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Is Telaprevir an Effective Addition to the Currently Available 48 Week Therapy Regimen in Chronic Hepatitis C Genotype 1 Patients?

Jeanette L. Nienaber

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Is telaprevir an effective addition to the currently available 48 week therapy regimen in chronic hepatitis C genotype 1 patients?

Jeanette L. Nienaber, PA-S

A SELECTIVE EVIDENCE BASED MEDICINE REVIEW

In Partial Fulfillment of the Requirements For

The Degree of Master of Science

In

Health Sciences – Physician Assistant

Department of Physician Assistant Studies
Philadelphia College of Osteopathic Medicine
Philadelphia, Pennsylvania

December 14, 2012
OBJECTIVE: The objective of this selective EBM review is to determine if the addition of telaprevir reduces treatment time and is more effective than the presently available pegylated interferon α2a-ribavirin combination 48 week therapy.


DATA SOURCES: Articles selected for review, are randomized, double blind, placebo controlled trials found in peer-reviewed journals using Cochrane, Medline and PubMed databases.

OUTCOMES MEASURED: Effectiveness of telaprevir was measured by determining the potentiality of reducing treatment outcome from 48 weeks to 24 weeks and the overall success rate of telaprevir as an add-on drug to the current therapy.

RESULTS: Jacobsen et al. (2011) found that 75% of patients using telaprevir as an add-on medication had SVR rates compared to 44% using placebo add-on. Sherman et al. (2011) found that a 24 week regimen of the currently available therapy with telaprevir added in the first 12 weeks was non-inferior to the same regimen at 48 weeks duration. No statistical differences were seen in variable dosing frequencies and intervals (P ≥0.787) and SVR was achieved in most patients with similar adverse reactions (Marcellin et al., 2011).

The safety profile monitored adverse reactions seen including anemia, nausea, diarrhea and skin rashes. Similar adverse reactions are seen with the current therapy; however incidence rate does increase with the triple therapy regimen. All adverse reactions dissipated once medication was discontinued.

CONCLUSIONS: Data shows that telaprevir is an effective addition to the currently available therapy of peginterferon α-2a/ribavirin for chronic HCV genotype 1 patients. The three studies selected for review suggest that a large proportion of patients would show promising results with addition of telaprevir for a 24 week treatment duration being used twice daily for the first 12 weeks with similar adverse reactions and greater patient compliance than seen with the currently available therapy.

KEY WORDS: chronic HCV therapy, hepatitis C, current HCV treatment, telaprevir
INTRODUCTION

An estimated 200 million individuals worldwide are living with chronic hepatitis C virus (HCV) infection. In 2010, the annual incidence rate in the United States (U.S.) was estimated at 17,000 individuals with 2.7 – 3.9 million living with chronic HCV.² Caused by a single stranded RNA virus, the infection is the most common blood borne pathogen, the primary cause of liver dysfunction and decompensation, and the leading indication for liver transplantation in the U.S.⁹

HCV increases the risk for hepatic fibrosis, portal hypertension, compensated cirrhosis and hepatocellular carcinoma. The virus is also very promiscuous in that it will manifest throughout the body making its presence well known to healthcare providers.⁴ Patients often seek multi-specialty healthcare visits due to extra-hepatic manifestations affecting the skin, eyes, thyroid, immune system, small vessels, kidneys and salivary glands all of which are theorized to be immune mediated.⁴,⁵,⁶,¹⁰ Quantified annual healthcare visits are unknown due to variability in disease symptomatology, however it is known that costs correlate with disease severity. Healthcare visits and costs are lower for individuals unaware of viral infection and asymptomatic compared to individuals suffering from end stage liver disease.² Annual costs per patient vary greatly and range from a mild chronic HCV condition ($145) to compensated cirrhosis ($585 - $1110) to hepatocellular carcinoma ($23,755 - $44,200) to liver transplantations ($201,110).³

