We are IntechOpen, the world's leading publisher of Open Access books Built by scientists, for scientists

4,800
Open access books available

122,000
International authors and editors

135M
Downloads

Our authors are among the

154
Countries delivered to

TOP 1%
most cited scientists

12.2%
Contributors from top 500 universities

CLARIVATE ANALYTICS BOOK CITATION INDEX INDEXED

WEB OF SCIENCE™

Selection of our books indexed in the Book Citation Index in Web of Science™ Core Collection (BKCI)

Interested in publishing with us?
Contact book.department@intechopen.com

Numbers displayed above are based on latest data collected.
For more information visit www.intechopen.com

Chapter 16

Cardiomyopathies in Sub-Saharan Africa: Hypertensive Heart Disease (Cardiomyopathy), Peripartum Cardiomyopathy and HIV-Associated Cardiomyopathy

Okechukwu S. Ogah and Ayodele O. Falase

Additional information is available at the end of the chapter

http://dx.doi.org/10.5772/67023

Abstract

Cardiomyopathy is an important cause of cardiac-related morbidity and mortality in sub-Saharan Africa. Dilated cardiomyopathy is responsible for 20–30% of adult heart failure (HF) in the region. It is only second to hypertensive heart disease as etiological risk factor for HF in many parts of the continent. The aim of the chapter is to review the current epidemiology, clinical features, management, and prognosis of hypertensive heart disease, peripartum cardiomyopathy, and HIV-associated cardiomyopathy in sub-Saharan Africa.

Keywords: cardiomyopathy, heart muscle disease, hypertensive heart disease, peripartum cardiomyopathy, HIV-associated cardiomyopathy, heart failure

1. Introduction

Cardiomyopathies are common in Africa. Common causes of myocardial diseases in the region are hypertensive heart disease, endemic cardiomyopathies such as dilated cardiomyopathy, endomyocardial fibrosis, and peripartum cardiomyopathy and most recently heart diseases due to HIV/AIDS and ischemic cardiomyopathy. They are often associated with high morbidity and mortality due to late presentation, lack of modern day treatment available in high-income countries, as well poverty, which limits access to healthcare. **Figure 1** shows the common causes of heart failure (HF) in sub-Saharan Africa (SSA) based on a recent survey of acute HF in the region [1]. The chapter deals with hypertensive heart disease (hypertensive cardiomyopathy) peripartum cardiomyopathy and HIV-associated cardiomyopathy.

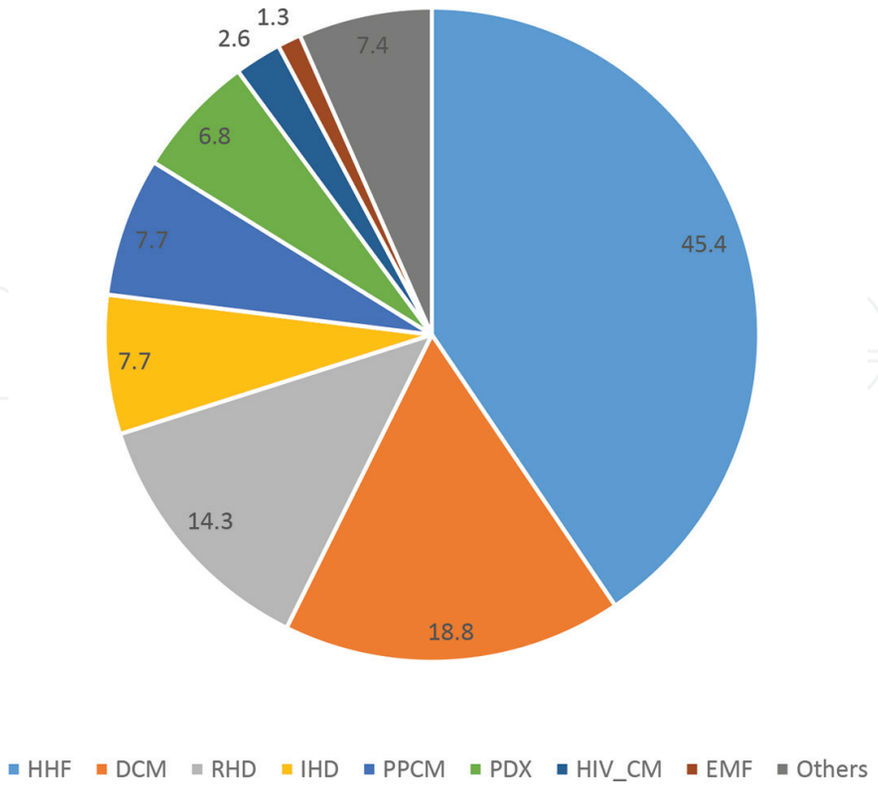

Figure 1. Etiological risk factors for heart failure in sub-Saharan Africa. (Adapted from Damasceno et al. HHF = Hypertensive heart failure, DCM = Dilated cardiomyopathy, RHD = Rheumatic heart disease, IHD = Ischemic heart disease, PPCM = Peripartum cardiomyopathy, PDX = Pericardial diseases, HIV_CM = HIV associated cardiomyopathy, EMF = Endomyocardial fibrosis.

2. Hypertensive heart disease (hypertensive cardiomyopathy)

2.1. Epidemiology

More than 30% of adults in SSA have hypertension. The prevalence rate is among the world highest. Worse still, the region has some of the world's lowest rates of hypertension awareness, treatment, and control. About 66% of the people are not aware, 82% are not treated and 93% are uncontrolled. In the year 2010, the age standardized prevalence of hypertension in adults aged 20-years and above was estimated as 36.9 and 36.3% for men and women, respectively (this is compared to 20.9 and 20.3%, 10 years earlier). This translates to 64.8 and 63.8 million men and women with elevated blood pressure in 2010 (compared to 25.5 and 24.9 million hypertensive men and women in the year 2000).

In a recent systematic analysis of population-based studies of hypertension in SSA, the pooled prevalence in the region rose from 19.7% in 1990 to 30.8% in 2010. It is estimated that there were about 54.6 million people with hypertension in Africa in 1990. This rose to 130.2 million cases in 2010. It is also projected that by the year 2030, there will be about 216.8 million cases of hypertension in the region [2].

http://dx.doi.org/10.5772/67023

Hypertension is the commonest and strongest risk factor for cardiovascular disease (CVD) in SSA [3]. It is also the commonest cause of disability and death from non-communicable diseases (NCDs) in the region [4]. The condition often manifests in young and middle aged adults in their productive years [4]. It is estimated to cause over 500,000 adult deaths annually and about 10-million years of life lost. Over 50% of heart disease and HF in the region is attributed to elevated blood pressure.

