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HEPATITIS B AND C CO-INFECTION AND ALANINE AMINOTRANSFERASE ARE ASSOCIATED WITH INCREASED INSULIN RESISTANCE AND DIABETES IN PATIENTS WITH FAT REDISTRIBUTION

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OBJECTIVES: There is growing evidence of increased insulin resistance and diabetes among patients with chronic liver infection. Therefore, we evaluated hepatocellular function and insulin resistance in patients with HIV-lipodystrophy, with and without viral hepatitis.

METHODS: Cross-sectional study of 91 HIV-infected men and women with fat redistribution without previous diagnosis of diabetes or symptoms of active hepatitis. Each subject completed a 2 h oral glucose challenge. Liver function tests, hepatitis B surface antigen (HBsAg), anti-hepatitis C virus (HCV) and HCV RNA by polymerase chain reaction (PCR) were also determined. HOMA-IR was calculated from fasting glucose and insulin as a measure of insulin resistance.

RESULTS: Five subjects were HBsAg(+) and nine were anti-HIV, HCV PCR(+). No subject was positive for both. Liver aminotransferase levels were not significantly different between groups, however HBsAg(+) subjects had increased fasting glucose, 2 h post-challenge glucose, and HOMA-IR compared to HCV and HIV controls without viral hepatitis (HOMA-IR: HBV 12.8 \pm 6.4, HCV 5.4 \pm 1.1, HIV control 4.0 \pm 0.4, $P < 0.05$). Subjects with HBV and HCV were significantly more likely to meet criteria for type II diabetes mellitus (HBV 60%, HCV 11%, HIV control 6.5%, $P = 0.01$). In a multivariate analysis including age, sex, BMI, protease inhibitor (PI) use, waist-to-hip ratio, hepatitis status and ALT, ALT ($P < 0.0001$) and hepatitis status ($P = 0.005$) were strong positive predictors of HOMA-IR. There was also a significant interaction between hepatitis status and ALT ($P < 0.0001$) and a significant effect of PI use ($P = 0.04$). In similar analyses, ALT

was a significant predictor for fasting glucose ($P=0.003$), 2 h glucose ($P=0.006$), fasting insulin ($P<0.0001$) and insulin area under the curve ($P=0.02$).

CONCLUSIONS: Insulin resistance in patients with fat redistribution is associated with ALT elevation, after adjusting for the presence of hepatitis. In addition, asymptomatic patients with HBV or HCV had significant insulin resistance and increased rates of diabetes. These data suggest that ALT elevations and, by inference, fatty liver disease correlate with extent of insulin resistance, and are exacerbated by chronic viral hepatitis. Careful screening and management of insulin resistance and diabetes among HIV-infected patients with fat redistribution and viral hepatitis co-infection is warranted.

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