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Case Study

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A CASE REPORT OF NEWLY DIAGNOSED HIV PRESENTING WITH DILATED CARDIOMYOPATHY AND ISCHAEMIC STROKE AT FEDERAL MEDICAL CENTRE, OWO

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ABSTRACT

Background: HIV as a cardiovascular risk factor has undergone back and forward debates. **Objectives:** To emphasise that HIV is a cardiovascular risk factor and an ounce of prevention is key. **Case report:** A 34 years old lady presented with 3months history of cough, recurrent fever and exertional dyspnoea, 2 weeks history of passage of frequent loose stool and sudden onset of weakness of left upper and lower limbs a day prior to presentation. Genotype AS, not a known hypertensive or diabetes and she neither smoke nor take alcohol or any recreational drug. Examination revealed a young lady, conscious, wasted, afebrile, no finger clubbing but has bilateral pitting pedal oedema. Neurological findings includes left facioparesis UMN, initial hypotonia and hyporeflexia with power 0 in the left upper and lower limbs with extensor planter response but became hyperreflexia with progressive improvement in the power and tone. Cardiovascular examination revealed tachycardia 108/m, BP 120/80mmHg, elevated JVP, apex beat at 6LICS AAL with s1 s2 s3. There was tachpnoea, bibasal coarse crackles and mild hepatomegaly. Diagnosis of HIV confirmed by polymerase chain reaction (PCR). Brain computerized tomography (CT) scan revealed a hypodense lesion at the right middle cerebral artery territory and echocardiography revealed dilated cardiac chambers with global hypokinesia, left ventricular ejection fraction (EF) 35%, biventricular diastolic dysfunction and moderate mitral and tricuspid regurgitation. She made significant recovery of neurological deficit and achieve good exercise tolerance after 3 months of HAART. **Conclusion:** A newly diagnosed case of HIV in a young lady with no other identifiable cardiovascular risk factor, presenting with dilated cardiomyopathy and ischaemic stroke is remarkable.

INTRODUCTION

A 34 years old lady presented via the emergency unit on referral from Unimed, Akure, on account of cough and recurrent fever, exertional dyspnoea of 3months duration, and passage of frequent loose stool of 2weeks duration and a sudden onset of inability to move the left upper and lower limbs a day prior to presentation. Cough was said to be dry, with associated weight loss, night sweat and chest pain. Fever was recurrent, associated with catarrh and often relieved with paracetamol and antimalarial. Patient has been experiencing symptoms of easy fatigualibilty, dyspnea on exertion for about 3months which progress to dyspnea at rest, orthopnea, paroxysmal nocturnal dyspnoea. There was associated bilateral pedal swelling. There was history of passage of loose stool of 2weeks duration. Stool was non-bloody, non-mucoid, no history of patronizing food vendors. There was history of vomiting of about 3 episodes before presentation containing recently ingested meal, projectile, non-bilous, non-bloody. A day prior to presentation, patient developed sudden weakness of both left upper and lower limbs. There was no associated slurred speech, loss of consciousness, neck pain, neck stiffness, photophobia, convulsion or preceding history of headache. She is not a known hypertensive, diabetes or asthmatic. Genotype is AS. Patient had one pint of blood transfused 1years ago at delivery of her 2nd child. Patient was reactive to HbsAg and Retroviral screening done at referral centre. This was repeated and subsequently confirmed by PCR. She is a single mother with 2 children. Husband abandon her following her illness, took away the 2nd child (male). She does not smoke cigarette or tobacco or drink alcohol or use any recreational drugs.

On examination she was conscious and not in any painful nor respiratory distress. Chronically ill, wasted, not pale, anicteric, afebrile, acyanosed, no finger clubbing, not dehydrated but has bilateral pitting pedal edema. Glasgow coma scale was 13/15 (E.O-4, BVR-4, BMR-5). Kenign's or brudinzki's signs were negative. Pupils were 3mm dilated and reactive to both direct and concentual light. Left Cranial nerves 7th palsy UMN. Muscle bulk was reduced globally but no fasciculation,

Tone were normal on both right upper and lower limbs but hypotonia in both left upper and lower limbs. Power were 4+ in both right upper and lower limbs but 0 in both left upper and lower limbs with extensor planter response. Cardiovascular examination revealed pulse rate108bpm small volume and regular. Blood pressure 120/80mmHg supine. Jugular venous pulse (JVP) was elevated. Apex beat 6LICS AAL diffuse. Heart sounds- S1, S2, S3 heard. Chest findings includes respiratory rate 30cpm. SpO2- 96% in room air, Trachea was central. Chest was symmetrical with equal chest expansion. Vesicular breath sounds but there were bibasal coarse crackles. Abdominal examination revealed no area of tenderness. Liver span was 14cm firm to touch and smooth surface and no splenomegaly. Kidneys were not ballotable, ascites not demonstrable and bowel sounds normoactive.

