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Poster Session VI
Viral hepatitis and HIV/HCV co-infection

FACTORS ASSOCIATED WITH HCV-RNA REBOUND IN HIV/HCV-COINFECTED PATIENTS AFTER INITIAL RESPONSE TO TRIPLE THERAPY WITH HCV-PROTEASE INHIBITORS

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Objective and Methods: to determinate factors associated to HCV-RNA rebound in 42 consecutive HIV/HCV-coinfected subjects with at least 12 weeks of triple therapy with BOC (n=2) or TPV (n=40) in clinical practice.

Results: the mean time on therapy to date is 24 weeks (1-48), with therapy completed in 37 (88%), and a rate of SVR of 22% (7/32 patients with complete follow-up data). Mean age 49±5, 76% male, 81% previously treated (n=34): null responders 26% (n=9), relapsers 20% (n=7), partial responders 44% (n=15), HCV-G1a 55% (n=23), IL28B CC 36% (n=15). Cirrhosis 90% (n=38), CHILD PUGH >6 55% (n=21), MELD 9±2. Baseline HCV-RNA 6,14±0,52 $\log_{10}$ IU/ml. The overall rate of negative HCV-RNA during triple therapy was 71% (n=30). HCV-RNA rebound was observed in 13 subjects (43%), before completing 48 weeks of peg-IFN/RBV in 69% (n=9). By univariate analysis, factors associated with HCV-RNA rebound were prior therapy (52% vs 14% among naive, p=0.10), pattern of previous response (14% in relapsers vs 69% among the remaining, p=0.027), IL28B genotype (29% in CC vs 56% among non-CC, p=0.13), and CHILD-PUGH score (77% if CP >6 vs 20% among subjects with CP≤5, p=0.003). Neither age, gender, peg-IFN-α2a vs peg-IFN-α2b, RBV dose, baseline HCV-RNA, early HCV-RNA kinetics, RVR, cirrhosis, baseline MELD, nor HCV subtype (56% in G1a vs 27% in G1b, p=0.25) showed to have an impact on HCV-RNA rebound.

Conclusions: in HIV/HCV-coinfected subjects with histological severity and mostly pretreated, 71% reached negative HCV-RNA while on PI-based triple therapy, but HCV-RNA rebound was 43%, before completing the 48-weeks of therapy in 69%. Naïve patients and those with a better CHILD-PUGH showed less rates of viral rebound, whereas among pretreated subjects, HCV-RNA rebound was related to sensitivity to peg-IFN (prior response and IL28B genotype).