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Primary Left-Ventricular Tumor: Presenting as Congestive Heart Failure in an HIV-Positive Young Woman

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Abstract **Case Studies**

Background: Cardiac involvement is detected in up to two-thirds of people living with HIV on echocardiography, with pericardial effusion, diastolic dysfunction and dilated cardiomyopathy topping the list of structural lesions. In Nigeria, Cookey, reported cardiac abnormalities in 63 % of asymptomatic PLWHA, with pericardial effusion as the most prevalent (44.5 %). Although primary cardiac tumours are exceptionally rare (≈0.02 % at autopsy), malignant sarcomas may mimic heart-failure phenotypes, especially when diagnostic resources are limited.

Case Summary: A 29-year-old woman on tenofovir/lamivudine/efavirenz presented with jaundice 4weeks after commencement of antiretroviral which included effivarenz, progressive dyspnoea, orthopnoea, night sweats and pedal oedema. Examination revealed cachexia, raised jugular venous pressure and bibasal crackles; blood pressure was 100/60 mm Hg and heart rate 112 bpm. Point-of-care transthoracic echocardiography uncovered a lobulated, echogenic mass that appeared in direct continuity with the infero-media LV wall. Its echogenicity mirrored adjacent myocardium, it displayed no "echo-smoke," and it moved synchronously—not flail—with ventricular contraction. Marked regional hypokinesia of the involved wall reduced biplane LVEF to 35 %. The constellation made mural thrombus unlikely and raised strong concern for a primary cardiac sarcoma masquerading as congestive heart failure. Advanced imaging and biopsy were planned, but the patient absconded against medical advice before definitive work-up.

Conclusion: This vignette underscores the need for high clinical suspicion and immediate bedside imaging when we are caring for PLWHA in refractory heart failure. Recognition of tumor-like features—tissue-matched echogenicity, wall continuity and absence of spontaneous contrast—can steer clinicians away from empirical anticoagulation toward urgent cardio-oncology referral, even in resource-constrained settings.

Keywords: HIV, Cardiac Sarcoma, Congestive Heart Failure, Echocardiography, Cardiac Mass, Nigeria.

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1. INTRODUCTION:

The clinical focus of human immunodeficiency virus (HIV) infection has steadily shifted from opportunistic illnesses to chronic, non-infectious comorbidities. Contemporary meta-analyses show that **6 – 10 % of people living with HIV/AIDS (PLWHA) develop symptomatic heart failure,** nearly twice the rate reported in HIV-negative cohorts.[1] Overt failure, however, represents only the visible portion of a much larger problem. A Hospital based echocardiographic screening in HAART Naive HIV positive individuals attending clinic at the University of Port Harcourt Teaching Hospital, Port Harcourt, Nigeria, revealed structural or functional abnormalities in **63 % of asymptomatic Patients Living with HIV /AIDs (PLWHA)**, accounted majorly by pericardial effusion (44.5 %), isolated left-ventricular (LV) diastolic dysfunction (27.5 %) and dilated cardiomyopathy (3.5 %)[2]. Across sub-Saharan Africa, prospective series corroborate a **pericardial-effusion prevalence of 11 – 60 %** and consider **9 – 18 % of new-onset heart-failure cases to HIV-associated cardiomyopathy.** [3 4]

Primary cardiac tumours form a striking counterpoint. In the general population they appear in only **0.02 % of**

autopsies, and most are benign myxomas[5]. Immunodeficiency alters that presentation: necropsy studies in the antiretroviral era detect cardiac malignancy—predominantly Kaposi sarcoma or non-Hodgkin lymphoma—in **up to 28 % of PLWHA who die of AIDS-related complications[6]**. Even among clinically stable individuals the burden is greater than expected; Cookey and colleagues identified a discrete intracardiac mass in **0.5 % of screened PLWHA**, an incidence at least twenty-fold higher than autopsy-based estimates for the general population.[2]

Several mechanisms underlie this disparity**. Oncogenic co-infections (HHV-8, EBV), chronic cytokine activation and endothelial injury** foster tumorigenesis, while **long-term survival on highly active antiretroviral therapy (HAART)** allows time for non-infectious malignancies to declare themselves. [6] When a myocardium-dense mass remains in tissue continuity with the LV wall, regional hypokinesia ensues; the resulting systolic impairment presents **as conventional congestive heart failure.** Early recognition of tumor-specific echocardiographic signatures iso-echogenicity to myocardium, wall continuity, absence of spontaneous "echo-smoke" and non-flail motion—is thus essential for rapid cardio-oncology referral and potentially curative resection, particularly in resource-constrained settings.