Brain CT scan revealed a hypodense lesion at the right middle cerebral artery territory involving the posterior limb of the right internal capsule, basal ganglia and thalamus with evidence of cerebral oedema compressing the anterior horn of the lateral ventricle. Echocardiography revealed dilated cardiac chambers with exception of the left ventricle and preservation of the left posterior wall, global hypokinesia, left ventricular EF 35% (systolic dysfunction), biventricular diastolic dysfunction and moderate mitral and tricuspid regurgitation and mild pericardial effusion. Patient was admitted in the medical ward in cardiac position and commenced antifailure regimen, digoxin, parenteral frusemide, aldactone and uperio, Antibiotic, anticoagulant, and HAART were commenced. Patient was comanaged by neurology and physiotherapy unit. Patient was discharged home after 2weeks of admission due to financial constraint, she was still unable to walk or sit. She was in NYHA class 2 Congestive cardiac failure secondary to dilated cardiomyopathy. She continued medications and physiotherapy as outpatient. Patient achieved remarkable recovery of neurological deficit and exercise tolerance after 3months HAART. She is able to walking unaided but the left hand function is still impaired.

INVESTIGATION RESULTS

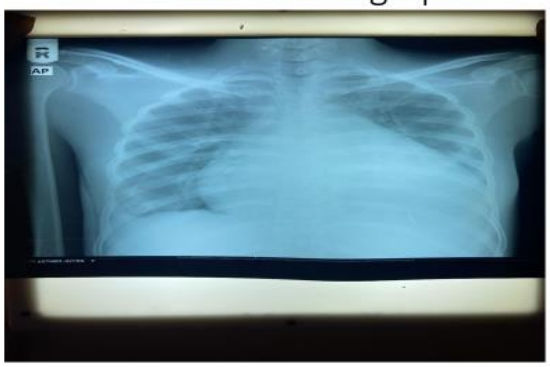
DATE	INVESTIGATION	RESULTS
25/02/24	Urinalysis	Blood- positive (+) Urobilinogen- Normal Bilirubin- Negative Protein- Positive (+) Nitrite- Negative Ketones- Negative Glucose- Negative pH- 5.0
26/02/24	E/U/Cr	Na-124mmol/l HCO3- 27mmol/l K- 2.7mmol/l Cl-102mmol/l Creatinine- 73umol/l Urea- 1.2mmol/l
	Serology	Reactive to HBsAg and RVS Non-reactive to anti-HCV
	CXR	Cardiomegaly with globular cardiac shadow and upper lobe diversion

PREVIOUS INVESTIGATION RESULTS FROM UNIMED

DATE	INVESTIGATION	RESULTS
24/02/24	Serology	Reactive to RVS and HBV Non-reactive to anti-HCV
24/02/24	E/U/Cr	Na- 144mmol/l K- 3.1mmol/l HCO_3- 25mmol/l Cl- 106mmol/l Creatinine- 130umol/l Urea- 3.6mmol/l
25/02/24	FBC	PCV- 40% WBC- 5390cmm Neutrophils- 66.6% Lymphocytes- 23.0% Monocytes- 8.7% Eosinophils-0.2% Basophils- 1.5% PLATELETS- 168,000cmm

26/02/24	Fasting Lipid Profile	Total Cholesterol 4.8mmol/l
		Triglycerides 1.3mmo/l
		LDL- cholesterol 2.9mmo/l
		HDL- cholesterol 0.7mmol/l

