosphamide, doxorubicin and prednisone; rituximab, vincristine, cytarabine, etoposide, methotrexate, ifosfamide and dexamethasone; intrathecal prophylaxis with cytarabine, dexamethasone and methotrexate	Complete remission	(37)
Rector *et al*, 2022	70	M	Constipation, eating discomfort and unintentional weight loss	Negative	Right atrium and right ventricle	CT scan, TTE and MRI	Vincristine, doxorubicin, dexamethasone and rituximab; etoposide, prednisone, vincristine, cyclophosphamide and hydroxydaunorubicin; intrathecal methotrexate without complication	Deceased	(38)

F, female; M, male; TTE, transthoracic echocardiogram; TEE, transesophageal echocardiogram; PET-CT, positron emission tomography-CT; EUS, endoscopic ultrasound.

progression and lactate dehydrogenase >3-fold the upper limit of the normal range, with a value of 879 U/l). The regimen included the following agents and dosing, and was administered according to the following protocol: On cycles 1, 3, 5 and 7 (3-4 weeks between cycles), 600 mg/m^2 cyclophosphamide was administered intravenously (IV) over 2 h every 12 h for six doses starting on day 1. Mesna (600 mg/m^2/day) was administred by continuous IV infusion on days 1-3, starting 1 h before, plus 2 mg vincristine IV on days 4 and 11, plus 50 mg/m^2 doxorubicin IV on day 4 and 40 mg dexamethasone orally on days 1-4 and 11-14. On cycles 2, 4, 6 and 8 (3-4 weeks between cycles), 200 mg/m methotrexate was administered IV over 2 h followed by 800 mg/m^2 IV over 22 h on day 1, plus 3 g/m^2 cytarabine IV over 2 h every 12 h for four doses starting on day 2, plus 15 mg leucovorin IV every 6 h for eight doses beginning 12 h after the completion of methotrexate infusion, and increased to 50 mg IV every 6 h if methotrexate levels were >20 μM/l at baseline, were >1.0 μM/l at 24 h or were >0.1 μM/l at 48 h after the end of methotrexate infusion, until levels were <0.1 μM/l. Methylprednisolone (50 mg) was administered IV every 12 h on days 1-3.

However, considering the markedly high cardiovascular risk according to the score developed by the Heart Failure Association in collaboration with the International Cardio-Oncology Society (HFA-ICOS) (25), namely, elevated baseline NT-proBNP, history of heart failure and a selected treatment plan with anthracyclines, the patient was closely monitored with repeated echocardiographic evaluations during chemotherapy initiation. The patient responded well to the regimen, with a notable reduction in all tumoral masses after chemotherapy initiation. In an echocardiographic evaluation made 5 days after chemotherapy initiation, the cardiac mass occupied <1/5 of the RA area (2.7 cm^2 with an RA area of 20.6 cm^2, right atrial dilation compared with the initial measurement and a marked tumor area reduction compared with the first and second evaluation), measured ~2.3x1.2 cm and was restricted to the roof of the RA and upper third of the interatrial septum, with minimal involvement of the LA roof and right upper pulmonary vein compared with pretherapeutic evaluation and no extension to the base of the right or left ventricles (Figs. 12 and 13).

After 2 weeks post-therapy initiation, a notable reduction in size of the latero-cervical and latero-thoracic tumoral masses was observed and the compression of the carotid arteries and the jugular vein subsequently remitted. The patient also demonstrated a notable improvement in symptoms, with remission of the pain that was present during initial and subsequent evaluations. The laboratory results exhibited a marked reduction in inflammatory syndrome biomarkers (CRP, 1.6 mg/dl and fibrinogen, 521 mg/dl) and a decrease in the platelet count (218,000 cells/μl), with the persistence of microcytic anemia (hemoglobin, 9 g/dl) and a reduction in white blood cell count (2,590 cells/μl, including 45% lymphocytes and 40.7% neutrophiles). The patient maintained the sinus rhythm and no other cardiovascular complications associated with treatment were observed during monitoring. The ECG showed a notable improvement compared with previous evaluations (namely, sinus rhythm with an HR of 90 bpm and no QRS morphology changes, indicative of remission of RBBB morphology) and the remission of the negative T waves in V2-V3, which became positive, with no other repolarization abnormalities and correction of the slightly prolonged QTc interval (now 435 msec with the Fridericia formula). However, a diffuse QRS microvoltage was still observed (Fig. 14).

There were no further changes in cardiac function or morphology observed in subsequent echocardiographic evaluations until discharge, which occurred 2 weeks after the initiation of the first cycle of the hyper CVAD protocol. The patient continued to be monitored after chemotherapy initiation for cardiovascular or systemic side effects through regular clinical evaluations every 2 weeks, laboratory tests, ECG monitoring and echocardiographic screenings. Furthermore, recommendations for the prevention of cardiovascular disease, including lifestyle changes, as well as the benefits and risks of the cardiovascular treatment prescribed, were offered, in addition to information regarding possible adverse cardiovascular effects of the chemotherapy regimen and the early identification of warning signs and symptoms requiring further evaluation (including sudden onset dyspnea or dyspnea aggravation, palpitations, dizziness, chest pain or lower extremity edema).