Acute symptoms are seen in only 16% of patients,⁹ which is why 80% of those that become infected, progress to chronic carriers. Up to 90% of the chronic carriers advance to chronic liver disease. Of those patients, 20 – 30% progress to cirrhosis and a further 5 – 10% will develop hepatocellular carcinoma.¹ Disease diagnosis is detection of anti-HCV or HCV RNA or both via serologic assay. Disease severity is determined via liver biopsy.⁹
Several genotypes of HCV exist. Genotype 1 is the most prevalent with patients yielding the poorest response to standard therapy. Current recommended therapy are weekly subcutaneous injections of pegylated interferon alpha-2a (PEG IFN-α2a) coupled with a daily weight based oral (PO) intake of 1000 – 1200 mg of ribavirin for 48 weeks. Response to therapy is clinically monitored by HCV RNA levels. Sustained vireologic response (SVR), defined as a suppressed HCV RNA level 24 weeks after last treatment dose, is undetectable by serologic assay in 42 - 52% of patients using the currently available 48 week treatment. Side effects of treatment results in many patients being non-compliant with the 48 week therapy duration. These symptoms include but are not limited to fatigue, pruritus, anemia, nausea, headache, rash, insomnia, diarrhea and influenza-like illness. If conventional pharmacologic therapy is unsuccessful, liver transplantation may be required.

Research into new therapies is of great importance. Primarily due to the high prevalence of genotype 1 infection worldwide, of which, have comparatively poor response rates with the same treatment therapy than seen with HCV 2, 3, 4 & 6 genotypes. Secondly, the length of therapy duration complicated by significant physical side effects, result in poor patient compliance. Poor response rates and poor patient compliance lead to disease progression ending with high morbidity/mortality rates associated with high health care costs. One such new therapy is the addition of telaprevir, a serine protease inhibitor, to the currently available PEG IFN-α2a/ribavirin regimen which has been recently studied in treatment naïve patients.

OBJECTIVE

The objective of this selective EBM review is to determine if the addition of telaprevir reduces treatment time and is more effective than the presently available PEG IFN-α2a/ribavirin combination 48-week therapy.
METHODS

Three randomized controlled studies (RCT), two of which were double blind studies and one exploratory, open-label study, were chosen for this selective EBM review. The population of interest was chronic HCV genotype 1 patients. The intervention chosen was the addition of telaprevir to the current therapy of PEG IFN-α2a/ribavirin dual combination. Control and treatment groups were slightly variable between studies (Table 1). Inclusion criteria for studies were diagnosed chronic HCV genotype 1 patients, articles published after 2006 with outcomes that were evidenced based relevant to the patient. Exclusion criteria were liver disease derived from causes other than the HCV genotype 1 and hepatocellular carcinoma. Further study specific demographics and characteristics are listed Table 1.

The author performed the article search in the Cochrane, Medline and PubMed databases using the keywords ‘chronic HCV therapy’, ‘hepatitis C’, ‘current HCV treatment’, and ‘telaprevir’. All articles were published in peer-reviewed journals in the English language in 2011.

If stated, 95% confidence intervals were used with significance level of $P < 0.05$, non-inferiority margin of -10.5% and an 80% power to rule out. Statistical tests used were a two-sided, continuity-corrected chi-square test, non-inferiority comparison, and a logistical regression model. The number needed to treat (NNT) was calculated for each efficacy study. Number needed to harm (NNH), experimental event rate (EER), control event rate (CER), relative risk (RR) and absolute risk increase (ARI) were calculated for safety comparisons done in the Jacobson et al. (2011) and Sherman et al. (2011) studies. Marcellin et al. (2011) was not compared in the safety profile, as all adverse reactions were documented even if not specific to the medication involved or severity of reaction as the two formerly mentioned papers had done.
Table 1. Demographics and characteristics of reviewed EBM articles