Chest Radiograph



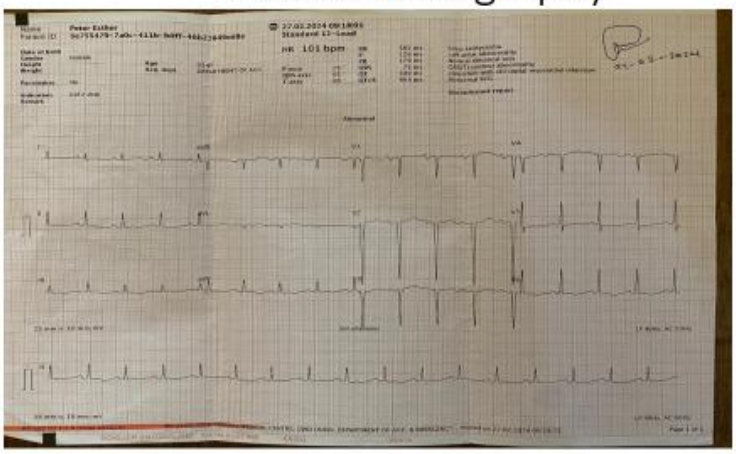
INVESTIGATION RESULTS

DATE	INVESTIGATION	RESULTS
26/02/24	LFT	SGOT- 12IU/l SGPT- 5IU/l ALP- 21IU/l Total protein- 62g/l Albumin-26g/l Globulin- 36g/l Total bilirubin- 26.6umol/l Conjugated bilirubin- 22.7umol/l
27/02/24	FBS	6.0mmol/l
	2HPP	6.9mmol/l
27/02/24	ECG	Sinus tachycardia, HR 101/m, Left atrial enlargement with normal axis deviation.

INVESTIGATION RESULTS

DATE	INVESTIGATION	RESULTS
28/02/24	FBC+ESR+PLATELETS	PCV- 40% WBC- 5,100cmm Platelet- 139,000cmm ESR 11mm/hr Differential count include: N-47% L-48% E-5% RBC- Target cells (+)
29/02/24	Hb Genotype	AS

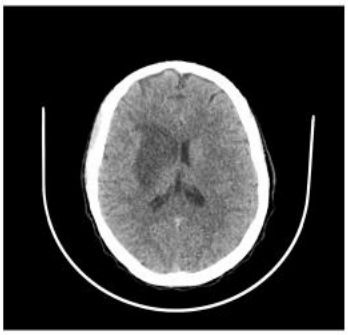
Electrocardiography



INVESTIGATION RESULTS

DATE	INVESTIGATION	RESULTS
29/02/24	Cranial Computed tomography	The scanogram shows engorgement of both inferior nasal turbinates. Images reveal a non-enhancing hypodense area (HU=19) in right parietal lobe involving the ipsilateral basal ganglia, thalamus and posterior horn of the internal capsule. There is associated mass effect on the right as evidenced by the compression of the ipsilateral anterior and body of the lateral ventricle and loss of normal grey-white matter differentiation. There is no associated shift of the midline brain structures. The brainsterm, cerebellar hemispheres and cerebello-pontine angles are preserved No intracranial mass or collection is seen The basal cisterns are within normal limits The left thalamus, pituitary gland, visualized orbits and its contents, para-nasal sinuses and the mastoid air cells are all preserved. There is engorgement of both inferior nasal turbinates in keeping with chronic rhinits. The calvarium, facial bones and the surrounding soft tissues are preserved. CONCLUSION; Acute cerebral infarct in the right MCA territory (involving the posterior limb of the right internal capsule, basal ganglia and thalamus).

Pre contrast Axial CT scan showing an hypodense lesion at the right Middle Cerebral Artery territory compressing on the anterior horn of the right lateral ventricle.

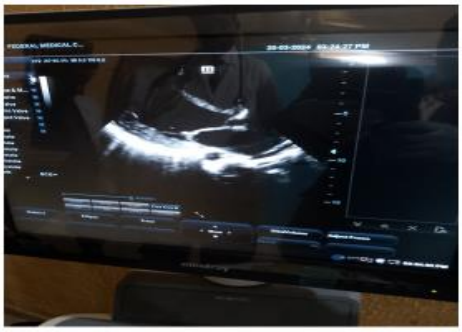


INVESTIGATION RESULTS

DATE	INVESTIGATION	RESULTS
06/03/24	ECHO	Reveals the following 1. Systolic dysfunction 2. Biventricular diastolic dysfunction 3. Dilated cardiac chambers with exemption of left ventricle and preservation of left posterior wall 4. Global hypokinesia 5. Moderate mitral and tricuspid regurgitation 6. Mild pericardial effusion
08/03/24	Repeat E/U/Cr	Na 135 mmol/l K 4.2 mmol/l Cl 102 mmol/l Hco3 28 mmol/l

Echocardiography

Parasternal Long Axis View



DISCUSSION

Human immunodeficiency virus (HIV) is the causative agent of HIV/AIDS (Acquired Immunodeficiency Syndrome). It is a RNA virus belonging to the family Lentiviridae. It is a disease of public health concern and occurs in endemic proportions in some regions.[1] HIV attacks the immune system by destroying CD4 positive (CD4+) T cells and leaves people living with HIV vulnerable to opportunistic infections, diseases and other complications. HIV is a global concern, 39.0 million people were living with HIV at the end of 2022. HIV as a cardiovascular risk factor has undergone back and forward debates.[2] Cardiac involvement in HIV infection has been described by several autopsy and echocardiography – based studies.[3] Prevalence is much more than is clinically apparent. Persons with HIV infection have up to 2 times risk of cardiovascular disease (CVD) compared to those without HIV infection.

