Clinical Outcomes of Hepatitis C Treatment in a Prison Setting: Feasibility and Effectiveness for Challenging Treatment Populations

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Background. More than one-third of people in the United States with hepatic C virus (HCV) infection pass through the correctional system annually. Data are lacking on outcomes of treatment with pegylated interferon plus ribavirin (PEG-RBV) in correctional settings.

Methods. During 2002–2006, we analyzed patients in the Connecticut Department of Correction who received PEG-RBV. We assessed the rates of sustained virological response, hospitalization, and use of medications to treat psychiatric disorders and anemia.

Results. Of 138 treatment-naive patients referred for treatment, 68 (49%) were approved. Overall, sustained virological response occurred in 47.1% of patients (for HCV genotype 1, 43.1%; for HCV genotypes 2 and 3, 58.8%). Only 9 patients (13%) discontinued treatment because of adverse effects. Multiple regression analysis revealed that not achieving a sustained virological response was correlated with HCV genotype 1 infection plus cirrhosis (adjusted odds ratio, 12.9; 95% confidence interval, 1.1–148) and baseline major depression (adjusted odds ratio, 3.4; 95% confidence interval, 1.01–11.6), but not with HIV infection, a baseline HCV RNA level ⩾400,000 IU/mL, or black race. Compared with baseline, the rate of prescription of a new mood stabilizer (2.2 vs. 0.8 prescriptions per person-year) or an opioid (1.8 vs. 0.5 prescriptions per person-year) was higher during treatment, whereas there was no change in the rate of prescription of benzodiazepines and antipsychotic medications.

Conclusions. These results support the feasibility and clinical effectiveness of PEG-RBV for the treatment of chronic HCV infection in correctional facilities.

Hepatitis C virus (HCV) is the most prevalent chronic, bloodborne infection in the United States, affecting >4 million people. HCV infection causes ~10,000 deaths per year [1], yielding direct health care costs that exceed $1 billion annually [2]. Nowhere is the burden of HCV infection greater than in the nation's correctional facilities [3]. Seroprevalences of chronic HCV infection in correctional facilities range from 16% to 49% [4–7], and it is estimated that more than one-third of all patients with chronic HCV infection in the United States pass through correctional facilities each year [4].

Prisons—and the correctional system in general—are among the cornerstones of public health in the United States [8–12]. By 2007, 7 million people in the United States lived under the jurisdiction of the criminal justice system [13], and 2.5 million individuals (or 1 in 100) were in jail or prison [14]. The millions of intermittently incarcerated people in America, many of whom have substance use disorders and profound psychiatric comorbidities, are among the most difficult people to reach with critical health information and treatment in community settings [3, 9, 15].

Improved diagnosis and access to medical care and prevention services for incarcerated populations, recognized as a public health disease-control strategy [13–15], can benefit communities by reducing disease transmission and medical costs [9, 16–18]. Inmates who participate in health-related programs while incarcerated have lower recidivism rates and are more likely to maintain health-conscious behavior [17]. Because the prevalence of HCV infection is high among incarcerated persons, community efforts to diagnose, treat, and pre-

Received 17 April 2008; accepted 6 June 2008; electronically published 20 August 2008.

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Clinical Infectious Diseases 2008; 47:952–61

1058-4838/2008/4707-0017$15.00
DOI: 10.1086/591707

vent these infections require inclusion of the correctional population [19].

Antiviral treatment with combination pegylated IFN and ribavirin (PEG-RBV) is now the preferred treatment for HCV infection and is a crucial component of the public health response to the epidemic [20–22]. This strategy can result in achievement of a sustained virological response (SVR; defined as a nondetectable HCV load 6 months after completion of treatment) in >50% of treated subjects [23, 24]. Patients who achieve SVR experience slower rates of progression of liver disease.

Unfortunately, adverse effects, difficulties with adherence to treatment, comorbid psychiatric and substance use disorders, and lack of access to clinicians experienced in the treatment of HCV infection have limited the full expansion of this strategy [25, 26]. In community settings, patients with alcohol dependence, other drug dependence, or mental illness typically do not receive treatment [27–34].