2. CASE DESCRIPTION

In August 2024 a twenty-nine-year-old(29) Nigerian woman with well-documented HIV infection six months into tenofovir/lamivudine/efavirenz, presented with four weeks of relentless scleral icterus that began shortly after antiretroviral initiation. Progressive exertional dyspnea with orthopnea and paroxysmal nocturnal dyspnea, with productive cough of mucopurulent sputum, drenching night sweats, significant weight loss, episodic palpitations, atypical chest discomfort and rapidly enlarging pedal oedema. She denied hypertension, diabetes, alcohol use, previous cardiac disease or pregnancy.

On arrival she was cachectic, deeply jaundiced and tachypnoeic; oxygen saturation was 89 % on room air. Blood pressure ranged from 100/60 mm Hg to 122/78mm Hg, pulse was a regular 112 beats min^{-1}. Jugular venous pressure reached the mandibular angle; the apex beat was displaced and diffuse; bibasal fine crackles were audible. Heart sounds were dual and appeared distant. Abdominal examination disclosed a tender, smooth hepatomegaly of fourteen centimetres; there were no peripheral stigmata of chronic liver disease, deep-vein thrombosis or focal neurological deficit.

3. DIAGNOSTIC ASSESSMENT:

Routine biochemistry showed a cholestatic pattern alanine aminotransferase 28 IU L^{-1}, aspartate aminotransferase 26 IU L^{-1}. B-type natriuretic peptide was elevated; 1760 pg mL^{-1}. Creatinine measured 120 µmol L^{-1}; electrolytes were normal except for borderline hypokalemia (K^+ 3.5 mmol L^{-1}). CD4 count was 268 cells μL^{-1}; HIV-1 RNA 380 copies mL^{-1}. Full blood count revealed normocytic anemia (Hb 9.8 g dL^{-1}). Thyroid, coagulation and lipid panels were unremarkable.

Electrocardiography demonstrated sinus tachycardia with low-voltage QRS complexes and non-specific ST-T flattening. A postero-anterior chest radiograph showed cardiomegaly, pulmonary venous diversion and a moderate left pleural effusion with mid-zone consolidation.

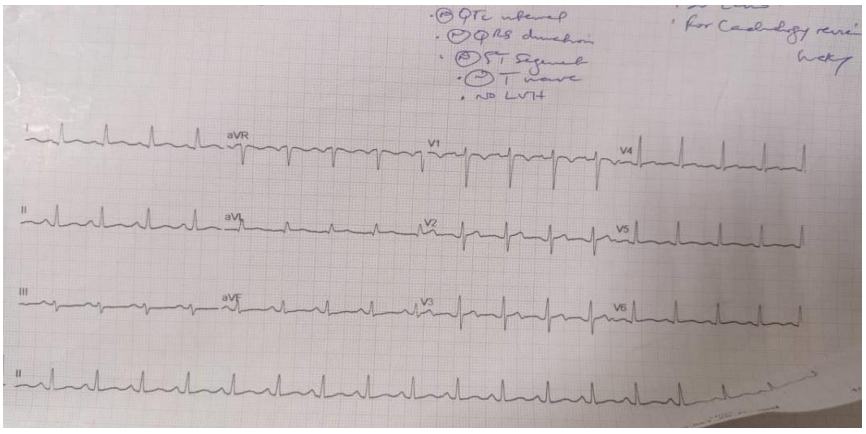

Figure 1ECG of patient showing Sinus Tachycardia

Point-of-care transthoracic echocardiography proved pivotal. Para sternal short-axis and apical four-chamber views revealed a solid mass (≈ 4..82 × 3.26 cm) in direct, unbroken continuity with the inferomedial left-ventricular

wall. There was a smaller well circumscribed mass at the base of the left ventricular wall measuring 0.5cm in diameter. The mass shared myocardial echogenicity, lacked a capsule, displayed no spontaneous echo-contrast ("smoke") and moved synchronously with systole rather than flailing. Adjoining segments were markedly hypokinetic, reducing biplane LVEF to 35 %. Right-sided chambers, valves and pericardium were otherwise normal save for trivial pericardial effusion and mild functional mitral regurgitation.