After three cycles of chemotherapy, there were no marked changes in infectious status (HIV immunological class C3 and moderate-severe immunosuppression after the hematological diagnosis and ART change) or cardiac tumor size compared with chemotherapy initiation, with the persistence of the small tumoral mass at the level of the RA roof, without any marked growth in size, but with an improved left ventricular function on longitudinal strain evaluation (including a GLS of -19.1%, with minimal anterior, septal and lateral basal dyskinesia and with a calculated EF of 54%, which was an increase from the initial 48%; Fig. 15). This was a marked improvement from the previous evaluation, perhaps secondary to tumor infiltration reduction at the level of the left ventricular base. The ECG objectified a slight reduction in the QRS microvoltage and sinus rhythm, with no repolarization abnormalities or QTc prolongation. However, a slightly increased NT-proBNP level was observed at the end of the third cycle of chemotherapy (1,810 pg/ml), without the presence of any symptoms or signs of heart failure, with no other cardiovascular complications to date.

Literature review and discussion

Hematological malignancies may have multiple cardiovascular complications, usually related to thrombosis, heart failure, cardiomyopathy or secondary cardiac metastases, which are either associated with the primary malignancy or with chemotherapy (1,2). These complications depend on the malignancy subtype, with fluid malignancies such as myeloproliferative neoplasms leading to an increased risk in thrombosis and lymphoid line neoplasms such as lymphomas occasionally leading to cardiac metastases (4,5).

Usually, cardiac tumors have a secondary origin, with primary cardiac tumors being benign in nature and rarely identified (6,7). Among cardiac tumors, lymphomas exhibit an incidence in the general population of ~10%, although the values vary between 8.7-20% according to previous autopsy studies (8,9). The majority of cardiac lymphomas appear secondary to disseminated disease and rarely present as primary cardiac malignancies (10,11). Even though lymphomas are not

the most frequent cardiac tumors identified, their prevalence increases markedly in PLWHIV, with >40% of HIV-positive individuals developing AIDS-associated lymphomas and thus having an increased risk of secondary cardiac metastases compared with the general population (13).

ART has notably improved the prognosis of PLWHIV in previous years. In the early ART era HIV infection had multiple secondary cardiovascular effects documented, such as pericarditis, endocarditis and dilated cardiomyopathy; nowadays, with improved immunological control, atherosclerotic heart disease has become the most important cardiovascular complication in these patients, mainly due to a higher prevalence of traditional risk factors such as chronic inflammation and endothelial dysfunction (16), leading to an increased risk in heart failure, arrhythmias, myocardial infarction or hypertension (15). However, the increased incidence of lymphomas, mainly NHL (with DLBCL being the most frequent subtype, followed by BL) (13), needs to be considered due to the rapid tumor growth and aggressive nature of these neoplasms, leading to the necessity of rapidly diagnosing and initiating treatment in these cases (21). BLs are a subtype of B-cell lymphomas, characterized by rapid growth; however, they are rarely a cause for secondary cardiac metastases, with limited cases documented to date and most of them having been identified in HIV-positive individuals (12,21). In this regard, a PubMed literature review was conducted to analyze adult cardiac BL cases published during the last 15 years (2010-2025). The search was performed using the keyword 'cardiac Burkitt lymphoma'. Inclusion criteria were study publication and diagnosis between 2010 and 2025, and patients diagnosed with primary or secondary cardiac BL with the intracardiac mass location mentioned and with documented age, sex and response to treatment. All cases of BLL or ambiguous line lymphomas were excluded, as well as patients aged <18 years (since the prevalence, incidence and etiology differ markedly compared with the adult population) and cases without a comprehensive documentation of the diagnostic or treatment response. Furthermore, 2 cases were excluded, as they were published in Chinese and could not be translated accurately. The 13 cases identified are summarized in Table I (21,22,28-38).

Out of these cases, there were no observed differences regarding sex (5 female patients and 8 male patients), with a median age of 55 years and an age range of 27-80 years. The main presenting symptom was dyspnea (7 cases), with palpitations being reported by ~1/3 of the patients, who were eventually diagnosed with cardiac BL (4 cases). There were 4 HIV-negative patients, 6 HIV-positive patients and 3 patients with their HIV status not available (no testing was mentioned), outlining the increased prevalence of cardiac BL in PLWHIV. All the cases featured RA involvement, with 5 cases having an isolated mass limited to the RA, without any extension to the other cardiac walls or chambers, confirming past reviews of literature that noted the RA as the main and preferred localization of cardiac BL (21,29,33-36). The majority of patients responded well to treatment, complete remission documented for 6 cases and partial remission for 5, although in 2 cases, the patients deceased shortly after therapy initiation. The chemotherapeutic regimens varied, with multiple regimens employed across 15 years of evolving oncological therapies. The main diagnostic method used was transthoracic echocardiography, which highlights the importance of echocardiographic evaluation and screening, even when a small level of suspicion is present. CT scans and MRI were also used as diagnostic or screening tools (particularly CT scans) or as methods of in-depth description of the nature and extension of the cardiac involvement (particularly MRI).