According to the African Union, hypertension is one of the greatest health challenges in adults in Africa after HIV/AIDS [4]. Recent data indicate that hypertension is rising in SSA at a faster rate compared to other regions of the world [5, 6]. This has been attributed to that adoption of western lifestyle, diet and culture, urbanization, urban migration from rural areas, ageing of the population, and increasing use of cigarettes and alcohol [3, 5–7].

It has been demonstrated that a chronic hyperadrenergic state is common among African hypertensives and may be responsible for the high prevalence of hypertension observed in Africans [8].

2.2. Clinical features

Heart disease secondary to elevated blood pressure (hypertensive heart disease), which may manifest in the following ways:

2.2.1. LV diastolic dysfunction

LV diastolic dysfunction is common in hypertensive subjects in the region [9–12]. About 62% of hypertensive individuals have various degrees of LV diastolic dysfunction compared to 12% of normal subjects [9, 13]. Diastolic dysfunction is worse in those with concentric LV geometry [9] as well as in individuals at risk of obstructive sleep apnea [14]. Diastolic dysfunction also occurs in offspring of hypertensive subjects [15–17].

2.2.2. RV diastolic dysfunction

Right ventricular systolic dysfunction has been reported in about 62% of a cohort of hypertensive patients [18, 19]. RV diastolic dysfunction may be an early clue to the development of hypertensive heart disease in Africans [19].

2.2.3. Atrial function and dysfunction

Absolute and indexed left atrial diameter, area, or volume is increased in African hypertensive subjects [20, 21]. Compared to their age- and sex-matched controls, hypertensive Africans show statistically significant left atrial structural and functional alterations [21].

2.3. Left ventricular hypertrophy

Electrocardiographic LVH occurs in 18–56% of hypertensive Africans depending on the criteria employed for the diagnosis. Sokolow-Lyon criteria appear to have the best sensitivity, while Estes score and Cornell criteria have the best specificity. Some workers in the region

have proposed new criteria for ECG diagnosis of LVH in Africans especially in obese subjects [22–28]. ECG LVH with strain pattern is associated with worse LV structure and function in hypertensive Africans [29, 30].

The prevalence of echocardiographic LVH ranges from 30.9 to 74%. This, however, depends on the threshold used for the indexation of LV mass. Adebiyi et al. report 61–74% prevalence of abnormal LV geometry in hospital patients at the University College Hospital, Ibadan, Nigeria [31].

In a similar study in Northern Tanzania [32], 70% of the hypertensive subjects have abnormal LV geometry. The distribution of the abnormal LV geometric patterns is 19.8, 28.2, and 22% for concentric remodeling, concentric hypertrophy, and eccentric LVH, respectively. The best yield appears to be when LV mass is indexed to height raised to the power of 2.7 (allomeric growth rate of the heart). Age, systolic blood pressure, and duration of hypertension are independent predictors of LVH.

2.3.1. LV systolic dysfunction

LV systolic dysfunction (LVSD) occurs in 18.1% (9.6, 3.7, and 4.8% for mild, moderate, and severe LVSD, respectively) of hypertensive Africans [33]. The independent predictors of LVSD are LV mass, body mass index, and male gender. Ojji et al. [34] report LVSD in 6.7% (mild—3.5%, moderate—2.3%, and severe—0.9%) of 1943 hypertensive subjects and LV dysfunction is associated with older age, male sex, presence of diabetes mellitus, and some indices of the LV structure.

The Tei index (index of global myocardial performance) is significantly higher in hypertensive Africans compared to controls. The index increases with severity of LVSD. It is negatively related to the LVEF.

2.3.2. RV systolic dysfunction

RV systolic dysfunction occurs in about in 32% of hypertensive subjects seen in tertiary centers in the region [18, 35, 36]. RVSD is worse in subjects with eccentric LV geometry. LVEF appears to be the main determinant of RVSD. Recently, Ojji et al. [37] reported RVSD in 44.5% of 611 hypertensive subjects. RVSD estimated by TAPSE <15mm is associated with worse prognosis. LVEF and right atrial area are the main determinants of RVSD.

2.4. Hypertensive heart failure

Hypertensive HF (HHF) is a common and major form of presentation of HF in Africa. **Table 1** shows the contribution of hypertension in the etiology of HHF in SSA. In the Heart of Soweto study [38], 54% of hypertensive patients visit the hospital on account of this disorder. This devastating form of HHD is often associated with concurrent LVH, renal dysfunction, and anemia. In a study of 180 HHF patients in Ghana, the mean age of presentation is 63.6 years (range-24–88 years) and seen more often in women. The mean systolic blood pressure at presentation is 162.4 mmHg. Shortness of breath, easy fatigability, and palpitation are common symptoms while pulmonary edema and displaced apex beat are the common signs.

S. No.	Author/Publication Year	Country	HHF (%)
1	Damasceno [1], 2012	9 countries	45.4
2	Stewart [41], 2008	South Africa (Soweto)	33.3
3	Ojji [42], 2009	Nigeria(Abuja)	62.6
4	Ojji [43], 2013	Nigeria(Abuja)	60.6
5	Ogah [44], 2014	Nigeria(Abeokuta)	78.5
6	Karaye [45], 2008	Nigeria(Kano)	57
7	Laabes [46], 2008	Nigeria(Jos)	44.1
8	Onwuchekwa [47], 2009	Nigeria(Port-Harcourt)	56.3
9	Adewuya [48], 2006	Nigeria(Ile-Ife)	54
10	Yonga [49], 2010	Kenya	64
11	Kingue [50], 2005	Cameroon (Urban)	54.5
12	Tantchou [51], 2011	Cameroon (rural)	15
13	Soliman [52], 2008	Malawi	24
14	Kuule [53], 2009	Uganda	24.2
15	Okello [54], 2014	Uganda	9.1
17	Owusu [55], 2013	Ghana (Outpatients-Kumasi)	45
18	Owusu [56], 2006	Ghana (In-patients-Kumasi)	42.6
19	Soliman [52], 2011	Sudan	28
20	Habte [57], 2010	Ethiopia	24.2
21	Makubi [58], 2014	Tanzania	45

Table 1. Summary of contribution of hypertension to HF in recent SSA studies.

Cardiomegaly on chest radiography is present in 75.6%. ECG-LVH or ECHO-LVH occur in 75.6 and 83.3%, respectively. About 62% have heart failure with preserved ejection fraction HFpEF [39].