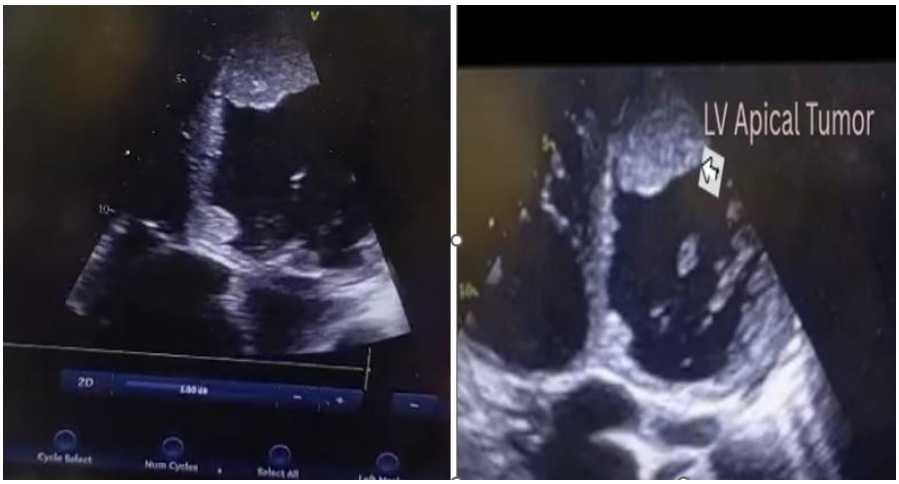

Figure 2A. Apical 4 chamber view showing Cardiac Tumors .
Figure 2B: Zoomed in Apical 4 chamber view

4. ASSESSMENT AND EARLY MANAGEMENT AND COURSE

The iso-echogenic, wall-contiguous lesion without intracavitary smoke or independent mobility rendered mural thrombus unlikely and favored an infiltrative primary malignancy—most plausibly an undifferentiated sarcoma. Infective vegetations and tuberculous granulomas were considered but deemed improbable in the absence of fever, leukocytosis or valvular destruction. Empirical anticoagulation was withheld to avoid intratumorally hemorrhage.

Cardiac magnetic-resonance imaging and surgical biopsy were scheduled off-site for definitive tissue characterization and staging. Guideline-directed quadruple heart-failure therapy commenced: intravenous furosemide, sacubitril/valsartan 24/26 mg, bisoprolol 1.25 mg, spironolactone 25 mg and dapagliflozin 10 mg daily. Selenium and thiamine were supplemented, and highly active antiretroviral therapy was continued excluding efavirenz.

Despite intensive counselling, the patient left hospital against medical advice on day 15, citing financial hardship and family obligations. Community health workers are attempting re-engagement, recognizing that early surgical excision—sometimes requiring cardiac auto transplantation offers the only realistic prospect of long-term survival for primary cardiac sarcoma.

DISCUSSION

Regional echocardiographic surveys from sub-Saharan Africa testify to a disproportionate burden of silent structural heart disease among people living with HIV/AIDS (PLWHA). In Port Harcourt, Cookey et al. identified abnormalities in 63 % of asymptomatic adults, with pericardial effusion (44.5 %) and isolated left-ventricular (LV) diastolic dysfunction (27.5 %) heading the list.[2] Although primary cardiac tumors surface in barely 0.02 % of autopsies worldwide,[5] immune dysregulation, oncogenic co-infections and chronic inflammation appear to magnify the risk in PLWHA; the same Port Harcourt cohort detected an intracardiac mass in 0.5 % of those screened roughly twenty-five times the population baseline.[2]

Our patient exemplifies this malignant outlier. The mass was iso-echogenic to surrounding myocardium, shared an unbroken tissue plane with the inferolateral LV wall, lacked spontaneous echo-contrast and exhibited synchronous—not flail—motion. In addition, a neatly circumscribed satellite nodule measuring roughly 0.4 cm was seen medially at the base of the left ventricle. The presence of this daughter lesion, together with the pronounced regional hypokinesia, makes mural thrombus highly improbable and instead strongly favors a malignant metastasis, [7-9] the ensuing systolic impairment produced the heart-failure phenotype that initially masked the tumor. These echocardiographic hallmarks—wall continuity, tissue-matched grayscale, absence of a hypoechoic "cap" and regional hypokinesia—have been validated as strong

discriminators of primary cardiac sarcoma in contemporary imaging-pathology series.[9]

In people living with HIV/AIDS (PLWHA), two further etiologies merit consideration: cardiac lymphoma and HIV-associated thrombus. Visceral non-Hodgkin lymphoma may infiltrate the myocardium, yet it most often involves the right heart or pericardium and tends to appear heterogeneous on echo and hyper-metabolic on positron-emission tomography.[10] Conversely, LV thrombus can arise in HIV-related cardiomyopathy or hyper-coagulable states, but it typically presents as echolucent, sessile layering along akinetic segments and may generate low-velocity spontaneous contrast—features absent in this case.[11] Collectively, the imaging signature, anatomical location and satellite seeding render primary malignant sarcoma the leading diagnosis, with lymphoma a distant second and thrombus improbable.

Current cardio-oncology guidance from the European Society of Cardiology recommends a rapid, multimodality work-up whenever a ventricular mass displays malignant traits.[12] Gadolinium-enhanced cardiac magnetic resonance (CMR) delineates tissue character; malignant tumours show early perfusion and heterogeneous late enhancement, whereas thrombi remain avascular and dark on all post-contrast sequences.[13] Computed-tomography angiography refines resection planning by mapping extracardiac extension, while ^{12}F-FDG PET scans screen for occult metastasis or lymphoma. Once malignancy is confirmed, complete surgical excision—often via auto-transplantation—offers the only realistic hope of durable survival; five-year survival exceeds 40 % after R0 resection but falls below 10 % when surgery is incomplete or delayed.[14] In resource-limited settings, timely access to CMR, PET and subspecialty surgery is challenging, underscoring the need for strengthened referral networks and financial protection mechanisms to prevent outcome-limiting treatment abandonment, as occurred in this patient.

