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Abstracts

HIV is associated with endothelial activation despite ART, in a sub-Saharan African setting

Objective To study the relationship between endothelial dysfunction, HIV infection, and stroke in Malawians.

Methods Using a cross-sectional design, we measured plasma levels of intercellular adhesion molecule-1 (ICAM-1), plasminogen activator inhibitor-1 (PAI-1), vascular endothelial growth factor (VEGF), and soluble thrombomodulin (sTM) in stroke patients and controls, stratified by HIV status. These biomarkers were measured using ELISA. After dichotomization, each biomarker was used as the dependent variable in a multivariable logistic regression model. Primary independent variables included HIV and stroke status. Adjustment variables were age, sex, hypertension, diabetes mellitus, tobacco and alcohol consumption, personal/family history of stroke, antiretroviral therapy status, and hypercholesterolemia.

Results Sixty-one stroke cases (19 HIV+) and 168 controls (32 HIV+) were enrolled. The median age was 55 years (38.5–65.0) for controls and 52 years (38.0–73.0) for cases ($p = 0.38$). The median $CD4^+$ T-cell count was 260.1 cells/mm^3 (156.3–363.9) and 452 cells/mm^3 (378.1–527.4) in HIV-infected cases and controls, respectively. HIV infection was independently associated with high levels of ICAM-1 (OR = 3.6, 95% CI 1.3–10.6, $p = 0.018$) in controls but not in stroke cases even after excluding patients with a viral load >1,000 RNA copies/mL (OR = 4.1, 95% CI 1.3–13.1, $p = 0.017$). There was no association between the clinical profiles of HIV-positive controls or HIV-positive stroke and high levels of PAI-1, VEGF, and sTM.

Conclusions HIV infection is associated with endothelial activation despite antiretroviral treatment. Our findings underscore the need for larger clinical cohorts to better understand the contribution of this perturbation of the endothelial function to the increasing burden of cardiovascular diseases in sub-Saharan Africa.

NPub.org/N2/9308a

Real-world validation of the 2017 McDonald criteria for pediatric MS

Objective To compare the diagnostic accuracy of the McDonald 2017 vs the McDonald 2010 criteria to predict a second attack of MS (clinically definite MS [CDMS]) at the first attack of acquired demyelinating syndromes (ADS).

Methods One hundred sixty-four children (aged <18 years) with an incident attack of ADS were included in a prospective multicenter study between June 2006 and December 2016. Brain (and spinal if available) MRI was performed ≤3 months after symptom onset. Sensitivity, specificity, positive predictive value, negative predictive value, and accuracy were compared at baseline between the 2010 and 2017 criteria.

Results Among the 164 patients, 110 patients (67%) presented without encephalopathy (ADS–, female 63%; median age 14.8 years, IQR 11.3–16.1 years) and 54 (33%) with encephalopathy (acute disseminated encephalomyelitis [ADEM], female 52%; median age 4.0 years, IQR 2.6–6.1 years). Of the 110 ADS– patients, 52 (47%) were diagnosed with CDMS within a median follow-up of 4.5 years (IQR 2.6–6.7 years). The sensitivity was higher for the 2017 criteria than for the 2010 criteria (83%; 95% CI 67–92, vs 49%; 95% CI 33–65; $p < 0.001$), but the specificity was lower (73%; 95% CI 59–84 vs 87%; 95% CI 74–94, $p = 0.02$). At baseline, 48 patients fulfilled the 2017 criteria compared with 27 patients when using the 2010 criteria. The results for children aged <12 years without encephalopathy were similar. In patients with ADEM, 8% fulfilled the 2010 criteria and 10% the 2017 criteria at baseline but no patient fulfilled the criteria for CDMS.

Conclusions The McDonald 2017 criteria are more sensitive than the McDonald 2010 criteria for predicting CDMS at baseline. These criteria can also be applied in children aged <12 years without encephalopathy but not in children with ADEM.

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