In Nigeria, HHF is more common in men (56%). The mean age of presentation is 58.4 and 60.6 years in men and women, respectively. Over 80% present in NYHA class III and IV. HFpEF is present in about 35% of cases. The median length of hospital stay is about 9-days while 3.4% die while on admission. A 30-, 90-, and 180-day mortality rates of 0.9, 3.5, and 11.7%, respectively have been reported. Renal dysfunction appears to be the main independent predictor of mortality [40].

Table 2 shows the characteristics of African patients with HHF compared with similar patients in other parts of the world.

2.5. Possible pathophysiologic mechanism of hypertensive heart disease in SSA

Hypertension is a common cause of pressure overload of the left ventricle. LVH develops as an adaptation to this overload. Hypertensive patient with ECG LVH has 10-fold higher risk

Characteristics	Ogah et al. [40] (*n* = 320)	Stewart et al. [41] (*n* = 281)	Nieminen et al. [59] (*n* = 200)	Spinar et al. [60] (*n* = 179)	Venskutonyte et al. [61] (*n* = 65)
Female (%)	42.5	61	39.6	65.4	33.3
Mean age (yrs)	59.3	61	69.8	74.8	65.5
Denovo HF (%)	85.6	NA	37.3	74.3	66.7
NYHA III+IV (%)	82.2	29	NA	34.0	NA
Previous history of hypertension (%)	90.6	100	94.6	94.3	100
Diabetes mellitus (%)	12.2	14	34.5	43.1	33.3
Previous MI or CAD (%)	0.3	1.0	53.8	26.4	46.7
COPD (%)	2.5	NA	18.0	17.8	26.7
Stroke or TIA in history (%)	0.3	12	16.0	26.4	20
Atrial fibrillation (%)	12.8	9	37.7	19.0	46.7
Mean systolic BP(mmHg)	144	140	NA	198	NA
Mean diastolic BP(mmHg)	91	80	NA	100	NA
Heart rate (beats/min)	96	NA	NA	93	NA
Body mass index (kg/m^2)	24.2	NA	NA	28.0	33.9
Hospitalization for HF within last 12 months (%)	82.2	NA	NA	45.1	46.6
Renal failure (%)	14.4	27	18.7	NA	NA
Anemia (%)	11.5	10	11.3	NA	NA
Infection (%)	63.4	NA	15.6	NA	13.3
Noncompliance with therapy (%)	74.1	NA	21.9	NA	66.7
ACE inhibitors (%)	99.1	NA	NA	71.3	NA
Beta-blockers (%)	2.7	NA	NA	77.0	NA
Calcium antagonists (%)	30.6	NA	NA	51.1	NA
Diuretics (%)	86.9	NA	NA	88.5	NA
Spironolactone (%)	81.3	NA	NA	36.2	NA
Digoxin (%)	73.1	NA	NS	13.8	NA
LVEDD (mm)	55	46	56	NA	50*
Mean ejection fraction	42.7	53	44	55	50.5
LA (mm)	47	NA	45	NA	42*
Mitral regurgitation (%)	79.1	7	77.6	NA	100
Tricuspid regurgitation (%)	60.8	6	53.7	NA	93.3
LOS days, median	9	NA	8	NA	13
Intrahospital mortality (%)	3.4	NA	1.5	2.2	6.6

Abbreviation: HOS = Heart of Soweto Study, EHFS II = European Heart Failure Survey II, AHEAD = Acute Heart Failure Database, HF = Heart Failure, NYHA = New York Heart Association, MI = Myocardial Infarction, CAD = Coronary Artery Disease, COPD = Chronic Obstructive Pulmonary Disease, TIA = Transient Ischemic Attack, BP = Blood Pressure, LVEDD = Left Ventricular End-Diastolic Diameter, LA = Left Atrium, LOS = Length of Hospital Stay

Table 2. Comparison of our findings with similar studies in other parts of the world.

of developing HF [41]. There is increased wall thickness at the expense of chamber volume in LVH due to hypertrophy of the myocyte and by a parallel alignment of the sarcomere [42]. Specific hypertensive cardiomyopathy has been proposed. This cardiomyopathy has been divided into four stages: in stage 1, there is diastolic dysfunction, which is present in 20–30% of patients. This is common in elderly women, hypertensive diabetics, and ischemic heart disease patients [43]. LV diastolic dysfunction precedes systolic HF and is therefore a more common mechanism of HF in hypertension. Stage 2 is hypertension with impaired LV relaxation abnormalities, while grade 4 is dilated cardiomyopathy with LV systolic dysfunction. It has been shown that apoptosis may be responsible for the reduction of myocyte mass that accompanies progression from compensated hypertrophy to HF.

Several theories have been proposed to explain the relationship between LVH and HF. This includes changes in the coronary microcirculation, which leads to poor myocardial perfusion, impaired cardiac function, loss of contractile protein, and thus reduced cardiac contractility [44]. The second theory is increased LV pressure overload, which leads to ventricular dilatation and reduced cardiac output [45].

Finally, LVH in hypertension is governed by different loading conditions, which involve both hormonal and paracrine factors such as the sympathetic nervous system and renin-angiotensin-aldosterone axis [46].

3. Peripartum cardiomyopathy (PPCM)

3.1. Definition

Peripartum cardiomyopathy (PPCM) is a form of heart disease characterized by "the development of HF in the last month of pregnancy or within the first 5 months postpartum in the absence of any other determinable cause for cardiac failure and in the absence of demonstrable heart disease before the last month of pregnancy, and bears echocardiographic evidence of left ventricular systolic dysfunction" [62]. In addition, the diagnosis of the condition requires evidence of impaired LV systolic function by echocardiography (LVEF < 45% or LVFS < 30%). LV dilation is common although in some patients, LV dimension may be normal but the LV systolic function is impaired [62, 63].

3.2. Epidemiology

In terms of epidemiology, PPCM is common in developing and poor communities. The incidence is 1/1000 in most parts of low- and middle-income countries [64]. However, very high incidence has been reported from Northern Nigeria (1/100 live births) [65–69] and Haiti (1/300 live births) [70, 71]. The incidence in high-income countries is in the range of 1/3000–1/4000 deliveries [64]. There has been an increase in the awareness of the disease worldwide with the establishment of a global registry.

PPCM is responsible for about 1.5% cases of HF in the Heart of Soweto study [41], 1.3% in the Abeokuta HF registry [44] and 3.2% in the Abuja Heart Study [42]. It is still the most prevalent form of cardiomyopathy (54.6%) in Northern Nigeria [45].

3.3. Risk factors

Risk factors for the development of this cardiac disorder include low socioeconomic status, women of African descent (although PPCM is a global disease), young pregnant women, multiparity, multiple pregnancy, and longer period of breast feeding [64]. However, recent prospectively collected data on PPCM do not support strong association with older age of pregnancy, multiparity, twin pregnancy, gestational hypertension, and the use of tocolytic agents [72].

