

3rd International Workshop on Adverse Drug Reactions and Lipodystrophy in HIV



23-26 October 2001, Athens, Greece

DIFFERENT LACTATE METABOLISM IN PATIENTS WITH AND WITHOUT HIV-ASSOCIATED LIPODYSTROPHY SYNDROME

Antiviral Therapy 2001; 6(Suppl. 4):7 (abstract no. 9)

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BACKGROUND: Increased serum lactate level and impaired insulin action have been reported in HIV associated lipodystrophy syndrome (HALS). The level of serum lactate in HALS patients has been suggested to be associated with nucleoside reverse transcriptase inhibitor (NRTI) treatment.

OBJECTIVES: We examined the possibility of associations between the serum lactate level, the glucose metabolism and the duration of NRTI-treatment in HIV patients with and without lipodystrophy.

METHODS: Seventeen male patients with HALS (cases), and 16 male HIV patients with a normal distribution of fat (controls) were studied. All patients had received highly active antiretroviral therapy (HAART) during more than 12 months. The total glucose disposal rate was measured by an euglycemic hyperinsulinemic (40 mU/min/m²) clamp and separated into oxidative and non-oxidative glucose metabolism by indirect calorimetry.

RESULTS: The patients with HALS exhibited increased s-lactate levels compared with those of the controls [1.62±0.09 mM versus 1.37±0.12 mM (mean±SEM), P<0.05, excluding one HALS patient with a very high s-lactate value=4.4 mM]. Basal as well as insulin-stimulated glucose oxidation levels were similar in cases and controls [2.5±0.2 versus 2.3±0.2, P=0.53 and 3.4±0.2 versus 3.8±0.2 (unit: mg glucose/min/kg lean-body-mass), P=0.17], although the total glucose disposal rate was impaired in HALS patients (5.5±0.5 versus 8.0±0.5, P=0.003). However, despite an inverse

correlation between the values of s-lactate and total glucose disposal rates in controls ($r=-0.77$, $P=0.0003$), no such relation was found in patients with HALS ($r=-0.31$, $P=0.23$). The duration of NRTI-treatment (median 36 months, range 13-111) was not correlated to the s-lactate level ($n=33$, $r=0.02$, $P=0.89$).

CONCLUSIONS: The missing inverse correlation between total glucose disposal and s-lactate in patients with HALS, in contrast to such a finding in HIV patients without lipodystrophy, suggests a different lactate metabolism in cases versus controls. The similar oxidative glucose metabolism, found under various circumstances irrespectively of HALS, may be attributable to a sufficient capacity of oxidative metabolism by the mitochondrial pool. The notion that the duration of the NRTI therapy may influence the level of s-lactate in HIV-infected patients with or without lipodystrophy, is not supported by our data.

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9

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