Although the feasibility of treating HCV-infected prisoners with standard thrice-weekly IFN has been demonstrated [35–37], there are, to our knowledge, no published reports on the efficacy of the contemporary and more efficacious therapy with PEG-RBV and on treatment outcomes among prisoners who receive PEG-RBV. The purpose of this retrospective, longitudinal study is to address the feasibility and effectiveness of use of PEG-RBV treatment in the correctional setting. Data from the Connecticut Department of Correction (CTDOC) HCV Management Program were analyzed using both primary outcomes (intention-to-treat SVR) and secondary outcomes (determinants of SVR and the use of additional pharmaceutical agents and hospitalizations) during treatment.

METHODS

Treatment setting. The CTDOC houses 22,000 inmates in 20 correctional facilities. Anonymous serosurveys that had been conducted previously demonstrated that the prevalence of chronic HCV infection is ~33% [38]; this is similar to the prevalence of HCV infection among incarcerated populations in the United States reported from other state surveys [4–7]. The University of Connecticut's Correctional Managed Health Care (CMHC) provides medical care to CTDOC inmates. The Yale University School of Medicine (New Haven, CT) provides infectious disease consultation services, including consultation regarding the management of HIV infection, HCV infection, and tuberculosis. A Hepatitis C Utilization Review Board (HepCURB), established in 2002, monitors the program.

HCV management protocol. There is no systematic policy for screening for HCV in the CTDOC. Most inmates with HCV infection come to medical attention through self-report, clinical indication, or routine screening for chronic hepatitis among HIV-infected patients. All HCV-infected prisoners are screened for hepatitis A and B viruses and are vaccinated if indicated. The primary care provider is responsible for discussing diagnosis, treatment options, prevention, and referrals to an infectious diseases specialist only after the inmate has evidence of persistently elevated transaminase levels for ⩾6 months. Persons who initially qualify are referred to the infectious diseases specialist for further evaluation.

Treatment eligibility. The treatment eligibility criteria established by the HepCURB in 2002 are listed in table 1. The infectious diseases specialist initiates a full evaluation for hepatitis and for HCV treatment, including laboratory testing and liver biopsy for HCV genotypes 1 and 4. HIV-HCV–coinfected patients are eligible for treatment. To receive treatment, inmates must agree to be housed in one of the correctional facilities where 24-h nursing is available and to waive parole eligibility until after HCV treatment has been completed. In instances in which the infectious diseases specialist does not favor treatment, the inmate may independently request referral to the HepCURB for an external decision.

Modified hepatic activity index necroinflammatory scores and Ishak fibrosis scores are used by the HepCURB in their interpretation of hepatic biopsy findings [39–41]. Typically, biopsy specimens with fibrosis scores ⩽1 and inflammation scores ⩽4 are considered to be normal. All patients who are eligible for and agreeable to treatment provide written, informed consent before initiating treatment. Patients are assessed by a psychiatrist, and HCV care is coordinated with a

Table 1. Eligibility criteria for treatment of hepatitis C virus (HCV) infection in the Connecticut Department of Correction.

Detectable HCV RNA
Persistent elevations in hepatic transaminase levels (aspartate aminotransferase level, >35 IU/mL; or alanine aminotransferase level, >39 IU/mL) for at least 6 months
No evidence of another etiology of chronic liver disease (e.g., hemochromatosis, autoimmune hepatitis, or Wilson disease)
Stability of other chronic illnesses (e.g., HIV, diabetes, or mental illness)
No evidence of decompensated cirrhosis
No evidence of chronic renal insufficiency (creatinine level, >1.2 mg/dL)
Pretreatment mental health screening with evidence of stable mental health, with findings confirmed by a psychiatrist
Sufficiently long prison sentence to obtain liver biopsy (~3 months) and complete treatment while incarcerated:
9 Months for HCV genotypes 2 and 3
15 Months for all other genotypes
Patient is willing to defer any early-release programs (e.g., halfway houses or parole) until treatment has been fully completed
Patient is willing to be transferred to and remain at a correctional facility where 24-h nursing is available
Patient is willing to sign a treatment contract regarding adherence with treatment and recommendations by the infectious diseases specialist

mental health team. Chemical dependence issues are assessed, but enrollment in a treatment program is not required. HIV-infected patients continue to receive their HAART regimens.