3.4. Clinical features

Shortness of breath is common form of presentation. Other common clinical features include, cardiomegaly, tachycardia, pulmonary rales, high blood pressure and dysrhythmias. Dyspnea, cough, orthopnea, palpitation, hemoptysis, chest pain, and abdominal pain are other common features. Most patients in SSA present in NYHA class III/IV [68, 72]. Thromboembolic complications are common in the form of pulmonary embolism and stroke from mural thrombus [73, 74].

There are some differences between PPCM and hypertensive heart failure of pregnancy (HHFP). Patients with HHFP are more likely to present in the last trimester, while PPCM patients are more likely to present within the first month of the postpartum period. Family history of hypertension and history of hypertension in previous pregnancy is commoner in HHFP. Twin pregnancy and presence of leg edema are more common in PPCM. Blood pressures are generally higher in HHFP and they are also more likely to have basal rales. Furthermore, functional murmurs (tricuspid and mitral regurgitation) occur more often in PPCM compared to HHFP [75].

3.5. Laboratory findings

Arrhythmias are also common. In severe cases, anemia and renal dysfunction may be present. The liver enzymes may be normal or mildly raised from hepatic congestion. Some authors in Benin republic, Mali and Nigeria have reported the association of PPCM with micronutrient deficiencies, e.g., selenium, ceruloplasmin [76–78]. LV function and mortality in PPCM patients with HIV infection and those without have been found not to differ significantly [79].

The 12-leads ECG often show sinus rhythm, ST-T changes are common, which resolves after the postpartum. Ventricular arrhythmia occurs in about 20% [80–83].

Echocardiography is the diagnostic procedure of choice. Useful for the evaluation of LV systolic function ($EF < 45\%$), and diastolic function as well as assessment for presence of intramural thrombus formation. The mean LV internal dimension in diastole is often about 6 cm; however, some patients have nondilated LV. Where available in SSA, magnetic resonance imaging helps in the detection of myocardial fibrosis with late enhancement imaging. It also helps in the assessment function, shape, size, as well as contents. Immunohistochemistry of biopsy specimen from patient with PPCM is not different from that of idiopathic DCM. Similar viral particles, e.g., coxsackie, encephalomyelocarditis, parvovirus B19, adenoviruses, herpes simplex virus, Ebstein-Burr virus, and cytomegalovirus DNA. Inflammatory markers such as tumor necrosis factor alpha (TNF-alpha) and C-reactive protein levels are raised in

both conditions and cannot be used to differentiate one from the other. However, peculiar to PPCM are some immune activation processes, e.g., elevated levels of marker of apoptosis-FAS/APO 1. This has been shown to predict prognosis [84].

3.6. Recent advances in the pathophysiology

More recently, Sliwa and her colleagues have shown the role of cleavage of prolactin in the pathogenesis of PPCM. A 16-KDa fragment of prolactin may induce myocardial damage [85]. This has provided a new option of blocking prolactin secretion with bromocriptine in the treatment of PPCM.

3.7. Prognosis

Full recovery of LV function occurs in about half of PPCM patients [72]. About 25% recover by the end of 6 months and around 10–15% die within 6 months. Long-term prospective follow-up studies show that overall recovery occurs in about 25% of patients and this mostly occurs in the first 18–24 months of diagnosis [79].

In recent time, there has been an increased awareness of this condition, and it has been recognized in the guidelines of the American College of Cardiology and European Society of Cardiology. Large global or continental registries of PPCM exist and many centers in SSA are participating. The European society of Cardiology has recently released a position paper on the disorder [62].

4. HIV-associated cardiomyopathy

4.1. Prevalence

SSA contributes about 69 and 90% of the global adult and childhood HIV/AIDS burden. HIV-associated cardiomyopathy is therefore a significant contributor to CVD morbidity and mortality in the region [86, 87].

The true prevalence of HIV-associated cardiomyopathy is unknown. The prevalence of HIV-associated cardiomyopathy in the pre-HAART era was about 50%. The incidence of any cardiac abnormality in HIV-infected individuals was 55% over a 7-year period [88–90]. It was common in young persons with CD4 count of <100 cells/mm^3, lower socioeconomic class, longer duration of the infection, higher viral load, and advanced stage of the disease [89, 91]. In-hospital mortality was 15% [89].

Because of the availability of HAART, the prevalence has reduced by about 50% in high-income countries [92]. However, in low-income countries (where most of the countries in SSA belong to), the prevalence of the condition has increased by 32% due to poor and limited access to HAART as well the impact of malnutrition [93].

Echocardiographic studies have reported prevalence ranging from 5% (in Nigeria) to 57% in Burkina Faso [89, 91, 94]. Differences may be due to study design and lack of common definition of the disorder [95].

In the Heart of Soweto study, about 9.7% of the cohort were HIV infected, 54% of who were on HAART [41, 81]. They were younger, had lower blood pressure and body mass index, and higher heart rate compared to the general cohort. HIV associated HF was the commonest diagnosis. The mean LVEF was 46% and common in women who were also about 6-years younger than the men. HIV patients who had HF had lower CD4 count compared to those who did not have. They were also more likely to have right-heart failure and valve dysfunction [96].

About 2.6% of HF cases in the THESU-HF survey were due to HIV infection. They were younger by 10–15 years and were less often smokers, hypertensive, or diabetic. They had larger LV dimensions but had similar LVEF compared to the general cohort [1]. The findings from the Heart of Soweto and the THESUS-HF survey are similar to more recent observational studies in the region. The prevalence is in the range of 1–5% [67, 73, 74]. It is often diagnosed in the third decade of life and more often in women. Both systolic and diastolic HF are common (about 30%).

4.2. Pathophysiologic mechanism

The proposed mechanism in the pathogenesis of HIV-associated cardiomyopathy include the direct myocardial invasion by the HIV, post-viral autoimmunity, immune system dysregulation, adverse effect of the viral protein, endothelial dysfunction, transcriptional activation of cellular genes, and beta-adrenergic dysregulation. Others include HIV-immunosuppression-related myocarditis due to opportunistic infection with toxoplasmosis, cryptococcus, and mycobacteria. Myocardial dysfunction as a result of systemic effects of sepsis may also play a role. Some of the anti-retroviral medications may play a role in the pathogenesis. Nucleoside reverse transcriptase inhibitors cause mitochondrial damage by inhibiting mitochondrial DNA polymerase. Zalcitabine is thought to exhibit the greatest toxicity among this group [97]. Zidovudine causes cardiac and skeletal myopathy [98].

