Journal of the International AIDS Society

Poster presentation

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Impact of hyperglycemia and cholesterol levels on the outcome of hepatitis C treatment in HIV/HCV co-infected patients

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from Ninth International Congress on Drug Therapy in HIV Infection
Glasgow, UK. 9–13 November 2008

Published: 10 November 2008

Journal of the International AIDS Society 2008, **11**(Suppl 1):P274 doi:10.1186/1758-2652-11-S1-P274

This abstract is available from: http://www.jiasociety.org/content/11/S1/P274

Purpose of the study

A correlation between HCV infection and metabolic disorders, such as dyslipidemia and type II diabetes, has emerged. High serum cholesterol and LDL levels were demonstrated to enhance the probability of sustained viral response (SVR) of chronic hepatitis C. Conversely, insulin resistance impairs the SVR rates. Since HIV infection and antiretroviral therapies may also affect metabolic parameters, we investigated the influence of baseline glucose and lipid values on the outcome of HCV treatment in HIV-1 infected subjects.

Methods

We retrospectively reviewed the charts of the 140 HIV/HCV co-infected patients treated with an interferon-based regimen at our outpatient clinic from 2002 to 2007. Exclusion criteria were: alcohol abuse, HBV co-infection, CD4 cell count <200 cells/ml, concomitant hypocholesterolemic drugs, and suboptimal anti-HCV therapy. Fasting glucose levels, total cholesterol, LDL and tryglicerides levels were registered prior to treatment start.

Summary of results

Of the 55 patients enrolled in the study, 21 (38%) had genotype 1, 29 (52.7%) genotype 2 or 3, and five (9%) genotype 4. SVR was obtained in 29% (6/21) and 65% (22/34) in genotype 1 and 'other than 1', respectively. Hyperglycemia or diabetes was present at baseline in 15/27 (56%) non-responders but only in 4/28 (14%) of responders. In the multivariate analysis, the independent predictors of SVR were: genotype other than 1 [adjusted odds ratio (AOR) 5.45 (1.25–23.7), p 0.024], fasting glucose > 100 mg/dL [AOR 0.11 (0.024–0.528), p 0.006], and cholesterol levels > 200 mg/dL [AOR 8.37 (1.12–62.31), p 0.038]. Moreover, 34/51 (67%) patients of our cohort were defined lipodystrophic, mostly (27/51, 53%) showing peripheral lipoatrophy without significant relation at univariate analysis with anti-HCV therapy response. (Table 1.)

Conclusion

In conclusion, our study identified in a HIV/HCV co-infected population, hyperglycemia, serum cholesterol and, at a lesser extent, LDL as predictors of response to peg-IFN plus ribavirin therapy as previously reported in HCV-infected subjects. A possible explanation is that the entry of HCV into the cell is mediated by LDL-receptor that could be down-regulated by high levels of total and LDL-cholesterol, resulting in reduced HCV infectivity. HIV/HCV co-infected patients display a complex pattern of metabolic alterations in which viral features, host characteristics and drug toxicity interact and influence each other; prospective studies will better address this important issue and the underlying mechanisms.

Table 1:

Baseline characteristics	Overall (n = 55)	SVR (n = 28)	no SVR (n = 27)	p	AOR	95% CI	p
Age (years; SD)	43; 3.9	42; 4.3	44; 3.2	0.072	0.948	0.798–1.127	0.546
Sex (male)	46 (84%)	23 (82%)	23 (85%)	0.763			
Patients on HAART	49 (89%)	24 (85%)	25 (92%)	0.421			
Baseline CD4 cells/μL; SD	540; 298	536; 228	540; 356	0.184			
HIV-RNA cp/mL	<50	<50	<50				
HCV genotype							
1	21 (38%)	6 (21%)	15 (56%)	reference	reference	reference	reference
not 1	34 (62%)	22 (79%)	12 (44%)	0.019	5.457	1.256–23.721	0.024
HCV viremia (Log IU/mL); SD	5.87; 0.7	5.67; 0.8	5.96; 0.5	0.102			
ALT (IU/L); SD	100; 60	104; 62	90; 57	0.261			
BMI; SD	23.4; 2.2	23.6; 2	22.8; 3.2	0.460			
Hyperglicemia or diabetes	19 (34%)	4 (14%)	15 (55%)	0.001	0.113	0.024–0.528	0.006
Cholesterol >200 mg/dL	10 (18%)	8 (28%)	2 (7%)	0.078	8.373	1.125–62.316	0.038
LDL >100 mg/dL	15 (27%)	10 (36%)	5 (18%)	0.062			
Lipoatrophy	29 (53%)	13 (46%)	16 (59%)	0.455			