Treatment is initiated by the infectious diseases specialist in conjunction with an infection-control nurse and follows the standard protocol for PEG-RBV developed by CMHC. After the initial 12 months of the treatment protocol, pegylated IFN-α-2b was changed to pegylated IFN-α-2a to avoid correcting for weight-based dosing and pharmacy errors. Weight-based dosing replaced standard RBV dosing (400 mg twice per day) in August 2003. All treatment is directly observed. Patients may receive hematopoeitic growth factors at the discretion of the infectious diseases specialist [42]. Quantitative HCV RNA levels are measured by PCR at 12 weeks; if a ⩾2-log decrease in HCV RNA levels (i.e., early virologic response) has not occurred, treatment is terminated.

Study population, data collection, and analysis. Data from all patients reviewed by the HepCURB between September 2002 and October 2005 were analyzed. Patients for whom the 6-month posttreatment HCV RNA measurements occurred after October 2006 were censored. Patients who had previously received IFN therapy, either in the community or while in prison, were excluded from the study. Baseline demographic, laboratory, psychosocial, and histopathologic data were collected on standard forms by chart review. Quantitative HCV RNA levels were determined using the Cobas Amplicor HCV Monitor 2.0 assay (Roche Diagnostics) and were subsequently measured by PCR to detect early virological response at 12 weeks, at the end of treatment, and 6 months after completion of treatment (SVR).

Data were collected from 4 sources: (1) standardized forms to the HepCURB that included demographic information, end-of-sentence and parole dates, baseline laboratory assessments, HIV information, a clinical problem list, concomitant medications, Beck's Depression Inventory, and psychiatric assessment findings; (2) the CMHC laboratory database; (3) the CMHC pharmacy database; and (4) inpatient discharge summaries.

For analyses of pharmacy use, we assessed the receipt of medication during the 6 months before treatment, during treatment, and the 6 months after treatment. For calculating the per-person-month rate of dosing, the denominator for the rate was calculated by summing up the number of months for which follow-up data were present. For the erythropoietin and granulocyte colony-stimulating factor analyses, we tabulated the number of doses for each of the treatment periods. For psychiatric medications, for each of the categories analyzed (antidepressant, anxiolytic, antipsychotic, and mood stabilizer), we recorded the dates on which a patient received a new psychiatric medication or an increased dose. Decreases in doses were not analyzed, because such changes were presumed to be unrelated to anti-HCV treatment.

The primary outcome was SVR (i.e., achievement of undetectable HCV RNA levels at 6 months after treatment). Any subject released from prison ⩽6 months after completion of PEG-RBV treatment was considered not to have achieved an SVR. Intention-to-treat analysis was performed, with missing values considered to be treatment failures. Multivariable analysis predicting SVR was conducted using the Proc Genmod procedure in SAS software, version 8.2 (SAS Institute). Initial univariate analyses were made to first assess the predictor variables. Subsequently, a multivariate model was constructed using forward and backward regression and optimizing the Aikeke's information criterion. All analyses were conducted using SAS software, version 8.2. The study received approval by the institutional review boards of Yale University and the University of Connecticut (Farmington).

RESULTS

From 2002 through 2006, 138 treatment-naive patients were evaluated for PEG-RBV therapy in the CTDOC, and 68 (49%) were approved by the HepCURB to receive therapy. The clinical characteristics of these subjects are shown in table 2. Among the 70 patients who were not approved for therapy, 40 (57%) had a release date that we judged to be too early to consider treatment outcome (median time to release, 10 months; interquartile range, 6.5–13 months). Other reasons given by the HepCURB that therapy was declined are presented in table 3.