<table>
<thead>
<tr>
<th>Study</th>
<th>Type</th>
<th># Pts (n)</th>
<th>Age</th>
<th>Inclusion Criteria</th>
<th>Exclusion Criteria</th>
<th>W/D</th>
<th>Interventions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Jacobson^4 (2011)</td>
<td>RCT, double blind, placebo-controlled</td>
<td>total n = 724</td>
<td>18 – 70 yo</td>
<td>Chronic genotype 1 HCV – determined by liver biopsy within 12 mos of study</td>
<td>Decompensated liver disease Liver disease derived from other causes</td>
<td>n = 57</td>
<td>Control (PR): placebo add on x 12 wks, then 36 current therapy Treatment (T12PR): telaprevir add on x 12 wks, then 12 - 36 wks of current therapy</td>
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<tr>
<td></td>
<td></td>
<td>PR: n = 361</td>
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<td></td>
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<td>T12PR: n = 363</td>
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<tr>
<td>Sherman^10 (2011)</td>
<td>RCT, open label, double blind, phase 3 non-inferiority trial</td>
<td>total n = 322</td>
<td>18 – 70 yo</td>
<td>Chronic HCV genotype 1 – determine via biopsy within 6 mos of study</td>
<td>Hepatic decompensation Liver disease from other causes Active cancer within past 5 years Co-infection of HBV or HIV</td>
<td>n = 42</td>
<td>Control (T12PR48): telaprevir add on x 12 wks, then current therapy x 36 wks. Treatment (T12PR24): telaprevir add on x 12 wks, then current therapy x 12 wks</td>
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<tr>
<td></td>
<td></td>
<td>T12PR48: n = 160</td>
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<td></td>
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<tr>
<td></td>
<td></td>
<td>T12PR24: n = 162</td>
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<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>Marcellin^6 (2011)</td>
<td>RCT, exploratory, prospective, multicenter, open-label, phase 2 clinical trial</td>
<td>total n = 80</td>
<td>18 – 65 yo</td>
<td>Chronic HCV genotype 1 – determine via biopsy within 18 mos of study</td>
<td>History of alcohol abuse Documented cirrhosis Evidence of other liver disease HIV co-infection</td>
<td>n = 6</td>
<td>Control (q8h): telaprevir 750 mg q8h x 12 wks + current therapy, then current therapy for 12 – 36 wks. Treatment (q12h): same as above except telaprevir 1125 mg q12h</td>
</tr>
<tr>
<td></td>
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<td>q8h: n = 40</td>
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<td>q12h: n = 40</td>
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</table>

Current therapy = PEG IFN-α2a/ribavirin
OUTCOMES MEASURED

The author was interested in patient oriented outcomes that mattered (POEM) for chronic HCV genotype 1 individuals. POEMs selected were to determine if the addition of telaprevir was effective and/or non-inferior in reducing treatment duration from 48 to 24 weeks, as well as determining the effectiveness in therapy outcome when increasing dosage and reducing interval frequency. Since the majority of chronic cases of HCV are asymptomatic, to determine these outcomes, plasma HCV RNA levels were quantified using the COBAS TaqMan HCV assay, version 2.0 via blood samples taken during follow up study visits. Follow up visits varied between studies (min = 17, max = 19) and was dependent on patient response to treatment. Patients that achieved early undetectable HCV RNA levels were more likely to end treatment at 24 weeks and therefore have less follow up study visits. All patients were included in a follow up post treatment study visit, which again varied between studies (min = 4 weeks, max = 48 weeks). HCV RNA levels were reported as either detectable (> 25 IU/mL) or undetectable (< 25 IU/mL).

The safety profile for control and treatment groups were monitored by documenting adverse reactions to the medications seen in physical examination by investigators and described by patients during study visits. The severity of each adverse reaction was dependent on study protocols and criteria used.4,6,10 A side effect of ribavirin is anemia which is managed by dose reductions, if reductions in telaprevir were required in order to manage anemia in a patient, treatment was discontinued.9 In all studies, serious adverse events in patients were subject to treatment discontinuation in those patients as determined by investigators.4,6,10
RESULTS