Considering the low prevalence and the aggressive nature of these cardiac tumors, as outlined in the literature review and in the aforementioned case presented, a high threshold of suspicion is necessary when diagnosing these types of cardiac metastasis and echocardiographic screening is key in these cases (24,39), particularly in PLWHIV, in whom the prevalence of cardiac BL is increased compared with the general population, irrespective of their $CD4^+$ cell count or immune status (13,21). Beyond standard transthoracic echocardiography, which is readily available and non-invasive, thus making it the method of choice when it comes to cardiac evaluation when an index of suspicion is present, CT scans, cardiac MRI or transesophageal echocardiography also have a diagnostic role when it is unclear whether cardiac involvement exists or when a more in-depth characterization is necessary (9,21). Often, cardiac BLs are localized solely at the RA level and, in the majority of cases, there is some RA involvement, even when other chambers and walls are occupied or infiltrated by tumoral masses, making the careful evaluation of right atrial morphology and function key when diagnosing these types of cardiac tumor (21).

Treatment with a hyper CVAD regimen is highly effective, particularly in BL with high-risk features, and is associated with a low incidence of CNS relapse (40), although cardiovascular risk stratification (clinical, biological, imaging modalities and risk scores, including the aforementioned HFA-ICOS score) and monitoring of the side effects related to anthracycline treatment (heart failure, left ventricular dysfunction and cardiomyopathy) is necessary in patients receiving this protocol (41).

In conclusion, hematological malignancies have numerous complications, which are either associated with treatment or with the disease, including, but not limited to, thrombosis, heart failure, cardiomyopathy, rhythm and rate disturbances. One notable complication is the potential of these malignancies to determine cardiac metastases, especially when in tumors of the lymphoid line. Out of these, NHL, particularly BL and DLBCL, even though rare in the general population, are much more frequent in PLWHIV and exhibit rapid and aggressive growth and subsequently, a markedly poor prognosis. Thus, patients with HIV infection, even those with a normal $CD4^+$ cell count, need a high index of suspicion for the diagnosis of these types of lymphoma (particularly BL and BLL) and secondary cardiac metastases, especially since rapid diagnosis and initiation of treatment improves the otherwise poor prognosis of these patients. However, patients still need to be monitored for adverse cardiovascular effects, considering that the use of multiple treatment regimens has demonstrated cardio-toxic effects and also considering the important chronic and acute cardiovascular complications of HIV infection, even though, to the best of our knowledge, at present, there is no clear standardized protocol regarding the evaluation and follow-up of this group of patients.

The literature review and the case presented in the present report underline the importance of a high index of suspicion, echocardiographic screening, rapid diagnosis and therapeutic intervention in patients with BL with secondary cardiac metastases and history of HIV infection, even in PLWHIV with a normal $CD4^+$ count, considering their low prevalence (even though higher than the prevalence in the general population) and aggressive nature, as outlined in the sequential echocardiographic evaluations in the present case report (the rapid growth of the tumor mass was observed in a short span, doubling in size over the course of ~3 weeks) and otherwise poor prognosis of this diagnosis in a occasionally neglected group of patients. The present case presentation also outlines, in conjunction with the literature review, the rapid and remarkable response to treatment (in this case with a hyper CVAD regimen) when a quick diagnosis is made and an adequate treatment regimen is employed. Additionally, the patient also particularly presented with no cardiac adverse effects to the treatment regimen administered, with an increase in left ventricular function and left ventricular longitudinal strain during treatment, even in the context of anthracycline treatment. However, considering the potential cardiotoxicity of the regimen and of the hematological diseases, periodic cardiologic follow-up is necessary in these patients, especially during anti-neoplastic treatment, to identify potential cardiovascular complications and address them as early as possible.

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Availability of data and materials

The data generated in the present study may be requested from the corresponding author.

Authors' contributions

AG participated in the design, drafting, reviewing and rewriting of the article throughout its development. IM participated in the drafting and reviewing of the manuscript, as well as in the acquisition and interpretation of the review data. AC contributed to the design and review of the article and provided insight into the main cardiovascular complications of hematological malignancies. MB further revised the article offering substantial contributions to its design and aided in the comprehensive description and presentation of HIV-associated cardiovascular complications. AM and CS were involved in patient care, acquisition of data and manuscript drafting, offering valuable insight into patient symptoms, signs and effects to treatment throughout monitoring. RR contributed by designing and reviewing the data and describing the patient's HIV status in chronological and logical order. ALG contributed to the conception of the article, revised the draft and performed further literature and article reviews, interpreting, adding and rearranging the data in a logical, detailed and coherent manner. AG and ALG confirm the authenticity of all the raw data. All authors read and approved the final version of the manuscript.

Ethics approval and consent to participate

Approval from the Medical Ethics Committee for Clinical Studies of the Coltea Clinical Hospital (Bucharest, Romania) was obtained prior to submission (approval no. 10/10.07.2025).

Patient consent for publication

Written informed consent for the publication of case data and images was obtained from the patient prior to publication.

Competing interests

The authors declare that they have no competing interests.

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