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EVALUATION OF β -OXIDATION USING CARBON-11 ACETATE POSITRON EMISSION TOMOGRAPHY IN HIV-INFECTED PATIENTS WITH LIPODYSTROPHY SYNDROME

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BACKGROUND: Lipodystrophy in HIV-patients receiving highly active antiretroviral therapy (HAART) is frequently associated with abnormal lipid metabolism and increased free fatty acids (FFA). Mitochondrial toxicity induced by nucleoside-analogue reverse transcriptase inhibitors (NRTI) has been proposed to contribute to these side effects.

METHODS: We performed carbon-11 acetate positron emission tomography (PET) to assess the β -oxidative capacity of the skeletal muscle in HIV-patients with lipodystrophy after long-term NRTI-treatment compared with untreated HIV-patients. Simultaneously, the patients underwent indirect calorimetry to measure oxygen consumption and resting energy expenditure (REE).

RESULTS: Patients with lipodystrophy were insulin resistant, had an impaired insulin-mediated suppression of FFA and an increased REE. Lactate levels in lipodystrophic patients were significantly higher at basal and following oral glucose load. Similarly, FFA levels were elevated in these patients and correlated significantly to ketone bodies ($r=0.8$, $P=0.001$) suggesting impaired flux through the citric cycle. Using a two compartment, two rate-constant model we evaluated the mitochondrial β -oxidation in the skeletal muscle. Rate constants for inward and outward transport of acetate were comparable between both groups as were the local metabolic rates (LMR). We found a significant correlation between the oxygen consumption and the outward rate constant k_2 ($r=-0.64$, $P=0.025$) and LMR ($r=0.82$, $P=0.024$), respectively. This is in agreement with studies using carbon-11 acetate as a tracer of myocardial oxygen consumption.

CONCLUSIONS: This is the first study employing carbon-11 acetate PET to assess the mitochondrial β -oxidation in the skeletal muscle. We found no evidence for impaired β -oxidation in patients with long-term NRTI treatment and lipodystrophy despite metabolic changes that suggest mitochondrial dysfunction. Increased FFA levels in these patients appear to be a result of increased lipolysis rather than impaired oxidative metabolism.

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