EFFICACY

Jacobson et al. (2011) found statistical significance in patients with undetectable HCV RNA levels at the follow up study visit 24 weeks after the final treatment dose in the telaprevir (T12PR) group compared to the placebo (PR) group (75%, 44% respectively, P < 0.001). This study visit is also known as the primary end point (Table 2). All patients independent of treatment duration were tested at a 72 week follow up visit. Significance was seen in the T12PR group with undetectable HCV RNA levels compared to the control group (73%, 44% respectively, P < 0.001). Over half of the patients tested on follow up visit week 4, and collectively at week 4 and week 12, in the T12PR had undetectable HCV RNA levels. This compared to 9% and 8% of patients in the PR group at those respective study visits. Patients were again tested on the last day of treatment dosing. In the T12PR, this study visit was completed on either week 24 or week 48, depending on patient treatment duration. Of the total study sample size of n = 363, 314 had undetectable HCV RNA levels on week 24 or 48. All patients in the PR group received 48 weeks of treatment. The study sample size was n = 361 with a total of 229 patients having undetectable HCV RNA levels at week 48. Relapse, meaning detectable HCV RNA levels, among patients were tested 24 weeks after their last treatment dose in those that had undetectable HCV RNA levels at their last day of dosing. Of the 314 patients with undetectable HCV RNA levels on their last day of dosing, relapse occurred in 27 (9%) patients, compared to 64 out of 229 (28%) patients in the PR group. Results for Jacobson et al. (2011) are also referred to in Table 2. Based on data from the primary end point, four patients would need to be treated with telaprevir in order to prevent one additional patient being sampled for detectable HCV RNA levels.
Table 2. Response during and after treatment period – n (%).\textsuperscript{4}

<table>
<thead>
<tr>
<th></th>
<th>T12PR n = 363</th>
<th>PR n = 361</th>
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</thead>
<tbody>
<tr>
<td>Undetectable HCV RNA during treatment period</td>
<td></td>
<td></td>
</tr>
<tr>
<td>At week 4</td>
<td>246 (68)</td>
<td>34 (9)</td>
</tr>
<tr>
<td>At weeks 4 and 12</td>
<td>212 (58)</td>
<td>29 (8)</td>
</tr>
<tr>
<td>Undetectable HCV RNA 24 weeks after end of treatment: Primary end point</td>
<td>271 (75)</td>
<td>158 (44)</td>
</tr>
<tr>
<td>Undetectable HCV RNA at 72 week follow up visit</td>
<td>265 (73)</td>
<td>158 (44)</td>
</tr>
<tr>
<td>Relapse among patients with undetectable HCV RNA levels at end of treatment period</td>
<td>27/314 (9)</td>
<td>64/229 (28)</td>
</tr>
</tbody>
</table>

Number needed to treat = 4

Sherman et al. (2011) was a non-inferiority trial testing a 24 week (T12PR24) vs. a 48 week therapy regimen (T12PR48) with telaprevir being used in the first 12 weeks, (Table 3). The non-inferiority margin was pre-defined at -10.5%. At the primary end point study visit, the majority of patients obtained undetectable HCV RNA levels (T12PR24, 92%; T12PR48, 88%). The absolute difference between both groups was 4 percentage points (95% CI, -2 to 11). Both groups had similar and high rates of undetectable HCV RNA levels at week 4 and collectively at week 4 and week 12 (T12PR24, 100%; T12PR48, 99%).\textsuperscript{10} Low rates of relapse were also seen (T12PR24, 6%; T12PR48, 3%).\textsuperscript{10} Based on data from the primary end point, 23 patients need to be treated for 24 weeks to prevent one patient from having to do the full 48 week treatment.

Table 3. Response during and after treatment period – n (%).\textsuperscript{10}

<table>
<thead>
<tr>
<th></th>
<th>T12PR24 n = 162</th>
<th>T12PR48 n = 160</th>
</tr>
</thead>
<tbody>
<tr>
<td>Undetectable HCV RNA during treatment period</td>
<td></td>
<td></td>
</tr>
<tr>
<td>At week 4</td>
<td>162 (100)</td>
<td>159 (99)</td>
</tr>
<tr>
<td>At weeks 4 and 12</td>
<td>162 (100)</td>
<td>159 (99)</td>
</tr>
<tr>
<td>Undetectable HCV RNA 24 weeks after end of treatment: Primary end point</td>
<td>149 (92)</td>
<td>140 (88)</td>
</tr>
<tr>
<td>Relapse among patients with undetectable HCV RNA levels at end of treatment period</td>
<td>9/159 (6)</td>
<td>4/154 (3)</td>
</tr>
</tbody>
</table>