Malnutrition especially selenium deficiency is another possible mechanism. Selenium has an antioxidant property and protects against endothelial dysfunction. Its deficiency is associated with cardiac dysfunction. Due to soil composition and agricultural practices in the region, selenium deficiency is common and 285 of the SSA population are at risk of selenium deficiency. Selenium deficiency has been demonstrated in HIV patients [99]. Selenium supplementation has also been shown to improve cardiac function in some studies [100].

Heavy alcohol use and smoking have also been implicated especially in high-income countries [101]. This was not demonstrated in a Rwandan study [101].

The role of genetic factor has not been demonstrated in Africa. " The mitochondrial DNA T16189C polymorphism, with a homopolymeric C-tract of 10-12 cystosines—a putative genetic risk factor for idiopathic dilated cardiomyopathy in the African and British populations—was not associated with HIV-associated cardiomyopathy in a South-African case control study" [102].

Author details

Okechukwu S. Ogah* and Ayodele O. Falase

*Address all correspondence to: osogah56156@gmail.com

Division of Cardiology, Department of Medicine, University College Hospital, Ibadan, Nigeria

References

[1] Damasceno A, Mayosi BM, Sani M, Ogah OS, Mondo C, Ojji D, et al. The causes, treatment, and outcome of acute heart failure in 1006 Africans from 9 countries: results of the sub-Saharan Africa survey of heart failure. Archives of Internal Medicine. 2012;172 (18):1386–94.

[2] Adeloye D, Basquill C. Estimating the prevalence and awareness rates of hypertension in Africa: a systematic analysis. PLoS One. 2014;9(8):e104300.

[3] Organization WH. A global brief on hypertension: silent killer, global public health crisis. World. 2016.

[4] WHO RO for Africa. The health of the people: the African regional health report. World Health Organization. 2006.

[5] Lawes CM, Vander Hoorn S, Law MR, Elliott P, MacMahon S, Rodgers A. Blood pressure and the global burden of disease 2000. Part 1: estimates of blood pressure levels. Journal of Hypertension. 2006;24(3):413–22.

[6] Lawes CM, Vander Hoorn S, Law MR, Elliott P, MacMahon S, Rodgers A. Blood pressure and the global burden of disease 2000. Part II: estimates of attributable burden. Journal of Hypertension. 2006;24(3):423–30.

[7] Beaglehole R, Bonita R, Alleyne G, Horton R, Li L, Lincoln P, et al. UN high-level meeting on non-communicable diseases: addressing four questions. The Lancet. 2011;378(9789):449–55.

[8] Adebiyi AA, Akinosun OM, Nwafor CE, Falase AO. Plasma catecholamines in Nigerians with primary hypertension. Ethnicity & Disease. 2011;21(2):158–62.

[9] Adamu UG, Kolo PM, Katibi IA, Opadijo GO, Omotosho AB, Araoye MA. Relationship between left ventricular diastolic function and geometric patterns in Nigerians with newly diagnosed systemic hypertension. Cardiovascular Journal of Africa. 2009;20(3):173–7.

[10] Adebiyi AA, Aje A, Ogah OS, Ojji DB, Oladapo OO, Falase AO. Left ventricular diastolic function parameters in hypertensives. Journal of the National Medical Association. 2005;97(1):41–5.

[11] Akintunde AA, Familoni OB, Akinwusi PO, Opadijo OG. Relationship between left ventricular geometric pattern and systolic and diastolic function in treated Nigerian hypertensives. Cardiovascular Journal of Africa. 2010;21(1):21–5.

[12] Ike SO, Onwubere JC. For the patient. Poor diastolic function and blood pressure in Blacks. Ethnicity & Disease. 2003;13(4):547.

[13] Adebayo AK, Oladapo OO, Adebiyi AA, Ogunleye OO, Ogah OS, Ojji DB, et al. Characterisation of left ventricular function by tissue Doppler imaging technique in newly diagnosed, untreated hypertensive subjects. Cardiovascular Journal of Africa. 2008;19(5):259–63.

[14] Akintunde A, Kareem L, Bakare A, Audu M. Impact of obstructive sleep apnea and snoring on left ventricular mass and diastolic function in hypertensive Nigerians. Annals of Medical and Health Sciences Research. 2014;4(3):350–4.

[15] Adeoye AM, Adebiyi AA, Oladapo OO, Ogah OS, Aje A, Ojji DB, et al. Early diastolic functional abnormalities in normotensive offspring of Nigerian hypertensives. Cardiovascular Journal of Africa. 2012;23(5):255–9.

[16] Kolo P, Sanya E, Ogunmodede J, Omotoso A, Soladoye A. Normotensive offspring of hypertensive Nigerians have increased left ventricular mass and abnormal geometric patterns. The Pan African Medical Journal. 2012;11:6.

[17] Kolo PM, Sanya EO, Omotoso AB, Soladoye A, Ogunmodede JA. Left ventricular hypertrophy is associated with diastolic filling alterations in normotensive offspring of hypertensive Nigerians. ISRN Cardiology. 2012;2012:256738.

[18] Karaye KM, Habib AG, Mohammed S, Rabiu M, Shehu MN. Assessment of right ventricular systolic function using tricuspid annular-plane systolic excursion in Nigerians with systemic hypertension. Cardiovascular Journal of Africa. 2010;21(4):186–90.

[19] Akintunde AA, Akinwusi PO, Familoni OB, Opadijo OG. Effect of systemic hypertension on right ventricular morphology and function: an echocardiographic study. Cardiovascular Journal of Africa. 2010;21(5):252–6.

[20] Adebiyi AA, Aje A, Ogah OS, Ojji DB, Dada A, Oladapo OO, et al. Correlates of left atrial size in Nigerian hypertensives. Cardiovascular Journal of South Africa: Official Journal for Southern Africa Cardiac Society [and] South African Society of Cardiac Practitioners. 2004;16(3):158–61.

[21] Adebayo AK, Oladapo OO, Adebiyi AA, Ogunleye OO, Ogah OS, Ojji DB, et al. Changes in left atrial dimension and function and left ventricular geometry in newly diagnosed untreated hypertensive subjects. Journal of Cardiovascular Medicine. 2008;9(6):561–9.

[22] Jaggy C, Perret F, Bovet P, van Melle G, Zerkiebel N, Madeleine G, et al. Performance of classic electrocardiographic criteria for left ventricular hypertrophy in an African population. Hypertension. 2000;36(1):54–61.

