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

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Additional information is available at the end of the chapter

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## 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.

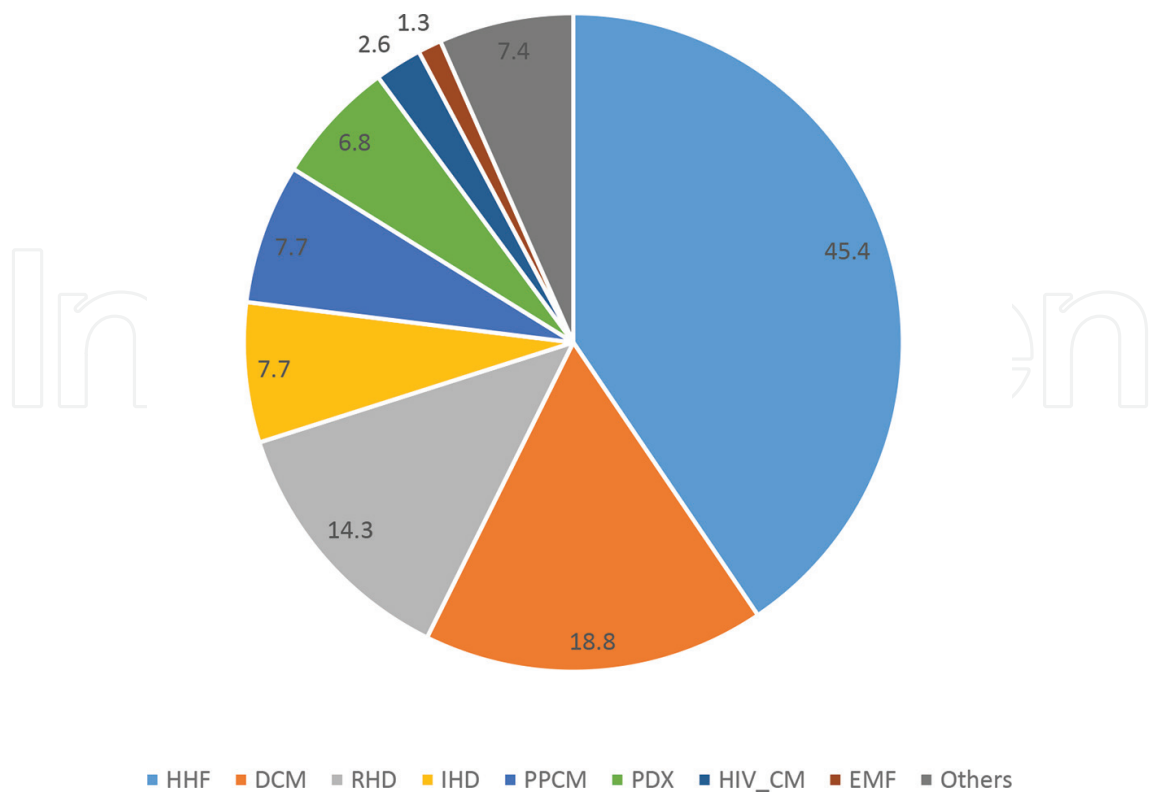
**Keywords:** cardiomyopathy, heart muscle disease, hypertensive heart disease, peripartum cardiomyopathy, HIV-associated cardiomyopathy, heart failure

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## 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.

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**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].

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. Adebisi 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 (allometric 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. Oji 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 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, Oji 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	Oji [42], 2009	Nigeria(Abuja)	62.6
4	Oji [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 <sup>2</sup> )	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, Epstein-Barr 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<sup>3</sup>, 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].

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