

## HIV-Infection and Cardiovascular Health Hazards

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### Editorial

Globally, around 40 million individuals are living with human immunodeficiency virus (HIV), with a global HIV prevalence of 0.8% among adults, and roughly half of these are recipient of antiretroviral therapy [1]. Nevertheless, the health status of HIV-infected individuals remains fraught with hazard since prevalence of cardiovascular disorders (CVDs), together with metabolic complications, are not only on the increase but encroach upon younger sufferers [2-5]. A cross-sectional design was applied to estimate the relationship between risk for CVD and insulin resistance among a Black African population (N=452, aged 30-74 years). Using the Framingham risk equations it was observed that absolute CVD risk was associated significantly with insulin resistance (47.3% were insulin resistant) whereas the DAD risk equation indicated a lower level [6,7]. HIV infection contributes also to endothelial dysfunction and inflammation/immune activation [8-11] with a consequently higher risk for CVD progression [12]. Large proportions of HIV-infected patients run the risk of developing not only CVD but also abnormal heart function with concomitant co-morbidity and mortality [13-15]. It has been indicated that in HIV-1 auxiliary protein nef-induced inhibition of autophagy, through compromise at the maturation stage of autophagosomes, the dysregulation of TFEB localization and cellular lysosome content, with consequent cytotoxicity leads to the death of [16]. The demonstration that the presence of Tat, impairing uptake of mitochondrial Ca<sup>2+</sup> ([Ca<sup>2+</sup>]<sub>m</sub>) and the electrophysiological activity of cardiomyocytes, in cardiomyocytes is accompanied by a decrease in oxidative phosphorylation, a decline in the levels of ATP, and an accumulation of reactive oxygen species further underlines the toxicity aspect of HIV co-morbidity [17]; loss of ubiquitin along with dysregulated degradation of autophagy proteins including SQSTM1/p62 and a reduction of LC3 II were detected in cardiomyocytes harboring Tat. HIV-infected individuals undergo acute myocardial infarction at lower age levels while remaining at higher risk for fatality during the month or so the follows discharge from hospital all of which confirms the CVD risk factor [18]. Among women infected with HIV in Europe and North America and presenting CVD the risk for myocardial infarct, stroke, and heart failure was elevated 2 to 4 fold [19]; this drastic increase may be taken in conjunction with the increased prevalence of neurobehavioral and psychosocial risk factors. A further risk factor for HIV patients is posed by the high levels of and the relatively unexplored presence of hypercholesterolemia among patients [20]. Among HIV patients in sub-Saharan Africa, heart failure appears to be associated with cardiopathology linked to coronary and pulmonary vasculature, the myocardium, valve parameters and conduction system in conjunction with autoimmune problems and presence of pro-inflammatory cytokines affecting a plethora of heart conditions including cardiomyopathy, fibrosis steatosis and pericardial diseases [21].

In conclusion, HIV-infected individuals are at risk for CVDs and several related conditions presenting serious health hazards. As indicated previously [22], certain lifestyle interventions ought to be considered in order to alleviate the threat to health.

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