HIV – infected patients live longer as a result of HAART resulting in increasing prevalence of CVD.[4] Causes includes direct effects of HIV infection and indirectly due to opportunistic infections, or its treatment as well as other established causes of cardiac disease in non HIV infected populations.[3]

Spectrum of Cardiovascular diseases in HIV Infection includes pericarditis, myocarditis / cardiomyopathy, pulmonary vascular disease and pulmonary hypertension, endocardial disease, cardiac tumour, coronary artery Disease.[3] Pericarditis is the most common finding in patients with HIV infection. Autopsy series have reported up to 37% prevalence. Echocardiographic series is as high as 59%. Spectrum ranges from asymptomatic effusions to potentially fatal tamponade. Most asymptomatic pericardial effusions do not have identifiable etiology. However etiology can be established in 2/3 symptomatic pericardial effusions. Causes of HIV-related pericardial diseases are wide and includes infections and malignancy. Infections includes; Mycobacterial organisms – M. tuberculosis and atypical M. avium intercellulare complex in 34%, Bacterial - Staphylococcus aureus, Streptoccus pneumoniae, Chlamydia spp, Listeria monocytogenes, Nocardia asteroids, Viral – herpes smplex, CMV and Coxsackie virus, Fungal – Histoplasma capsulatum and Cryptococcus neoformans. Neoplastic – Kaposi's sarcoma and lymphoma. Treatment includes; conservative treatment and follow up with serial echocardiography for asymptomatic effusion. Large effusions require pericardiocentesis and fluid analysis for cytology, culture and biochemical studies. Pericardial biopsy increase diagnostic yield especially tuberculosis and pericardiotomy is needed for refractory effusion. Antibiotic, antituberculous, antifungal, antiviral, chemotherapy and radiotherapy depending on the etiology. Our patient had mild pericardial effusion which resolved completely with 2weeks course of antibiotics.

Myocarditis and cardiomyopathy: Autopsy reported 52% myocarditis among HIV – infected patient. Echocardiographic series identified cardiomyopathy in 30 – 40%.[3,5,6] Histologic diagnosis of myocarditis in 83% of patients with dilated cardiomyopathy. Direct effect of HIV virus as cause of myocarditis has been established by detection of HIV in cardiac tissue by culture, molecular techniques and immunohistochemistry. Although, Lack of CD4 cell receptor in myocardial cells further argues against the direct role of HIV. HIV may indirectly cause myocarditis via release of cytokines (IL- 1beta, IL= 6, IL- 9, endothelin-1, TNFalpha), hypersensitivity from uncontrolled hypergammaglobulinemia and autoimmunity. Also opportunistic infections; Toxoplasmosis, M. tubrerculosis, M avium intercellulare complex, Cryptococcus neoformas, Aspergillous fumigatum, Candidas albicans, Histoplasma capsulatum Coccidioides iminitis, CMV, Coxsackie virus, Herpes simplex and Trypanosoma cruzi (Chagas disease). Nutritional deficiency; Selenium, L carnitine and vitamin B. Drug Abuse – Alcohol, Cocaine and methamphetamine. Medications; Interferon, Doxorubicin, Amphotericin B and Zidovudine have been implicated. Diagnosis of myocarditis and dilated cardiomyopathy is establishes through the following; Chest X Ray may show cardiomegaly but ECG findings are non-specific. Echocardiography is diagnostic which revealed dilated cardiac chambers, although left ventricular cavity size may be normal in early myocarditis. Diffuse left ventricular hypokinesia, reduced left ventricular ejection fraction (cardiac dysfunction), bi ventricular diastolic dysfunction.[3,5,6] Our patient CXR revealed gross cardiomegaly of biventricular configuration and upper lobe diversion. ECG was unspecific and showed sinus tachycardia HR 101/m, left atrial enlargement and normal axis. Echocardiography revealed systolic dysfunction (EF 35%), biventricular diastolic dysfunction, dilated cardiac chambers, global hypokinesia, moderate mitral and tricuspid regurgitations and mild pericardial effusion.

