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Mitochondrial impairment in HIV-infected children

C Morén*[1], G Garrabou[1], E Molina[2], A Noguera[2], M Nicolàs[1], F Cardellach[1], C Fortuny[2] and Ò Miró[1]

Address: [1]Mitochondrial Research Laboratory-IDIBAPS-Hospital Clínic of Barcelona and CIBERER, Barcelona, Spain and [2]Hospital Sant Joan de Déu, Barcelona, Spain

* Corresponding author

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Purpose of the study

Mechanisms of mitochondrial impairment induced by HIV infection itself and highly active antiretroviral therapy (HAART) are well-studied in adults. However, there is more left to know in children, since they are the first following-up generation with the disease. The aim of this study is to determine whether there are alterations in mtDNA content and MRC dysfunctions in peripheral blood mononuclear cells (PBMCs) in HAART-treated and non-HAART-treated HIV-infected children.

Methods

A transversal study in PBMC, isolated by a Ficoll's gradient, was performed in 33 HAART-treated and 14 non-HAART-treated HIV-infected children as well as nine healthy children. MtDNA amount was assessed by Real-Time PCR and MRC complex IV (CIV) function and mitochondrial mass (MM, estimated by cytrate synthase (CS) enzymatic activity), were measured by spectrophotometry. CIV activity was expressed in absolute values, as nmols oxidated substrate/minute/mg protein, and relative values by dividing absolute CIV per MM (CIV/CS). Subunits COXII and COXIV of complex IV, as well as mitochondrial content, were assessed by western blotting.

Summary of results

No differences in mitochondrial parameters between HAART and non-HAART-treated HIV-infected patients were found. However, mtDNA significantly decreased (32%, p = 0.015) in both groups compared to healthy controls. Absolute and relative CIV activities did not differ among groups.

Conclusion

We found a reduction in mtDNA amount in HIV-infected children with respect to healthy controls. However, this depletion was not reflected in MRC CIV activity dysfunction. HAART does not seem to interfere with mitochondrial parameters. Future studies will be performed in order to determine whether this is caused by upregulatory mechanisms or longer time is required to detect alterations in MRC.