Scleral icterus in this context is readily explained by a double hit: passive hepatic congestion from right-sided heart failure and potential non-nucleoside reverse-transcriptase inhibitor (NNRTI) hepatotoxicity. Efavirenz, the NNRTI in her regimen, has been linked to transaminase elevation and clinical jaundice in up to 6 % of recipients, particularly within the first twelve weeks of therapy.[15] Vigilant biochemical surveillance is therefore warranted whenever unexplained cholestasis complicates HIV-related cardiovascular illness.

Management must straddle two evidence streams. For heart failure with reduced ejection fraction, the 2022 AHA/ACC/HFSA guideline endorses simultaneous initiation of quadruple pharmacotherapy—angiotensin receptor-neprilysin inhibition, beta-blockade, mineralocorticoid receptor antagonism and an SGLT-2 inhibitor to improve survival and manage ventricular reverse-remodeling.[16] This regimen was commenced alongside intravenous loop diuretics. Antiretroviral therapy was suspended initially following the suspicion of drug reactions. Effivarenz was substituted with dolutegravir at recommencement of antiretroviral therapy. For the suspected sarcoma, the 2022 ESC cardio-oncology[12] guideline recommends urgent referral to a dedicated multidisciplinary team; complete surgical excision, often with cardiac auto-transplantation and adjuvant chemotherapy or radiation, remains the cornerstone of potentially curative treatment.[23] Outcomes hinge on timing: five-year survival exceeds 40 % after R0 resection but plummets below 10 % when intervention is delayed or incomplete. [17]

Unfortunately, financial constraints prompted our patient's self-discharge before definitive imaging or surgery. Her prognosis is therefore guarded. The patient's outlook is compromised not only by the biological aggressiveness of a presumed cardiac sarcoma but also by formidable health-system barriers typical of resource-constrained settings such as Nigeria. Financial hardship forces most patients to pre-pay for every investigation, drug and procedure; even a short admission rapidly exhausts disposable income, as happened here. Human resources are another limiting factor: cardio-oncology teams and perfusionists with experience in tumor resection or auto transplantation are concentrated in a handful of tertiary centers, and their availability cannot always match clinical urgency.[18-27] Infrastructure gaps compound these challenges. Intermittent mains electricity and scarce back-up power jeopardize prolonged cardiopulmonary bypass; manual (hand-cranked) heart–lung pumps are occasionally deployed as a stop-gap, but they add logistical complexity and operator fatigue during surgery.

Out of pocket financing for healthcare services further curtails definitive care. Nigeria's National Health Insurance coverage remains patchy, so most high-cost cardiovascular interventions rely on out-of-pocket payment or humanitarian funding. [28-30]. The lancet reported that 77% of healthcare funding in Nigeria is out of pocket. [30]

Although renal transplantation is gaining traction [31-35] with several centers performing dozens of procedures annually—cardiac transplantation has yet to progress beyond policy drafts. Key obstacles include the absence of an enforceable deceased-donor program, limited organ-sharing infrastructure and an underdeveloped legal framework for brain-death certification. Consequently, heart-lung transplantation—or even isolated heart transplantation as a salvage option after sarcoma excision remains aspirational.

Overcoming this confluence of constraints will require multisectoral investment: expanded health-insurance pools, public–private partnerships to finance equipment and back-up power, regional training in perfusion technology, and the gradual establishment of a regulated national transplant registry. Until such structural reforms take root, the prognosis for patients like ours whose curative window hinges on timely, resource-intensive surgery will unfortunately remain guarded.

The case accentuates three system-level imperatives for resource-limited settings: (i) broaden point-of-care echocardiography capacity to detect tumour-like signatures early; (ii) establish subsidised pathways for advanced cardiac imaging and oncological surgery; and (iii) secure social-support mechanisms to prevent treatment abandonment. Only by marrying rapid bedside recognition with accessible specialist care can we hope to convert a lethal masquerader into a survivable diagnosis.

6. CONCLUSION

When a young, immunocompromised patient presents with new-onset heart failure, clinicians must think beyond traditional viral cardiomyopathy. An iso-echogenic, wall-contiguous mass without intracavitary smoke should trigger suspicion of primary cardiac sarcoma and prompt urgent oncological evaluation. Strengthening diagnostic infrastructure and financial-support mechanisms is indispensable if such elusive but lethal entities are to be intercepted in time.

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