

13th Conference on Retroviruses and Opportunistic Infections



Denver, Colorado - February 5-8, 2006

INSULIN RESISTANCE, LIPODYSTROPHY AND ASSOCIATED METABOLIC CHANGES IN HIV-INFECTED CHILDREN

Conf Retrovir Opportunistic Infect 2006 Feb 5-8;13:abstract no. 23

Raffaella Rosso¹, A Parodi², A Di Biagio¹, L Di Stefano¹, C Torrasi², C Dentone¹, G D'Annunzio², C Viscoli³, and M Vignolo²

¹Univ of Genoa, San Martino Hosp, Italy; ²Univ of Genoa, G Gaslini Inst, Italy; and ³Univ of Genoa, Italy

BACKGROUND: Insulin resistance (IR) is accepted as the underlying fundamental defect that predates and ultimately leads to the development of type 2 diabetes mellitus in the general non-HIV-infected population. IR is also a major component of the metabolic syndrome that, in association with other factors—such as hypertension, hypercholesterolemia, and central obesity—defines a pre-diabetic atherogenic state that leads to adverse cardiovascular events. We estimate IR and its relationship to lipodystrophy and metabolic changes in a cohort of HIV-infected children with comparison to HIV-seronegative pediatric controls.

METHODS: Fasting plasma glucose (FPG), insulin (FPI), triglycerides (TG), cholesterol (CHO), IR (determined by homeostasis model assessment [HOMA-IR], HOMA of percentage of β -cell function [HOMA- β %], and quantitative insulin-sensitivity check index [QUICKI]), presence of clinical lipodystrophy, and detailed histories of ART were obtained for 47 infected (26 males, aged 4 to 23 years) and 98 uninfected children (66 males, aged 3 to 19 years).

RESULTS: Of the total, 36 HIV-infected children were receiving protease inhibitor (PI)- or non-nucleoside reverse transcriptase inhibitor (NNRTI)-based HAART, 8 were receiving only 2 NRTI, and 3 were naïve to any therapy. FPI/FPG ratio lower than 7, usually considered a cut-off value for IR (reduced insulin sensitivity), was present in 25 infected children. Associated lipodystrophy and insulin resistance occurred in 7 infected children, who also presented higher triglycerides with not significant increase in cholesterol levels. Height was slightly impaired (mean height SDS: -1.2) in subjects with lipodystrophy as compared with HIV-infected subjects without lipodystrophy (mean height SDS: -0.5).

		FPG (mmol/L)	FPI (mU/L)	HOMA- IR	QUICKI	HOMA- B%
patients	mean	4.31	13.48	2.55	0.63	382.42
	sd	0.43	8.38	1.61	0.15	390.02
controls	mean	4.64	8.13	1.68	0.69	174.00
	sd	0.38	4.79	1.01	0.15	165.80
	<i>p</i> values	n.s.	<i>P</i> <0.001	<i>P</i> <0.001	<i>P</i> <0.01	<i>P</i> <0.001

CONCLUSIONS: IR in HIV-infected children seems common and significantly different than in the control population. Fasting surrogate markers suggest increased IR as in the HIV-infected adults, which could be related to not only the cumulative ART exposure but the HIV infection itself. The association between type and duration of HAART and IR was not considered, due to the small group and the widely different type of therapy received by patients.

2006-02-05
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