



## Letter to the Editor

# Treatment of genotype one hepatitis C virus (HCV) infection in HIV co-infected patients using telaprevir based regimen



Dear Editor,

Over 4 million individuals in the United States and over 170 million worldwide are living with hepatitis C virus (HCV). Among HIV infected patients, the estimated HCV co-infection rate can be as high as 25%.<sup>1</sup>

Prior to the approval of the direct-acting antivirals (DAA), HCV treatment in HCV co-infected patients with pegylated interferon and ribavirin (PIFN/RBV) had very low sustained virological response (SVR) rates ranging from 7 to 29%.<sup>2,3</sup> The recently approved DAA-based HCV regimens have markedly improved the SVR rate in HIV co-infected patients.<sup>4,5</sup>

Telaprevir is an oral DAA protease inhibitor (PI) approved for treatment-naïve and treatment-experienced patients with HCV genotype 1 infection.

At East Carolina University Infectious Diseases Clinic, once DAA were approved for monoinfected HCV patients, all genotype 1 HIV co-infected patients were started on telaprevir-based regimen for HCV. Here, we present the efficacy, safety and tolerability of telaprevir-based therapy for HCV genotype 1 in 12 patients.

Twelve HIV/HCV-1 co-infected patients have been treated with telaprevir-based therapy for HCV. Eight patients (66.7%) were African-American. Eleven patients (92%) were on HAART. One patient was long-term non-progressor. In all except one, HIV therapy was switched to a regimen of tenofovir-emtricitabine and raltegravir. All but one patient (92%) were treatment-naïve for HCV therapy. Seven patients (58%) had HCV genotype 1a, three (25%) had genotype 1b, and two did not have subtype identification. Eight patients (66.7%) had a viral load greater than 800,000 IU/mL.

At week 4 of therapy, 12 patients (100%) achieved HCV viral load <25 IU/mL. At week 12, 10 patients had viral load measurement and of those 90% (9/10) had undetectable HCV viral load. Three patients stopped therapy at weeks 5, 7, and 34, respectively, because of side-effects (depression and severe pancytopenia). One patient who received therapy for only five weeks eventually attained SVR. Eight of the twelve patients (66.7%) attained SVR six months after completing therapy. There was only one case of viral breakthrough while on therapy. The remaining three patients had undetectable viral load when they stopped therapy either because of noncompliance

(one patient) or side-effects (two patients). All three eventually failed therapy.

The most common side-effects were anemia (100%), thrombocytopenia (100%), neutropenia (58.3%), and skin rash and itching (33.3%). Despite the need for dose adjustment, only three patients had to discontinue therapy due to side-effects.

HIV viral load remained undetectable throughout the study in all HIV infected patients who were on therapy. There was a drop of CD4 count in all patients, but CD4 count remained above 200 cells/mm<sup>3</sup> and the drop was not clinically significant. Ten patients were switched to a raltegravir-based regimen and all tolerated the regimen well.

HCV progresses faster in HIV co-infected patients and has become a prominent cause of morbidity and mortality in co-infected patients. Therapy for HCV genotype 1 with just PIFN/RBV has been disappointing, with low SVR rates and thus few patients were willing to consider therapy.<sup>3</sup>

In this study, out of 12 patients seven had HCV genotype 1a, eight were African American, and eight (66.7%) had high viral loads. Despite multiple negative predictors, 8/12 (66.7%) attained SVR and there was only one viral breakthrough while on therapy. The results were comparable to the 74% SVR rate in recently presented data.<sup>4</sup> Three other patients failed therapy due to early drug discontinuation secondary to non-compliance or side-effects.

As predicted by the data from the monoinfected HCV studies, anemia was the most common side-effect. Recently, FDA has approved 12-week DAA-based therapy for HIV/HCV co-infection, and the recently updated AASLD/IDSA guideline on HCV therapy also endorsed this short course regimen.<sup>5,6</sup> Our data, which showed the early clearance of HCV virus during therapy, concur with these recommendations.

Ten out of 11 patients (91%) who were on HAART were switched to a raltegravir-tenofovir-emtricitabine regimen prior to starting HCV therapy. HIV viral load remained undetectable in all patients throughout their HCV therapy.

In conclusion, the addition of DAAs to the treatment of HCV genotype 1 among HIV/HCV co-infected patients has shown a remarkable improvement in efficacy without compromising safety or compliance, nor increasing morbidity or mortality. Our study is additional evidence to the growing body of literature that shows comparable SVR rates between

HIV co-infected and HCV monoinfected patients when treated with DAA-based regimens. Recently approved and ongoing studies on HCV therapy will likely build on this already achieved SVR rates while shortening treatment duration and improving adherence.

### Conflicts of interest

Dawd S. Siraj is member of the speaker's bureau for Gilead Sciences hepatitis branch and ViiV HIV branch and has received honorarium for speaking.

### REFERENCES

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