Cardiac Magnetic Resonance (CMR) is increasingly becoming an important tool for the diagnosis of myocarditis but associated with claustrophobia, tachyarrhythmias and hemodynamic instability and contraindicated in patients with implantable devices.[3] Serial CMR usingT1-weighted images with gadolinium is useful to visualize myocardial injury and track the progression of myocarditis. Myocardial Delay Enhancement (MDE) provides improved sensitivity and useful for guided endomyocardial biopsies.[3] Gold standard for the diagnosis of acute myocarditis is transvenous right ventricular endomyocardial biopsy (RVEMB) and histopathologic evaluation of biopsy. Main drawback of RVEMB is sampling error. However, absence of histologic evidence do not preclude the diagnosis of myocarditis. PCR of viral RNA and DNA for viral myocarditis is diagnostic. Ziehl Nelsen stain for mycobacterial disease. Immunoperoxidase-based stains for HLA-ABC and HLA-DR antigens is highly sensitive 80% and specific 85% in a case of autoimmune cause. Specific forms of Myocarditis such as lymphocytic myocarditis, Giant-cell myocarditis, cardiac sarcoidosis, hypersensitivity and eosinophilic myocarditis, Lyme myocarditis, Chagas cardiomyopathy and HIV myocarditis can be established.[3] We were constraint by the lack of these facilities.

Dilated Cardiomyopathy (DCM) is a significant health concern worldwide, affecting millions of people. Myocarditis often than not results to dilated cardiomyopathy with resultant congestive heart failure as in our case. Prognosis of HIV-associated myocarditis is significantly poorer than that of lymphocytic myocarditis and it is the strongest predictor of death.[3,7] DCM is a leading cause of heart failure, sudden cardiac death, and heart transplantations. According to the World Health Organization (WHO), cardiovascular diseases, including cardiomyopathy, account for a substantial proportion of

global morbidity and mortality. The impact of cardiomyopathy extends beyond physical health, affecting individuals, families, and healthcare systems. A day prior to presentation, our patient developed stroke confirmed by brain CT as ischaemic stroke. HIV infection can impact CVD and co-morbidities known to increase CVD risk. Untreated HIV can cause proatherogenic elevations in serum lipids. Chronic HIV viremia results in increases in systemic inflammation, hypercoagulation and reductions in endovascular reactivity, all of which are at least partially in addition to dilated cardiomyopathy may be responsible for our patient cerebrovascular accident/ stroke. Our patient is a young woman, newly diagnosed HIV not on any medication and no identifiable traditional CV risk factors such as smoking, hyperlipidaemia /dyslipidaemia, hypertension, diabetes, obesity, drugs abuse and others and her CD 4+ count was greater than 200. Our patient presented with typical pre-ART phenotype of HIV-associated cardiomyopathy with overt left ventricular systolic dysfunction.[7]

Other Cardiovascular Diseases in HIV includes; Pulmonary Vascular Disease and Pulmonary hypertension- 10%, Endocardial Disease are uncommon and occurs exclusively in IV drug users, Cardiac Tumours- 1. Kaposi sarcoma involves myocardium and pericardium and typically present as pericardial effusion, Primary cardiac lymphoma is rare. Coronary artery Disease occurs with HAART (Protease inhibitors) and longer survival resulting into dyslipidaemias, insulin resistance, hyperlipidaemia, lipodystrophy and lipoatrophy.[3] These are less common and are not documented in our patient. We are glad to report the dramatic response of our patient to HAART with resolution of mild pericardial effusion within 2 weeks and significant improvement of neurological deficit within the first 3 months of treatment. There is no doubt that early diagnosis and prompt treatment will improve the prognosis of HIV associated cardiovascular diseases. There must be equally a concerted effort to embark on various preventive strategies ranging from primordial, primary, secondary and tertiary prevention[8]. Primordial prevention involves sensitization and awareness campaigns in various public places, social and mass media. Primary prevention entails regular counselling, voluntary screening, abstinence or safe sex practices, safe blood transfusion, and universal safety protection for health workers, pre and post exposure prophylaxis and prevention of maternal child transmission. Secondary prevention also includes regular adherence counselling of HAART patients, regular CD4 count and viral load monitoring, risk modification and treatment of comorbidities and TB/HIV integrated service delivery. Finally the tertiary preventive strategies includes regular psychological assessment to improve the quality of patient life, social & occupational rehabilitations will no doubt improve patient outcome and significant reduce the prevalence, prognosis and overall burden of cardiovascular disease associated with HIV/AIDs.

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