Combination PEG-RBV treatment was prescribed for 68 subjects. Fifty-nine subjects (87%) received IFN-2a, and 9 (13%) received IFN-2-b; 16 (24%) received 800 mg of RBV per day, whereas 2 (3%) received 1000 mg and 50 (74%) received 1200 mg per day. Fifty-one patients (75%) were infected with HCV genotype 1, 58 (85%) were male, 33 (49%) were nonwhite, and 29 (43%) had a history of major depression (table 4). The median age was 41.9 years. Among the 19 patients (28%) who were HIV infected, the median CD4 cell count was 584 cells/mL (interquartile range, 490–696 cells/mL); 15 (79%) had an HIV-1 RNA level <50 copies/mL, and 15 (79%) were receiving combination antiretroviral therapy. Overall, 21 (31%) of 68 patients did not complete treatment; the median time to cessation of therapy among the 17 patients infected with non-2/3 HCV genotypes was 17 weeks (interquartile range, 14–26 weeks), whereas 3 patients infected with HCV genotype 2 or 3 prematurely discontinued therapy (2 within the first 2 weeks and 1 after 22 weeks). The most common reason for premature discontinuation of PEG-RBV treatment was lack of early virological response (12 [57%] of 21 patients); only 9 treated subjects (13%) discontinued therapy for medical reasons. The overall intention-to-treat SVR rate was 47.1%; the SVR rate was 43.1% among patients infected with non-2/3 HCV geno-

Table 2. Characteristics of 138 patients referred for treatment of hepatitis C virus (HCV) infection.

Characteristic	Treatment request denied (n = 70)	Treatment request approved (n = 68)
Age, median years (IQR)	41.4 (35.9–46.3)	41.7 (38.3–46.1)
Sex		
Male	61 (87.1)	58 (85.3)
Female	9 (12.9)	10 (14.7)
Ethnicity		
Black, not Hispanic	29 (41.4)	16 (23.5)
Hispanic	12 (17.1)	17 (25.0)
White or other	29 (41.4)	35 (51.5)
HCV genotype		
1	46 (65.7)	51 (75.0)
2 or 3	5 (7.1)	6 (8.8)
4	1 (1.4)	11 (16.2)
Unknown	14 (20.0)	0 (0.0)
ALT level		
Median IU/L	74 (52–123)	101 (68.5–137.5)
<35 IU/L[a]	6 (8.6)	0 (0.0)
35–70 IU/L[b]	24 (34.3)	18 (26.5)
>70 IU/L[c]	35 (50.0)	46 (67.6)
Data missing	5 (7.1)	4 (5.9)
HCV RNA level		
<400,000 copies/mL	0 (0.0)	11 (16.2)
⩾400,000 copies/mL	60 (85.7)	57 (83.8)
Data missing	10 (14.3)	0 (0.0)
HIV status		
Infected	27 (38.6)	19 (27.9)
Uninfected	40 (57.1)	49 (72.1)
Unknown	3 (4.3)	0 (0.0)
Hepatic activity index necroinflammatory score		
Median (IQR)	4 (3.75–6.75)	8 (5.5–10)
Minimal (0–3)	2 (4.3)	2 (4.3)
Mild (4–8)	8 (17.0)	31 (66.0)
Moderate (9–12)	0 (21.3)	10 (21.3)
Severe (13–18)	1 (2.1)	4 (8.5)
Ishak fibrosis score (n = 47)		
0	1 (1.4)	0 (0.0)
1/2	7 (10.0)	17 (25.0)
3/4	3 (4.3)	20 (29.4)
5/6	0 (0.0)	11 (16.2)
Not performed	59 (84.3)	20 (29.4)

NOTE. Data are no. (%) of patients, unless otherwise indicated. ALT, alanine aminotransferase; IQR, interquartile range.