[23] Huston SL, Bunker CH, Ukoli FA, Rautaharju PM, Kuller LH. Electrocardiographic left ventricular hypertrophy by five criteria among civil servants in Benin City, Nigeria: prevalence and correlates. International Journal of Cardiology. 1999;70(1):1–14.

[24] Lodha SM, Makene WJ. Electrocardiographic changes in systemic hypertension. (A study of Tanzanian Africans). East African Medical Journal. 1976;53(8):424–34.

[25] Maunganidze F, Woodiwiss AJ, Libhaber CD, Maseko MJ, Majane OH, Norton GR. Left ventricular hypertrophy detection from simple clinical measures combined with electrocardiographic criteria in a group of African ancestry. Clinical Research in Cardiology. 2014;103(11):921–9.

[26] Maunganidze F, Woodiwiss AJ, Libhaber CD, Maseko MJ, Majane OH, Norton GR. Obesity markedly attenuates the validity and performance of all electrocardiographic criteria for left ventricular hypertrophy detection in a group of black African ancestry. Journal of Hypertension. 2013;31(2):377–83.

[27] Sliwa K, Ojji D, Bachelier K, Bohm M, Damasceno A, Stewart S. Hypertension and hypertensive heart disease in African women. Clinical Research in Cardiology. 2014; 103(7):515–23.

[28] Robinson C, Woodiwiss AJ, Libhaber CD, Norton GR. Novel approach to the detection of left ventricular hypertrophy using body mass index–corrected electrocardiographic voltage criteria in a group of African ancestry. Clinical Cardiology. 2016;39(9):524–30.

[29] Ogah O, Oladapo O, Adebiyi A, Salako B, Falase A, Adebayo A, et al. Electrocardiographic left ventricular hypertrophy with strain pattern: prevalence, mechanisms and prognostic implications. Cardiovascular Journal of Africa. 2008;19(1):39.

[30] Ogah O, Adebiyi A, Oladapo O, Aje A, Ojji D, Adebayo A, et al. Association between electrocardiographic left ventricular hypertrophy with strain pattern and left ventricular structure and function. Cardiology. 2006;106(1):14–21.

[31] Adebiyi AA, Ogah OS, Aje A, Ojji DB, Adebayo AK, Oladapo OO, et al. Echocardiographic partition values and prevalence of left ventricular hypertrophy in hypertensive Nigerians. BMC Medical Imaging. 2006;6(1):1.

[32] Silangei LK, Maro VP, Diefenthal H, Kapanda G, Dewhurst M, Mwandolela H, et al. Assessment of left ventricular geometrical patterns and function among hypertensive patients at a tertiary hospital, Northern Tanzania. BMC Cardiovascular Disorders. 2012;12:109.

[33] Ogah OS, Akinyemi RO, Adegbite GD, Udofia OI, Udoh SB, Adesina JO, et al. Prevalence of asymptomatic left ventricular systolic dysfunction in hypertensive Nigerians: echocardiographic study of 832 subjects. Cardiovascular Journal of Africa. 2011;22(6):297–302.

[34] Ojji D, Atherton J, Sliwa K, Alfa J, Ngabea M, Opie L. Left ventricular systolic dysfunction in asymptomatic black hypertensive subjects. American Journal of Hypertension. 2015;28(7):924–9.

[35] Karaye KM. Relationship between Tei Index and left ventricular geometric patterns in a hypertensive population: a cross-sectional study. Cardiovascular Ultrasound. 2011;9:21.

[36] Karaye KM, Sai'du H, Shehu MN. Right ventricular dysfunction in a hypertensive population stratified by patterns of left ventricular geometry. Cardiovascular Journal of Africa. 2012;23(9):478–82.

[37] Ojji DB, Lecour S, Atherton JJ, Blauwet LA, Alfa J, Sliwa K. Right ventricular systolic dysfunction is common in hypertensive heart failure: a prospective study in sub-Saharan Africa. PLoS One. 2016;11(4):e0153479.

[38] Stewart S, Libhaber E, Carrington M, Damasceno A, Abbasi H, Hansen C, et al. The clinical consequences and challenges of hypertension in urban-dwelling black Africans: insights from the Heart of Soweto Study. International Journal of Cardiology. 2011;146(1):22–7.

[39] Owusu IK, Adu-Boakye Y, Tetteh LA. Hypertensive heart failure in Kumasi, Ghana. Open Science Journal of Clinical Medicine. 2014;2(1):39–43.

[40] Ogah OS, Sliwa K, Akinyemi JO, Falase AO, Stewart S. Hypertensive heart failure in Nigerian Africans: insights from the Abeokuta Heart Failure Registry. Journal of Clinical Hypertension. 2015;17(4):263–72.

[41] Stewart S, Wilkinson D, Hansen C, Vaghela V, Mvungi R, McMurray J, et al. Predominance of heart failure in the Heart of Soweto Study cohort: emerging challenges for urban African communities. Circulation. 2008;118(23):2360–7.

[42] Ojji DB, Alfa J, Ajayi SO, Mamven MH, Falase AO. Pattern of heart failure in Abuja, Nigeria: an echocardiographic study. Cardiovascular Journal of Africa. 2009;20(6):349–52.

[43] Ojji D, Stewart S, Ajayi S, Manmak M, Sliwa K. A predominance of hypertensive heart failure in the Abuja Heart Study cohort of urban Nigerians: a prospective clinical registry of 1515 de novo cases. European Journal of Heart Failure. 2013;15(8):835–42.

[44] Ogah OS, Stewart S, Falase AO, Akinyemi JO, Adegbite GD, Alabi AA, et al. Contemporary profile of acute heart failure in Southern Nigeria: data from the Abeokuta Heart Failure Clinical Registry. JACC Heart Failure. 2014;2(3):250–9.

[45] Karaye KM, Sani MU. Factors associated with poor prognosis among patients admitted with heart failure in a Nigerian tertiary medical centre: a cross-sectional study. BMC Cardiovascular Disorders. 2008;8:16.

[46] Laabes EP, Thacher TD, Okeahialam BN. Risk factors for heart failure in adult Nigerians. Acta Cardiologica. 2008;63(4):437–43.

[47] Onwuchekwa AC, Asekomeh GE. Pattern of heart failure in a Nigerian teaching hospital. Vascular Health Risk Management. 2009;5:745–50.

[48] Adewuya AO, Ola BA, Ajayi OE, Oyedeji AO, Balogun MO, Mosaku SK. Prevalence and correlates of major depressive disorder in Nigerian outpatients with heart failure. Psychosomatics. 2006;47(6):479–85.

[49] Prevalence, causes and risk factors for left ventricular dysfunction and heart failure in Kenya population [Internet]. 2009 [cited 14 Dec 2014]. Available from: http://spo.escardio.org/eslides/view.aspx?eevtid=40&fp=3567.

