

Care of Patients with Chronic HCV/HIV Co-Infections



AETC Capitol Region Telehealth Project

Howard University College of Medicine

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Clinical Case Summary:

This is a case of a woman with a long history of illegal substance abuse who was diagnosed to be HIV/HCV co-infected. Due to her substance abuse and her involvement with the penal system, she was initially unable to receive continuous care which may have improved her health outcomes. However, she was able to successfully become rehabilitated from her illegal substance abuse and working together with an experienced medical care team, she was able to place herself in a position of HIV infection control and is currently actively involved in having SVR (“cure”) for HCV infection.

Numerous studies have shown an acceleration of fibrosis in HIV/HCV co-infected patients compared with HCV mono-infected patients. HIV immunosuppression plays a role. There is now very good evidence that being on HIV treatment with virologic suppression slows progression of fibrosis. In a paper published by Johns Hopkins University, there was a 66% decrease in risk of liver death in patients suppressed on antiretroviral therapy. Therefore we know that it is important to treat the HIV component of those co-infected with HIV/HCV.

This case sheds light on many of the issues which must be considered when identifying a successful HIV treatment regimen while it highlights several aspects unique to the treatment of chronic HCV infection in HIV/HCV co-infected patients. Firstly, there is little research and clinical information on the long term outcomes of treating HCV in HIV/HCV co-infected individuals. Also that the HCV and HIV clinicians need to be aware of potential interactions between antiretroviral therapy and HCV therapy. Therefore, a thorough evaluation, careful approach, and careful monitoring of the patient is required for the best chance at success.

Clinical Case:

Aletha is a 47-year-old black woman who was diagnosed with HIV infection at age 39 when she began methadone rehabilitation from years of daily use of intravenous heroin. At the time of her diagnosis, her CD4+ cell count was 290 cells/mm³ (12%), and her HIV viral load was 120,470 copies/mL. An HIV genotyping assay obtained a short time after her diagnosis showed only wild type HIV and a few months after she entered rehab, she was started on zidovudine, lamivudine, and ritonavir-boosted lopinavir with good results (viral load < 400 copies/mL).

After two years of treatment, she relapsed to using heroin and dropped completely out of care.

After an absence of approximately 2 years, as a part of a court ordered program, the patient returned to substance use rehabilitation and HIV care. Laboratory studies at her reentry of care revealed her CD4+ cell count was 244 cells/mm³ (15%), and her HIV viral load was 107,800 copies/mL. Also, the patient was identified as HCV antibody positive with a mildly elevated serum liver panel. No HIV genotyping or

phenotype assay was obtained. At that time she was switched from zidovudine plus lamivudine to tenofovir plus emtricitabine while continuing ritonavir-boosted lopinavir as HIV therapy and was referred to a Hepatitis disease specialist for evaluation of her HCV infection.

Her HCV related evaluation revealed that she is infected with genotype 1a HCV and her IL28B haplotype is CT. At that time her HCV RNA was 4 million IU/mL. The patient was informed of the new therapies for treatment and possible cure of HCV infection which were available at that time and after multiple discussions, she agreed to a liver biopsy in preparation for HCV treatment. Liver biopsy results in August 2011 showed a Metavir Score of F2, A1 (portal fibrosis with mild inflammation).

The patient's total time in care was approximately 5 months due to her enrollment into the penal system in a nearby state. During her brief time in care she was not able to reach a non-detected HIV-1 viral load and she did not receive HCV related therapy.

In February 2013, the patient returned to care. She had been without alcohol or illegal substance use for greater than one year. Laboratory studies at her reentry of care revealed her CD4+ cell count was 201 cells/mm³ (11%), and her HIV viral load was 127,450 copies/mL. Her HCV RNA was 5 million IU/mL. Albumin: 1.8 g/L. Total bilirubin: 1.0 mg/dL. Alanine aminotransferase: 50 U/L. Aspartate aminotransferase: 52 U/L. Blood urea nitrogen: 17 mg/dL. Creatinine: .9 mg/dL. And a calculated Creatinine clearance of 77 ml/min. An HIV genotyping assay obtained a short time after her return showed no HIV therapy related mutations. She was started on a regimen of Elvitegravir/cobicistat tenofovir/emtricitabine for her HIV infection and reached an HIV viral load <50 copies/mL within 4 months. However, by agreement of the patient and the Hepatitis specialist, HCV treatment was withheld.

In March 2014, the Hepatitis specialist began discussions with the patient concerning new HCV therapies with improved outcomes which were likely to be available in the near future. With the patient's approval, the specialist began research into possible HCV therapies appropriate for the patient.

In July 2014, the Hepatitis specialist recommended a HCV treatment regimen for the patient. Although the patient had history of good adherence to her HIV related regimen with a non-detected HIV-1 viral load for more than one year, in consultation with the HIV specialist and patient, her once daily Elvitegravir/cobicistat tenofovir/emtricitabine regimen was discontinued and she was switched to tenofovir/emtricitabine plus raltegravir. She has tolerated the combination therapy well.

In December 2014, with her laboratory study results of CD4+ cell count 467 cells/mm³ (16%). HIV-1 viral load <50 copies/mL. HCV RNA 8 million IU/mL. Albumin: 2.8 g/L. Total bilirubin: 1.0 mg/dL. Alanine aminotransferase: 55 U/L. Aspartate aminotransferase: 62 U/L. Blood urea nitrogen: 15 mg/dL. Creatinine: .7 mg/dL. and Calculated Creatinine clearance of 72; through a special program supported by a local academic medical institution and a federal medical agency, she was started on a HCV regimen of 90 mg ledipasvir and 400 mg sofosbuvir orally once daily.

The most recent follow up with the patient's medical care team reveals her 8 week and 12 week HCV RNA to be <25 IU/mL (lower limit of quantification (LLOQ)). Her HIV-1 viral load remains <50 copies/mL. Her creatinine clearance remains above 60 ml/min. Her main side effect complaints have been occasional episodes of fatigue and nausea. However, the patient and her treatment team have agreed to a course duration of a total of at least 24 weeks of HCV therapy.