[a] Normal level.
[b] One to 2 times the normal level.
[c] More than 2 times the normal level.

types and 58.8% among those infected with HCV genotype 2 or 3 (table 5).

The results of a multiple regression analysis of associations with SVR are presented in table 6. Only the presence of HCV genotype 1 with cirrhosis (adjusted OR, 12.8; $P = .04$) and major depression at baseline (adjusted OR, 3.4; $P = .05$) significantly predicted the failure to achieve an SVR.

Data on use of hematopoietic growth factor data are presented in table 7. Both erythropoietin (1.3 doses per patient-month; $75 per patient-month) and granulocyte colony-

Table 3. Reasons for deferral of hepatitis C therapy.

Reason for deferral	No. (%) of patients (n = 70)
Patient's release was too soon	40 (57.1)
Normal liver function test results	8 (11.4)
Normal biopsy findings	7 (10.0)
Patient refused consent/change of facilities	2 (2.9)
Patient refused consent/other	5 (7.1)
Hepatic decompensation	2 (2.9)
Patient deemed to be noncompliant	1 (1.4)
Patient had uncontrolled HIV disease	3 (4.3)
Patient had uncontrolled diabetes	1 (1.4)
Unclear	1 (1.4)

stimulating factor (0.3 doses per patient-month; $52 per patient-month) were used extensively. Data on use of psychiatric medications are presented in table 8. Only 1 patient received a mood-stabilizing medication during the period of observation; thus, this class of pharmaceutical agents was not analyzed. Patients were approximately twice as likely to receive a new antidepressant medication while receiving HCV treatment as they were before receiving HCV treatment (2.2 and 0.8 per-person-year, respectively). This trend was similar for opioid medications (1.8 and 0.5 per-person-year, respectively) used to treat pain.

During the 552 person-months of observation, 4 subjects were hospitalized 5 times while receiving anti-HCV therapy for a total of 33 days (0.06 hospitalized days per patient-month). Two patients were hospitalized (for 3 and 5 days, respectively) for RBC transfusions necessitated by symptomatic anemia; neither hospitalization resulted in discontinuation of anti-HCV treatment, and neither patient achieved an SVR. One patient developed *Staphylococcus aureus* septicemia and disseminated intravascular coagulation associated with a perirectal abscess; she was initially hospitalized for 9 days for stabilization and to receive intravenous antibiotics. After discharge, she was rehospitalized for 15 days because of a central line infection and *Escherichia coli* sepsis, and she developed decompensated cirrhosis. Anti-HCV treatment was discontinued, and the patient did not achieve an SVR. The remaining patient was hospitalized for 1 day because of a laceration that occurred during an altercation. He completed therapy but did not attain an SVR.

DISCUSSION

Our study is, to our knowledge, the first to establish the feasibility and clinical effectiveness of PEG-RBV treatment in a correctional setting. The overall SVR rate of 47% found among a study population in which 28% of subjects had HIV coinfection and 75% were infected with HCV genotype 1 compares favorably with results from community settings [43, 44], including results for drug users and persons with psychiatric comorbidities [45]. The SVR rate of 47% may have been considerably higher because 9 subjects (13%) were released from prison after their treatment but were not assessed for SVR 6 months later. It is also consistent with results from programs in correctional settings that administer standard IFN therapy. Our results were attained despite the high prevalence of alcohol and substance use disorders, high prevalence of psychiatric comorbidities, and the large proportion of ethnic minorities. Our results are similar to the SVR rate of 52% among 59 prisoners in Canada who received standard IFN treatment; more than one-half of the subjects in that study were infected with the "favorable" HCV genotypes 2 or 3, and HIV status was not reported [36]. With regard to other programs that used standard IFN-RBV treatment, the SVR rate was reported to be 29% among 93 prisoners in Rhode Island [46] and 36% among 119 prisoners in Virginia [37]; neither of those studies included HIV-infected prisoners.

