

Left ventricular systolic dysfunction assessed by speckle tracking in asymptomatic HIV patients: prevalence and associations with clinical characteristics

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Background

Human immunodeficiency virus (HIV) primarily affects young, otherwise healthy, individuals. Cardiomyopathy in these persons has been attributed to the combined effect of inflammation, immune dysregulation, opportunistic infections, myocyte invasion and cardiac steatosis, while peripheral artery disease to immune activation, abnormalities in lipid metabolism associated with antiretrovirals, and increased prevalence of traditional risk factors. Pre-symptomatic diagnosis of myocardial dysfunction and peripheral artery damage could enable prompt and potentially more effective implementation of therapeutic measures. However, the data that are available to date on the specific topic are limited.

Methods

We investigated the association between global longitudinal strain (GLS), an established index of subclinical left ventricular systolic dysfunction (SLVSD), assessed by 2-D speckle tracking and a) patient history, b) demographic and clinical baseline characteristics, c) carotid intima-media thickness (IMT) and presence of carotid atheromatic plaque(s), measured by ultrasonography, d) temperature difference (ΔT) along each carotid artery, measured by microwave radiometry and e) basic blood panel measurements, including high-sensitivity troponin-T (hsTnT) and NT-proBNP in people living with HIV (PLWHIV) and no history of cardiovascular disease.

Explaining GLS

GLS has been recommended to follow patients at risk of myocardial dysfunction related to cancer chemotherapy.

It is being investigated in other patient populations (ie type 1 diabetics).

GLS is a very simple parameter that expresses longitudinal shortening as a percentage.

Peak GLS of -18.7% is defined as normal for a healthy person and the higher the value (ie closer to zero), the more likely it is for strain to be abnormal.

Positive correlations imply that the factor investigated is detrimental to myocardial function.

Results

We prospectively enrolled 103 consecutive PLWHIV. Ninety-eight individuals (95.1%) were male; mean age was 46.7 ± 11 years. **SLVSD was detected in 44% of PLWHIV.** Univariate analysis results are presented.

The value of GLS was significantly associated with body mass index (BMI, $r=0.345$, $P<0.001$), CD4/CD8 ratios ($r=0.206$, $P=0.036$), natural killer (NK) cell percentage counts ($r=-0.221$, $P=0.025$) and HDL cholesterol ($r=-0.195$, $P=0.048$).

hsTnT levels were significantly associated with age ($r=0.547$, $P<0.001$), serum creatinine ($r=0.336$, $P=0.001$), current CD4 counts ($r=-0.226$, $P=0.023$) and presence of carotid plaque ($r=0.302$, $P=0.004$).

Levels of NT-proBNP were significantly associated with age ($r=0.373$, $P<0.001$), history of diabetes ($r=0.277$, $P=0.007$), CD4/CD8 ratios ($r=-0.222$, $P=0.031$) and serum creatinine ($r=0.306$, $P=0.003$).



Variable	% (n/N) or Value \pm SD
Age, years	46.7 \pm 11
Male gender, %	95.1 (98/103)
Greek descent, %	89.3 (92/103)
Body mass index (BMI), kg/m ²	26.1 \pm 4.4
Waist circumference, cm	95.7 \pm 12.8
Smoking history	
Never smoked	28.2 (29/103)
Current smoker	45.6 (47/103)
Ex-smoker	16.5 (17/103)
Vaping/e-cig	9.7 (10/103)
Hypertension, %	23.3 (24/103)
Diabetes, %	5.8 (6/103)
Dyslipidemia, %	42.7 (44/103)
Chronic kidney disease, %	5.8 (6/103)
Thromboembolic disease, %	1.9 (2/103)
Log HIV RNA at diagnosis	4.97 \pm 0.97
Nadir CD4 cell count, c/mm ³	291 \pm 205
Current CD4 cell count, c/mm ³	688 \pm 333
Current CD4 cell percentage, %	33.8 \pm 10.6
Current CD4/CD8 ratio	0.93 \pm 0.44
Current natural killer cell percentage, %	12.3 \pm 6.7
GLS, %	-19.3 \pm 2.96
CRP, mg/dl	2.84 \pm 3.87
Creatinine, mg/dl	0.97 \pm 0.2
LDL, mg/dl	114.7 \pm 34.2
HDL, mg/dl	45.2 \pm 12
Troponin-T, pg/ml	6.25 \pm 4.78
NT-proBNP, pg/ml	44.6 \pm 57.6
Carotid ΔT , °C	0.55 \pm 0.24

Table 1: Baseline characteristics of study cohort

Conclusion

Our results indicate that apart from age, a dysmetabolic component, may be implicated in the pathogenesis of premature systolic myocardial dysfunction. The multiple intricate effects of immune dysregulation on myocardial function are not yet fully understood.