Number needed to treat = 23
Marcellin et al. (2011) looked at different dosages of frequency interval in telaprevir usage. There was no significant difference in HCV RNA response between a 750 mg every 8 hours (q8h) dose and an 1125 mg every 12 hours (q12h) dose at week 4, week 12 and end of treatment (P > 0.05, Table 4). Analysis also showed that patients had no significant difference in achieving SVR between the two treatment therapies (P ≥ 0.787). Patients that had undetectable HCV RNA levels between week 4 and week 20 were assigned to stop treatment at week 24 (Table 4). Among the patients assigned to 24 weeks of treatment, 96.7% (29/30) from the q8h group and 100% (29/29) from the q12h group achieved SVR. Amongst patients assigned to the 48 week treatment duration, 83.3% (5/6) and 75% (3/4) from the q8h and q12h groups achieved SVR respectively. Based on primary end point data, 40 patients need to be treated at the 1125 mg q12h before one additional adverse outcome would happen using the 750 mg q8h dosing.

Table 4. Response during and after treatment period from pooled 24 and 48 week treatment durations – n (%).

<table>
<thead>
<tr>
<th></th>
<th>q12h n = 40</th>
<th>q8h n = 40</th>
</tr>
</thead>
<tbody>
<tr>
<td>Undetectable HCV RNA during treatment period</td>
<td></td>
<td></td>
</tr>
<tr>
<td>At week 4</td>
<td>33 (82.5)</td>
<td>32 (80)</td>
</tr>
<tr>
<td>At week 12</td>
<td>33 (82.5)</td>
<td>37 (92.5)</td>
</tr>
<tr>
<td>Undetectable HCV RNA 24 weeks after end of treatment:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Primary end point</td>
<td>33 (82.5)</td>
<td>34 (85)</td>
</tr>
<tr>
<td>Undetectable HCV RNA at end of treatment</td>
<td>37 (92.5)</td>
<td>37(92.5)</td>
</tr>
<tr>
<td>Undetectable HCV RNA at week 4 through week 20</td>
<td>29 (72.5)</td>
<td>30 (75)</td>
</tr>
</tbody>
</table>

Number needed to harm = 40

SAFETY

In all three articles selected for review, 98 – 100% of patients in all groups independent of study experienced an adverse reaction. Most common adverse events were fatigue, pruritus, nausea, anemia and headache. Marcellin et al. (2011) recorded all events independent of
severity or drug relatedness making safety comparisons with the following two studies not
directly comparable. In Jacobson et al. (2011), 33 patients (9%) in the T12PR group had a
serious adverse event compared to 24 patients (7%) in the PR group. Reactions were similar
collectively between the two groups, most commonly seen being anemia (T12PR n = 8, PR n =
4), psychiatric (T12PR n = 2, PR n = 3), musculoskeletal (T12PR n = 2, PR n = 3) and cardiac
disorders (T12PR n=2, PR n = 2). The T12PR had two cases of rash which was not seen in the
PR group, while the PR group had four cases of renal and urinary disorders, with zero cases
notated in the T12PR group.4 In Sherman et al. (2011), serious adverse event incidences
increased with duration of treatment (T12PR24, n = 4; T12PR48, n = 16). Most commonly seen
were blood and lymphatic system disorders (T12PR24 n = 2, T12PR48 n = 5) and infections and
infestations (T12PR24 n = 1, T12PR48 n = 5). Anemia was seen in one case in the T12PR24
group, while pneumonia, metabolism and nutrition disorders, and dehydration were seen in the
T12PR48 group.10 Treatment effects were calculated for all serious adverse reactions seen during
the two studies that required patients to discontinue treatment (Table 5). For every 50 patients
treated in the Jacobson et al. (2011) study, one additional patient would experience a serious
adverse reaction. For every 12 patients treated in the Sherman et al. (2011) study, one fewer
patient would have a serious adverse reaction.

Table 5. Treatment effects for serious adverse reactions requiring treatment discontinuation.