The completion rate in our study (69%) is comparable to the rates from other prison-based studies. Most of the discontinuations in our study resulted from preplanned treatment terminations based on the lack of achievement of an early virological response at 12 weeks. Indeed, only 13% of our study population discontinued therapy because of adverse consequences—a better rate than in other North American studies of prisoners and a rate similar to rates reported from prospective clinical trials of HCV treatment among HIV-infected [44, 47, 48] and HIV-uninfected [23, 49] subjects.

These results have several important public health implications. First, our study population included a large number of

Table 4. Psychiatric comorbidities among 68 patients who were approved for hepatitis C therapy.

Characteristic	Value
Self-reported psychiatric history	
Major depressive disorder	29 (42.6)
Bipolar disorder	4 (5.9)
Anxiety disorder	6 (8.8)
Previous psychiatric hospitalization	11 (16.2)
Received psychiatric medication	26 (38.2)
Suicide attempt	8 (11.8)
Drug use	
Cocaine	52 (76.5)
Heroin	46 (67.6)
Injection drug use	46 (67.6)
Age at first injection drug use, years (IQR)	20 (17–25)
CAGE score (n = 57)	
0	22 (38.6)
1	6 (10.5)
⩾2	29 (50.9)

NOTE. Data are no. (%) of patients, unless otherwise indicated. IQR, interquartile range.

Table 5. Treatment disposition and outcomes, by hepatitis C virus (HCV) genotype.

Disposition	HCV genotype		All
	Genotype 1	Genotype 2/3	
Completed therapy[a]	34 (66.7)	13 (76.5)	47 (69.1)
Stopped treatment early[a]	17 (33.3)	4 (23.5)	21 (30.9)
Median duration of therapy, weeks (IQR)	17 (14–26)	1 (1–24)	18 (14–25)
Nonresponse to treatment	11 (64.7)	1 (25.0)	12 (57.1)
Mental health issues	2 (11.8)	1 (0.0)	3 (14.3)
Adverse effects of treatment	2 (11.8)	0 (0.0)	2 (9.5)
Early release, patient discontinuation of treatment, or missing chart	2 (11.8)	2 (50.0)	4 (19.0)
Virologic response among all treated subjects[a]			
Early virologic response	40 (78.4)	14 (82.4)	54 (79.4)
End-of-treatment response	31 (60.8)	12 (70.6)	43 (63.2)
Sustained virologic response	22 (43.1)	10 (58.8)	32 (47.1)
Response among HIV-uninfected patients[b]			
Early virologic response	30 (83.3)	11 (84.6)	41 (83.7)
End-of-treatment response	26 (72.2)	10 (76.9)	36 (73.5)
Sustained virologic response	17 (47.2)	8 (61.5)	25 (51.0)
Response among HIV-infected patients[c]			
Early virologic response	10 (66.7)	3 (75.0)	13 (68.4)
End-of-treatment response	5 (33.3)	2 (50.0)	7 (36.8)
Sustained virologic response	5 (33.3)	2 (50.0)	7 (36.8)

NOTE. Data are no. (%) of patients, unless otherwise indicated. IQR, interquartile range.

[a] Data are for 51 patients with HCV genotype 1 infection, 17 patients with HCV genotype 2 or 3 infection, and 68 patients in total.
[b] Data are for 36 patients with HCV genotype 1 infection, 13 patients with HCV genotype 2 or 3 infection, and 49 patients in total.
[c] Data are for 15 patients with HCV genotype 1 infection, 4 patients with HCV genotype 2 or 3 infection, and 19 patients in total.

subjects who would normally not receive treatment in community settings because of substance use disorders and/or mental illness. Although a few model programs have demonstrated success treating such individuals [28, 32, 45, 50], few have successfully done so in community settings [45]. Similarly, a large proportion of treated subjects were of racial and ethnic minorities. As such, treatment of HCV infection in correctional settings has the potential to reduce the gap in health disparities witnessed in North America and elsewhere.

