HCV Treatment with Pegylated Interferon and Ribavirin in Patients with Haemophilia and HIV/HCV Coinfection: A Retrospective Review

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Background: Adult patients with haemophilia are frequently coinfected with HIV and HCV, usually due to blood and plasma factor transfusion prior to effective screening programs. The current standard therapy for HCV is pegylated interferon and ribavirin (PEG + RBV), which has been investigated in HCV monoinfection and HIV/HCV coinfection. Little data exists regarding the effectiveness of HCV therapy in coinfected patients with haemophilia. We reviewed HCV treatment outcomes in our cohort of patients with haemophilia and HIV/HCV coinfection.

Methods: We performed a retrospective review of all patients with haemophilia and HIV/HCV co-infection treated with PEG + RBV at the Alfred Hospital between 2001—2007 ($n = 13$). Patient histories and medical records were reviewed for demographics, adverse events and effectiveness, including early, end of treatment and sustained virological response (EVR, EOTR and SVR, defined as undetectable HCV viral load/PCR at 12, 48 and 72 weeks respectively) of HCV

Results: All patients were male with haemophilia A, and a median age of 43 (range 27—62) at initiation of HCV therapy. Nine of thirteen (69%) patients had genotype (gt1) 1 HCV (gt3 = 3, gt4 = 1). Due to bleeding tendencies, only 1/13 patients underwent liver biopsy prior to HCV therapy. Twelve of 13 (92%) were receiving ART, with a mean CD4 count of 428 cells/uL (range 175—928 cells/μL) at initiation of HCV therapy. Six of 11 (55%) patients achieved EVR (3 × gt1, 2 × gt3) at 12 weeks, 4/13 (31%) had EOTR (2 × gt1, 2 × gt3) and 1/13 (8%) achieved SVR (gt1). 7/11 (64%) patients normalised ALT during therapy wherein mean ALT fell from 101 to 76 U/L. Only 1/13 (8%) patients prematurely stopped therapy prematurely due to side effects. CD4 cell counts and HIV viral load remained stable during HCV treatment, with a mean 437 cells/uL and <50 copies/mL at 48 weeks, respectively

Conclusions: Treatment responses in patients with haemophilia and HIV/HCV coinfection were significantly lower from that reported in other coinfected populations. This is despite a low drop out rate and suggests that haemophilia is an independent risk factor for poor treatment response. Risk factors in this cohort for poor response may include long duration of coinfection, advanced liver disease and unfavourable genotype profile. Prospective studies in this patient population are needed to further evaluate HCV treatment efficacy.

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Continuing Evidence Supporting the Role of Early Kinetic Monitoring (RVR) in Predicting SVR for HIV/HCV Coinfected Patients

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Introduction: Hepatitis C virus (HCV) is a major cause of cirrhosis and need for liver transplant. With common risk factors for acquisition, HIV co-infection is common, and leads to faster progression of liver fibrosis. We have previously reported that combination therapy for HCV can result in comparable sustained virological response (SVR) rates for co-infected patients when compared to Hep C monoinfected patients. Monitoring viral kinetics, specifically baseline HCV viral load and week 4 HCV PCR (rapid virological response (RVR)) can allow tailoring of treatment duration for specific patients and give prognostic information regarding likelihood of primary treatment success.

Methods: Data for 80 HIV-HCV co-infected patients treated with pegylated-interferon and ribavirin between 2001 and 2007 was collected. Genotype 2/3 infected patients were treated for 24 weeks only with weight-based ribavirin. Baseline characteristics and viral kinetics were analysed using Excel and Epi info

Results: 80 co-infected patients were treated, 84% male. 45% patients had Genotype 1 disease. Overall SVR rate was 53% using an intention-to-treat (ITT) analysis. Baseline HCV viral load was noted to be lower in those who achieved a SVR (6×10^6 iu vs 13×10^6 iu) ($p < 0.05$). In ITT analysis 33% achieved a RVR response. These patients were significantly more likely to have a SVR than those without a RVR (96% vs 29%) ($p < 0.05$). Using an on-treatment analysis 100% of patients achieving RVR achieved SVR. Predictors of achieving RVR were HCV viral load at baseline ($p < 0.05$) and HCV genotype ($p < 0.05$).

Conclusion: Monitoring of early viral kinetics can be used to accurately predict those patients who will achieve SVR, especially those for whom only 24 weeks of therapy is required.

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