HIV/hepatitis C co-infection

Table 1. Baseline characteristics

Age, mean (SD)	50.9 (5.5)
Gender: male/female, n (%)	93 (71.5)/37 (28.5)
Risk group: PWID/heterosexual/MSM, n (%)	113 (86.9)/13 (10.0)/4 (3.1)
CDC (93) classification system's C stage, n (%)	48 (36.9)
Basal HCV RNA, medIAn (UI/mL) (IQR)	2,047,181.5 (3,879,143.0)
Prior INF-based therapy: none/failure/relapse/intolerance, n (%)	79 (60.8)/35 (26.9)/8 (6.2)/8 (6.2)
Liver fibrosis assessed by transient elastography: F0–1, F2, F3, F4, n (%)	1 (0.8)/54 (41.5)/14 (10.8)/61 (46.9)

MSM, men who have sex with men; PWID, people who inject drugs.

before, during and after HCV therapy. Table 1 shows baseline characteristics. SVR was achieved in 127 patients (97.7%). TC and LDL-c values statistically increased on and after treatment ($p < 0.001$) versus pre-treatment. There were no significant changes when comparing TC and LDL-c values on versus after-treatment, nor between TG and HDL-c values pre-treatment versus on-treatment or post-treatment (Table 2). Changes in TC and LDL-c values are not influenced by gender ($p = 0.55$ and $p = 0.86$, respectively), age ($p = 0.07$ and $p = 0.06$), basal HCV RNA ($p = 0.21$ and $p = 0.1$), presence of PI in the ART regimen ($p = 0.50$ and $p = 0.46$) nor cirrhosis ($p = 0.41$ and $p = 0.19$). Moreover, changes between LDL-c values are not influenced by the presence of PI in the DAA regimen ($p = 0.18$), but DAA regimens including a PI were associated with increased TC values ($p = 0.005$).

Conclusion: TC and LDL-c values increase during the HCV treatment using DAA, INF-free regimens, and remain increased after stopping the HCV therapy.

References

1. Cooper CL, Mills E, Angel JB. Mitigation of antiretroviral-induced hyperlipidemia by hepatitis C virus co-infection. AIDS. 2007;21:71–6. doi: http://dx.doi.org/10.1097/QAD.0b013e3280110ada
2. Lange CM, von-Wagner M, Bojunga J, Berg T, Farnik H, Hassler A, et al. Serum lipids in European chronic HCV genotype 1 patients during and after treatment with pegylated interferon-a-2a and ribavirin. Eur J Gastroenterol Hepatol. 2010;22:1303–7. doi: http://dx.doi.org/10.1097/MEG.0b013e32833de92c

P272

HIV/hepatitis C co-infection: successfully treating hepatitis C with direct-acting antivirals and managing those who do not access traditional care

Rebecca Metcalfe[1]; Naomi Bulteel[1]; Joe Schofield[2]; Patricia McGinness[1]; Claire Glover[1]; Lorna McLean[1]; Fiona Marra[1]; Aliceann Murphy[1]; Erica Peters[1]; David Bell[1]; Ray Fox[1] and Emma Thomson[1]
[1]Brownlee Centre, NHS Greater Glasgow and Clyde, Glasgow, UK.
[2]Public Health, NHS Greater Glasgow and Clyde, Glasgow, UK

Introduction: In our HIV/hepatitis C virus (HCV) co-infected cohort, we are successfully treating HCV with direct-acting antivirals (DAAs) regardless of genotype, regimen, disease stage or prior treatment exposure. However, we recognize a proportion of patients for whom we are unable to provide treatment, because they do not engage in the traditional care setting. We report on the efficacy and safety of DAA therapy in our cohort of HIV/HCV co-infected individuals, the demographics of those not engaging in care and the strategies employed to tackle this population.

Methods: All patients co-infected with HIV and HCV in our cohort were included, and case notes were reviewed. Those who spontaneously cleared HCV infection, transferred care or died were excluded.

Results: At May 2016, the HIV/HCV co-infected cohort comprised 181 patients, of whom 89 (49%) had commenced HCV treatment. Thirty-three of these patients were treated successfully with interferon and ribavirin. Fifty-seven patients received ≥ 1 dose second-generation DAA, including 20 patients with cirrhosis, six in clinical trials. The majority were male (46/57) with a history of injecting drug use (35/57). The majority were HCV genotype 1 infected (48/57). Most were treatment naïve (43/57); six prior null responders; four relapsers after previous IFN/RBV; none were DAA experienced. Fifty-five of 57 were on a suppressive HIV antiretroviral regimen. At the time of writing, 52/57 patients had reached end of treatment. Forty-two had achieved SVR12 (42/42, 100%). Despite high success rates with those engaged in care, 92 (51%) patients remain untreated, of whom the majority are not attending scheduled hospital appointments, and many are currently struggling with addictions. Some are recently diagnosed as part of an ongoing outbreak of HIV and HCV amongst people who inject drugs. To target this population, we are implementing service change. New strategies will include local pharmacy ''directly observed therapy'' dispensing, specialist nurse-led service in the community and in addiction services. We show the area of residence of those who have over 50% non-attendance rates, in relation to the hospital where care is traditionally delivered to highlight the need for local services.

Conclusions: In those who access care, we observe excellent SVR rates in HIV-infected patients receiving DAAs for HCV. Serious adverse events with DAAs are rare and delivering treatment in the community to difficult-to-treat populations will increase engagement in HIV care and HCV cure rates. Poor engagement in care should be tackled by service redesign to reach out to these populations.

P273

Improving of glycaemic control associated with DAAs HCV treatment persists at SVR12

Abstract P271–Table 2. Means of the values in mg/dL with standard deviation

	Pre-treatment	On-treatment	After-treatment	p*	p†	p‡
TG	156.4 (79.7)	142.7 (68.1)	161.5 (85.2)	0.073	1.000	0.011
TC	176.0 (37.9)	199.5 (55.0)	196.1 (51.9)	<0.001	<0.001	1.000
HDL-c	49.3 (20.5)	52.0 (23.4)	49.9 (18.4)	0.100	1.000	0.751
LDL-c	97.6 (37.1)	121.1 (46.8)	114.2 (44.7)	<0.001	<0.001	0.107

Statistical significance p-value established in 0.05. Bonferroni correction has been done. *compares values pre-therapy versus on-treatment; †compares pre-therapy versus after-treatment; ‡compares on-treatment versus after-treatment.
TG, triglycerides; TC, total cholesterol; HDL-c, HDL cholesterol; LDL-c, LDL cholesterol.