[50] Kingue S, Dzudie A, Menanga A, Akono M, Ouankou M, Muna W. [A new look at adult chronic heart failure in Africa in the age of the Doppler echocardiography: experience of the medicine department at Yaounde General Hospital]. In Annales de cardiologie et d'angeiologie 2005 Sep;54(5):276–283.

[51] Tantchou Tchoumi JC, Ambassa JC, Kingue S, Giamberti A, Cirri S, Frigiola A, et al. Occurrence, aetiology and challenges in the management of congestive heart failure in sub-Saharan Africa: experience of the Cardiac Centre in Shisong, Cameroon. The Pan African Medical Journal. 2011;8:11.

[52] Soliman EZ, Juma H. Cardiac disease patterns in northern Malawi: epidemiologic transition perspective. Journal of Epidemiology/Japan Epidemiological Association. 2008;18(5):204–8.

[53] Kuule JK, Seremba E, Freers J. Anaemia among patients with congestive cardiac failure in Uganda—its impact on treatment outcomes. South African Medical Journal = Suid-Afrikaanse tydskrif vir geneeskunde. 2009;99(12):876–80.

[54] Okello S, Rogers O, Byamugisha A, Rwebembera J, Buda AJ. Characteristics of acute heart failure hospitalizations in a general medical ward in Southwestern Uganda. International Journal of Cardiology. 2014;176(3):1233–4.

[55] Owusu IK, Boaky Y. Prevalence and aetiology of heart failure in patients seen at a Teaching Hospital in Ghana. Journal of Cardiovascular Diseases & Diagnosis. 2013;1:131.

[56] Owusu IK. Causes of heart failure as seen in Kumasi, Ghana. Internet J Third world med. 2007;5:201–14.

[57] Habte B, Alemseged F, Tesfaye D. The pattern of cardiac diseases at the cardiac clinic of Jimma University specialised hospital, South West Ethiopia. Ethiopian Journal of Health Sciences. 2010;20(2):99–105.

[58] Makubi A, Hage C, Lwakatare J, Kisenge P, Makani J, Ryden L, et al. Contemporary aetiology, clinical characteristics and prognosis of adults with heart failure observed in a tertiary hospital in Tanzania: the prospective Tanzania Heart Failure (TaHeF) study. Heart. 2014;100(16):1235–41.

[59] Nieminen MS, Brutsaert D, Dickstein K, Drexler H, Follath F, Harjola VP, et al. EuroHeart Failure Survey II (EHFS II): a survey on hospitalized acute heart failure patients: description of population. European Heart Journal. 2006;27(22):2725–36.

[60] Sato N, Kajimoto K, Asai K, Mizuno M, Minami Y, Nagashima M, et al. Acute decompensated heart failure syndromes (ATTEND) registry. A prospective observational multicenter cohort study: rationale, design, and preliminary data. American Heart Journal. 2010;159(6):949–55 e1.

[61] Venskutonyte L, Molyte I, Ablonskyte-Dudoniene R, Mizariene V, Kavoliuniene A. Characteristics and management of acute heart failure patients in a single university hospital center. Medicina. 2009;45(11):855–70.

[62] Sliwa K, Hilfiker-Kleiner D, Petrie MC, Mebazaa A, Pieske B, Buchmann E, Regitz-Zagrosek V, Schaufelberger M, Tavazzi L, Veldhuisen DJ, Watkins H. Current state of knowledge on aetiology, diagnosis, management, and therapy of peripartum cardiomyopathy: a position statement from the Heart Failure Association of the European Society of Cardiology Working Group on peripartum cardiomyopathy. European journal of heart failure. 2010 Aug 1;12(8):767–78.

[63] Pearson GD, Veille J-C, Rahimtoola S, Hsia J, Oakley CM, Hosenpud JD, et al. Peripartum cardiomyopathy: national heart, lung, and blood institute and office of rare diseases (national institutes of health) workshop recommendations and review. Journal of the American Medical Association. 2000;283(9):1183–8.

[64] Sliwa K, Fett J, Elkayam U. Peripartum cardiomyopathy. The Lancet. 2006;368(9536):687–93.

[65] Ford L, Abdullahi A, Anjorin FI, Danbauchi SS, Isa MS, Maude GH, et al. The outcome of peripartum cardiac failure in Zaria, Nigeria. QJM. 1998;91(2):93–103.

[66] Abengowe CU. Cardiovascular disease in Northern Nigeria. Tropical & Geographical Medicine. 1979;31(4):553–60.

[67] Abengowe CU, Das CK, Siddique AK. Cardiac failure in pregnant Northern Nigerian women. International Journal of Gynaecology & Obstetrics: the Official Organ of the International Federation of Gynaecology and Obstetrics. 1980;17(5):467–70.

[68] Isezuo SA, Abubakar SA. Epidemiologic profile of peripartum cardiomyopathy in a tertiary care hospital. Ethnicity & Disease. 2007;17(2):228–33.

[69] Ladipo GO, Froude JR, Parry EH. Pattern of heart disease in adults of the NigerianwSavanna: a prospective clinical study. African Journal of Medicine and Medical Sciences. 1977;6(4):185–92.

[70] Fett JD. Peripartum cardiomyopathy. Insights from Haiti regarding a disease of unknown etiology. Minnesota Medicine. 2002;85(12):46–8.

[71] Fett JD, Carraway RD, Dowell DL, King ME, Pierre R. Peripartum cardiomyopathy in the Hospital Albert Schweitzer District of Haiti. American Journal of Obstetrics & Gynecology. 2002;186(5):1005–10.

[72] Blauwet LA, Libhaber E, Forster O, Tibazarwa K, Mebazaa A, Hilfiker-Kleiner D, et al. Predictors of outcome in 176 South African patients with peripartum cardiomyopathy. Heart. 2013;99(5):308–13.

[73] Isezuo SA, Njoku CH, Airede L, Yaqoob I, Musa AA, Bello O. Case report: acute limb ischaemia and gangrene associated with peripartum cardiomyopathy. Nigerian Postgraduate Medical Journal. 2005;12(3):237–40.

[74] Talle MA, Buba F, Anjorin CO. Prevalence and aetiology of left ventricular thrombus in patients undergoing transthoracic echocardiography at the University of Maiduguri Teaching Hospital. Advances in Medicine. 2014;2014:731936.