Several studies have demonstrated low SVR rates among black persons [37, 51, 52]. Although increased dosages of pegylated IFN have not demonstrated improved outcomes among black persons [53], the weight-based RBV dosing, highly supervised treatment setting, and prompt management of consequences of therapy may have eliminated these disparities here. We note, however, that only 35% of black persons who were referred ultimately received treatment, compared with 55% of white persons. The assessment process was blinded to race; it is unclear why this inequity occurred.

The SVR rate in our study among HIV-seronegative subjects (51.0%) is similar to rates reported from prospective, randomized, controlled clinical trials [23, 49]; among HIV-seropositive subjects (SVR rate, 36.8%), it is higher [44, 47]. Treatment outcomes among the prisoners in our study are better than

Table 6. Multiple regression analysis of correlates of failure to achieve a sustained virologic response ($n = 68$).

Covariate	No. (%) of patients	Adjusted OR (95% CI)	*P*
HCV genotype 1 infection with cirrhosis	10 (15)	12.8 (1.1–147.8)	.04
Major depressive disorder	29 (43)	3.4 (1.01–11.6)	.05
HCV load, >400,000 IU	57 (84)	3.4 (0.5–21.6)	.20
HCV genotype 1 infection without cirrhosis	41 (60)	1.8 (0.4–7)	.42
HIV infection	19 (28)	1.4 (0.4–5.1)	.63

NOTE. Adjusted ORs were calculated with respect to the following base case: an HIV-uninfected individual with HCV genotype 2 infection, a hepatitis C virus (HCV) load <400 IU/mL, and no major depressive disorder. Other variables that did not remain in the model are not presented.

Table 7. Use of hematopoeitic growth factors during hepatitis C treatment.

Agent, time frame	Total duration of treatment, months	Total no. of doses	Dosage, μg per patient-month	No. of doses per patient-month	Price, $ per patient-month
G-CSF[a]					
6 Months before HCV therapy	408	0	0.00	0.00	0
During HCV therapy	552	163	88.59	0.30	52
6 Months after HCV therapy	408	1.3	0.98	0.01	2
Erythropoeitin[b]					
6 Months before HCV therapy	408	7	172	0.02	1
During HCV therapy	552	899	16,286	1.63	75
6 Months after HCV therapy	408	66	1618	0.16	7

NOTE. G-CSF, granulocyte colony-stimulating factor.

[a] The price of G-CSF was $177 per 300-μg dose. Seven (10%) of 68 patients received G-CSF at some point.

[b] The price of erythropoeitin was $46 per 10,000-U dose. Twenty-five (37%) of 68 patients received erythropoeitin at some point.

those reported in community settings [28–30, 52, 54–58] and in methadone maintenance programs [31, 59].

Our study also provides some preliminary data that could be useful for the planning of corrections-based and managed care treatment programs. The prescription of hematopoietic growth factors highlights their relevance in avoiding dose reductions in PEG-RBV, although it is unknown whether they influenced SVR [60]. In the community, the appropriate management of hematological perturbations with growth factors is critical to achieving sound outcomes [44, 61–63]. We also provide, to our knowledge, the first data on hospitalization among a cohort of patients treated with PEG-RBV. The low rate of hospitalization, most of which was not related to HCV treatment, underscores the safety of this treatment in correctional settings.

Given the negative impact of baseline depression (a 3-fold increase in the failure rate), screening and prompt treatment of this disease is particularly relevant. Furthermore, it has been well established that IFN is associated with worsening psychiatric symptomatology [45, 64]. Indeed, in our study, we found a substantial increase in use of both opioid and antidepressant medication during treatment. Incorporating rigorous methods

Table 8. Use of psychiatric medication during hepatitis C treatment.