<table>
<thead>
<tr>
<th></th>
<th>EER</th>
<th>CER</th>
<th>RR</th>
<th>ARI</th>
<th>NNT/NNH</th>
</tr>
</thead>
<tbody>
<tr>
<td>Jacobson et al. (2011)</td>
<td>0.09</td>
<td>0.07</td>
<td>1.29</td>
<td>0.02</td>
<td>50</td>
</tr>
<tr>
<td>Sherman et al. (2011)</td>
<td>0.02</td>
<td>0.10</td>
<td>0.20</td>
<td>-0.08</td>
<td>-12</td>
</tr>
</tbody>
</table>

DISCUSSION

Patients undergoing the current 48 week therapy of PEG IFN-α2a/ribavirin dual
combination experience low response rates with higher relapse rates than compared to the
addition of telaprevir (Table 4).\textsuperscript{4,12} In addition to these less than desirable outcomes, patients still experienced adverse reactions independent of duration length or addition of telaprevir.\textsuperscript{4,12}

Presently, the potential to reduce treatment duration is limited to patients that can attain an early virologic response (EVR) seen at week 4, which is greater than a 2 \( \log_{10} \) drop in HCV RNA level at baseline.\textsuperscript{4} Positive results have been shown with patients treated with response-guided therapy, in which patients that attain an EVR to the currently available treatment therapy benefit similarly in achieving SVR rates by stopping treatment at 24 weeks than at 48 weeks.\textsuperscript{7} In the three studies reviewed, EVR was achieved in 68 - 100\% of patients using telaprevir as an add-on medication\textsuperscript{4,6,10} compared to 9\% of patients observed in the placebo group in the Jacobson et al. (2011) study. This suggests that the majority of patients using telaprevir may benefit similarly by discontinuing treatment at 24 weeks than at 48 weeks. Sherman et al. (2011) concluded that the same triple combination therapy for 24 weeks was non-inferior to 48 weeks (Table 3).

Patient compliance improved in telaprevir groups that had reduced treatment durations.\textsuperscript{10} Reducing frequency interval at which a drug must be taken on a daily basis is also important.\textsuperscript{6} Currently, studies use telaprevir at a 750 mg dosage three times a day.\textsuperscript{4,6,8,10,11,12} Marcellin et al. (2011) showed similar adverse reactions (100\% of patients in both groups) and response rates (Table 4) when increasing dosage to 1125 mg and decreasing frequency to twice daily.

Safety profiles were consistent between the studies. Adverse reaction incidences increase with the use of telaprevir, with rash seen only with telaprevir use.\textsuperscript{4,6,9,12} Most commonly an eczematous type rash developing in the first 8 weeks\textsuperscript{4,6,10} and one case of Stevens-Johnson syndrome seen at 11 weeks after the final telaprevir dose.\textsuperscript{4} All rashes disappeared after telaprevir discontinuation.\textsuperscript{4,6,10} Overall, 99\% of patients (\( n = 161 \)) finished the 24 week treatment versus 74\% (\( n = 119 \)) in the 48 week regimen in the Sherman et al. (2011) study, the majority of
patients discontinuing treatment were due to adverse reactions. This suggests that a decrease in
treatment duration, even with an increased incidence of adverse reactions with telaprevir in the
first 12 weeks, still improves patient compliance in the majority of patients undergoing therapy.

All studies reviewed included treatment naïve patients with few that had advanced
disease that tends to be more difficult to cure.4,6,10 Follow up studies should include a greater
percentage of patients that are treatment naïve with advanced disease and patients with either no
response or a relapse to original treatment. Another important consideration is cost effectiveness.
Limited studies look at cost effectiveness using telaprevir as an add-on drug or comparatively to
current therapy. A study recently published by Thorland et al. (2012) showed that telaprevir with
the current dual combination therapy in treatment naïve patients and in response guided therapy
would cost anywhere from £29,930 - 32,530 (US $38,858 – 42,234). This is comparable to a
treatment naïve patient with advanced liver disease such as hepatocellular carcinoma, and much
less than the cost of liver transplantation.3,11 With the overall greater responses in EVR and SVR
achieved with the addition of telaprevir, health care providers may opt to treat a patient
diagnosed with chronic HCV genotype 1 initially with telaprevir as an add-on therapy rather than
putting financial efforts into the current therapy which yields low response rates.

CONCLUSIONS

The three studies selected for review suggest that a large proportion of chronic HCV genotype 1
patients would show promising results in the addition of telaprevir with a 24 week treatment
duration using telaprevir twice daily for the first 12 weeks with similar adverse reactions but
greater patient compliance than seen with the currently available therapy. Further studies should
look into patients with advanced liver disease, treatment experienced individuals, as well as the
cost effectiveness between all drug regimens.
REFERENCES