[75] Ntusi NB, Badri M, Gumedze F, Sliwa K, Mayosi BM. Pregnancy-associated heart failure: a comparison of clinical presentation and outcome between hypertensive heart failure of pregnancy and idiopathic peripartum cardiomyopathy. PLoS One. 2015;10(8):e0133466

[76] Cenac A, Simonoff M, Moretto P, Djibo A. A low plasma selenium is a risk factor for peripartum cardiomyopathy. A comparative study in Sahelian Africa. International Journal of Cardiology. 1992;36(1):57–9.

[77] Cenac A, Toure K, Diarra MB, Sergeant C, Jobic Y, Sanogo K, et al. Plasma selenium and peripartum cardiomyopathy in Bamako, Mali. Medecine Tropicale (Mars). 2004; 64(2):151–4.

[78] Cenac A, Sacca-Vehounkpe J, Poupon J, Dossou-Yovo-Akindes R, D'Almeida-Massougbodji M, Tchabi Y, et al. [Serum selenium and dilated cardiomyopathy in Cotonou, Benin]. Medecine Tropicale (Mars). 2009;69(3):272–4.

[79] Sliwa K, Forster O, Tibazarwa K, Libhaber E, Becker A, Yip A, et al. Long-term outcome of peripartum cardiomyopathy in a population with high seropositivity for human immunodeficiency virus. International Journal of Cardiology. 2011;147(2):202–8.

[80] Karaye KM, Lindmark K, Henein MY. Electrocardiographic predictors of peripartum cardiomyopathy. Cardiovascular Journal of Africa. 2015;27(2):66–70.

[81] Sliwa K, Wilkinson D, Hansen C, Ntyintyane L, Tibazarwa K, Becker A, et al. Spectrum of heart disease and risk factors in a black urban population in South Africa (the Heart of Soweto Study): a cohort study. The Lancet. 2008;371(9616):915–22.

[82] Karaye KM. Clinical characteristics and prognosis of Peripartum Cardiomyopathy. Print and Media, Umea University, 2016.

[83] Tibazarwa K, Mayosi B, Sliwa K, Carrington M, Stewart S, Lee G. The 12-lead ECG in peripartum cardiomyopathy. Cardiovascular Journal of Africa. 2012;23(6):322–9.

[84] Sliwa K, Forster O, Libhaber E, Fett JD, Sundstrom JB, Hilfiker-Kleiner D, et al. Peripartum cardiomyopathy: inflammatory markers as predictors of outcome in 100 prospectively studied patients. European Heart Journal. 2006;27(4):441–6.

[85] Hilfiker-Kleiner D, Sliwa K. Pathophysiology and epidemiology of peripartum cardiomyopathy. Nature Reviews Cardiology. 2014;11(6):364–70.

[86] WHO. Global update on HIV treatment 2013: results, impact and opportunities. 2013.

[87] HIV/AIDS JUNPo. Global AIDS response progress reporting 2013: construction of core indicators for monitoring the 2011 UN Political Declaration on HIV. AIDS. 2013.

[88] Hakim J, Matenga J, Siziya S. Myocardial dysfunction in human immunodeficiency virus infection: an echocardiographic study of 157 patients in hospital in Zimbabwe. Heart. 1996;76(2):161–5.

[89] Niakara A, Drabo Y, Kambire Y, Nebie L, Kabore N, Simon F. [Cardiovascular diseases and HIV infection: study of 79 cases at the National Hospital of Ouagadougou (Burkina Faso)]. Bulletin de la Societe de Pathologie Exotique (1990). 2002;95(1):23–6.

[90] Longo-Mbenza B, Seghers K, Phuati M, Bikangi FN, Mubagwa K. Heart involvement and HIV infection in African patients: determinants of survival. International Journal of Cardiology. 1998;64(1):63–73.

[91] Twagirumukiza M, Nkeramihigo E, Seminega B, Gasakure E, Boccara F, Barbaro G. Prevalence of dilated cardiomyopathy in HIV-infected African patients not receiving HAART: a multicenter, observational, prospective, cohort study in Rwanda. Current HIV Research. 2007;5(1):129–37.

[92] Patel K, Van Dyke RB, Mittleman MA, Colan SD, Oleske JM, Seage III GR. The impact of HAART on cardiomyopathy among children and adolescents perinatally infected with HIV-1. AIDS (London, England). 2012;26(16):2027.

[93] Barbaro G. WJC. World. 2010;2(3):53–7.

[94] Olusegun-Joseph DA, Ajuluchukwu JN, Okany CC, Mbakwem AC, Oke DA, Okubadejo NU. Echocardiographic patterns in treatment-naive HIV-positive patients in Lagos, south-west Nigeria. Cardiovascular Journal of Africa. 2012;23(8):e1–6.

[95] Ntsekhe M, Mayosi BM. Cardiac manifestations of HIV infection: an African perspective. Nature Clinical Practice Cardiovascular Medicine. 2009;6(2):120–7.

[96] Sliwa K, Carrington MJ, Becker A, Thienemann F, Ntsekhe M, Stewart S. Contribution of the human immunodeficiency virus/acquired immunodeficiency syndrome epidemic to de novo presentations of heart disease in the Heart of Soweto Study cohort. European Heart Journal. 2012;33(7):866–74.

[97] Currie PF, Jacob AJ, Foreman AR, Elton RA, Brettle RP, Boon NA. Heart muscle disease related to HIV infection: prognostic implications. BMJ. 1994;309(6969):1605–7.

[98] Nzuobontane D, Blackett K, Kuaban C. Cardiac involvement in HIV infected people in Yaounde, Cameroon. Postgraduate Medical Journal. 2002;78(925):678–81.

[99] Look M, Rockstroh J, Rao G, Kreuzer K, Barton S, Lemoch H, et al. Serum selenium, plasma glutathione (GSH) and erythrocyte glutathione peroxidase (GSH-Px)-levels in asymptomatic versus symptomatic human immunodeficiency virus-1 (HIV-1)-infection. European Journal of Clinical Nutrition. 1997;51(4):266–72.

[100] Kavanaugh-Mchugh AL, Ruff A, Perlman E, Hutton N, Modlin J, Rowe S. Selenium deficiency and cardiomyopathy in acquired immunodeficiency syndrome. Journal of Parenteral and Enteral Nutrition. 1991;15(3):347–9.

[101] Mondy KE, Gottdiener J, Overton ET, Henry K, Bush T, Conley L, et al. High prevalence of echocardiographic abnormalities among HIV-infected persons in the era of highly active antiretroviral therapy. Clinical Infectious Diseases. 2011;52(3):378–86.

[102] Hsue PY, Deeks SG, Hunt PW. Immunologic basis of cardiovascular disease in HIVinfected adults. Journal of Infectious Diseases. 2012;205(suppl 3):S375–82.