Time frame	Total duration of therapy, months	New medication prescription		Dose increase	
		No. of prescriptions	No. of prescriptions per patient-year	No. of dose increases	No. of dose increases per patient-year
Antidepressant (n = 25)					
6 Months before HCV therapy (n = 10)	150	12	1.0	11	0.9
During HCV therapy (n = 16)	122	22	2.2	21	2.1
6 Months after HCV therapy (n = 7)	150	9	0.7	9	0.7
Benzodiazepine (n = 7)					
6 Months before HCV therapy (n = 2)	54	3	0.7	0	0.0
During HCV therapy (n = 4)	75	4	0.6	0	0.0
6 Months after HCV therapy (n = 1)	54	2	0.4	0	0.0
Antipsychotic medication (n = 9)					
6 Months before HCV therapy (n = 9)	54	4	0.9	0	0.0
During HCV therapy (n = 12)	83	6	0.9	0	0.0
6 Months after HCV therapy (n = 11)	54	1	0.2	0	0.0
Narcotic (n = 12)					
6 Months before HCV therapy (n = 3)	66	3	0.5	0	0.0
During HCV therapy (n = 11)	109	16	1.8	1	0.1
6 Months after HCV therapy (n = 2)	66	2	0.4	0	0.0

NOTE. Data regard the prescription of a new medication or a dose increase among patients who received antidepressants during the peri–HCV-treatment period.

of assessment of depressive and pain symptomatology into treatment protocols will be important in improving adherence and decreasing morbidity in any setting.

A major barrier to expanding treatment is the relatively long duration of treatment and the short duration of incarceration for many persons who might have benefited from treatment. In our study, inmates with insufficient remaining incarceration time were denied the opportunity to undergo biopsy or to proceed with treatment; this is similar to the Virginia program, in which the time to sentencing had to be >24 months for subjects to be eligible for treatment [37]. The rationale in both cases was to ensure that treatment could be completed, because medications and health insurance are seldom available to those who leave prison. Other states, such as New York, do not have such restrictions [65]. Expanding access to treatment for many other patients, however, hinges on the creation of effective transitional programs that would link patients to community-based care after release [66].

Furthermore, given the positive treatment outcomes seen in our study, we agree with other authors that screening should be performed routinely in correctional facilities, with performance of HCV antibody testing either for all patients or for patients who answer positively to key screening questions, such as persons who indicate former injection drug use or prior positive hepatitis B virus or HCV test results [3, 12, 67]. In our study, the presence of HCV genotype 1 combined with evidence of cirrhosis on liver biopsy was associated with a 12-fold increased likelihood that an SVR would not be achieved. These findings confirm findings reported by others [3, 43, 68] that early diagnosis and treatment of HCV infection before the development of cirrhosis should be part of the public health infrastructure. Passive detection, which is used by nearly all correctional settings, has not been sufficient for effectively achieving early detection.

Routine HCV screening programs are lacking, however, largely because of the concern that increasing testing for HCV would massively increase costs in settings where it is unpopular to increase budgets for prisoners. In the Rhode Island study, treatment represented ~5% of the total health care budget for the state correctional system [69]. Currently, the costs for screening and treatment in correctional settings are largely under the jurisdiction of already financially strapped correctional systems. The consequence of this approach is that the cost to society increases as these same individuals return to the community and incur costs in the public sector [9]. Future efforts will require cost sharing for HCV treatment in correctional settings as a means to increase treatment and reduction in end-stage liver disease among members of society.

In summary, these results support the utility of PEG-RBV for the treatment of hepatitis C in correctional settings. The challenge now is for correctional settings to implement active detection and comprehensive treatment programs.

Acknowledgments

We thank Paula Dellamura, for her administrative oversight; Mark Buchanan, Edward Pesanti, Edward P. Blanchette, and John Gittzus, for their clinical expertise in managing these patients; the Infection Control Nurses, who coordinated the excellent care for these inmates; and, especially, the inmates.

Financial support. The National Institutes on Drug Abuse (career development awards K24 DA 0170720 [to F.L.A.] and K23 DA 022143 [to R.D.B.]) and the National Institutes of Health Medical Science Training Program (GM07205 to D.S.-R.M. and S.B.).

Potential conflicts of interest. All authors: no conflicts.

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