The Role of Weight Loss as On-Treatment Predictive Factor for SVR in the Treatment of Chronic Hepatitis C Patients with Peginterferon alfa-2a and Ribavirin

Thomas Witthöft, Peter Buggisch, Dietrich Hüppe, Christine John, Axel Baumgarten, Gerlinde Teuber, Thomas Lutz, Gero Moog, Ulrich Alshuth, Stefan Mauss, Albrecht Stoehr

ABSTRACT

AIM: Pegylated interferon-alpha (PEG) and ribavirin (RBV) is the mostly used treatment for patients with chronic hepatitis C (CHC) leading to a strong and in many patients sustained viral response (SVR). In this study we analyzed the value of weight loss under PEG/RBV treatment as an easy to recognize on-treatment predictive factor besides viral response.

Methods: We conducted a Phase IV, multi-center, open-label, non-interventional study with treatment of Peginterferon alfa-2a and ribavirin. 1100 patients treated with a complete course of CHC therapy according to consensus recommendations (GT-1/4/5/6 for 48±4wks, GT-2/3 for 24±2wks). For these patients descriptive, univariate and multivariate analyses were performed to determine on-treatment predictive factors associated with SVR.

Results: In 737 genotype 1/4/5/6 infected patients the SVR rate was 39.3%. However, patients with a weight loss ≥2 kg under therapy achieved a statistically significant increased SVR of up to 44.4% whereas patients with a lower weight loss <2 kg showed only a SVR of 25.3%. In 363 patients with genotype 2/3 the overall SVR rate was 64.5% and in case of weight loss of more than 2 kg they achieved a SVR of 68.8% whereas patients who lost less than 2 kg showed a SVR of only 55.2%. A multivariate regression analysis revealed that EVR, RVR, age and weight loss under therapy but not genotypes have been positive and significant on-treatment predictors for SVR.

Conclusion: The present study demonstrates that weight loss is a reliable on-treatment predictive factor for estimating treatment outcome.

© 2014 ACT. All rights reserved.

Key words: Chronic hepatitis; Hepatitis C Virus; Pegylated interferon; Ribavirin; Body weight; Weight loss; Rapid virological response; Early virological response; Sustained virological response

INTRODUCTION

Hepatitis C infection is a global burden, with up to 170 million people being infected. The estimated number of carriers in Western Europe is 9 million and 66% to 75% of all HCV-infected patients have not been identified yet. Untreated CHC may lead to progression of fibrosis and cirrhosis and therefore may result in the development of
hepatocellular carcinoma.\textsuperscript{12-15}

Since 2001 the standard of care in HCV genotype 1 infected patients included pegylated interferon alfa-2a or 2b (PEG IFN-alpha 2a/2b) in combination with ribavirin (RBV) for a duration of 48 weeks and in genotype 2/3 a treatment duration of 24 weeks is recommended.\textsuperscript{16-18} Data from these randomized controlled trials demonstrated rates of sustained virological response (SVR) in 40%–50% of genotype 1 and in about 70%–80% of genotype 2/3-infected individuals. These study results are the basis of current treatment guidelines.\textsuperscript{3,19,20}

PEG IFN is given subcutaneously once weekly and ribavirin is being taken orally twice daily. Both medications may cause early or late side-effects (e.g. PEG causes flu-like symptoms, drop in platelet and white blood cell counts, ribavirin causes anemia). Therefore a shortened duration of therapy is highly appreciated in order to minimize adverse events for patients without lowering the therapeutic outcome. Further on especially due to rising costs in the global health care system and in country specific financial crisis a reduction of treatment duration and therefore application of medication is warranted. Besides achieving high SVR rates, a personalized healthcare becomes increasingly important as therapy should be as tolerable and effective as possible for every single patient. The knowledge of different baseline and on-treatment factors is required for early decision making for intensifying or facilitating therapy.

Next to the baseline predictive variables [e.g. GGT (gamma-glutamyltranpeptidase), age, IL-28B] most reliable on-treatment predictive factors of RVR and EVR for treatment success have been identified in trials of PEG plus RBV.\textsuperscript{4-6,12-17} Data from these trials are the basis for development of clinical guidelines and for decisions regarding patient management.

The aim of our study was to investigate whether age, body weight at start of therapy, genotypes, and on-treatment parameters like different virological responses and weight loss occurring throughout therapy might be suitable to predict therapeutic outcome and therefore abbreviating the treatment duration as soon as possible.

PATIENTS AND METHODS

From 01/2008 to 02/2011, 3810 patients were fully documented in a prospective non-interventional study conducted by the Association of German Gastroenterologists in Private Practice (bng) together with Roche. Of those, 1100 patients were treated in 240 centers according to consensus recommendations (GT-1/4/5/6 for 48±4 wks, GT-2/3 for 24±2 wks) and were documented with a complete dataset concerning virological responses of week 4, 12, end of treatment and 24 weeks after cessation of treatment. For these patients descriptive, univariate and multivariate analyses were performed to determine factors associated with SVR. Patients with HIV or HBV co-infection and after liver transplantation were excluded. Additionally patients who discontinued therapy due to compliance issues or were lost to follow-up, were excluded from analysis.

Patients had undergone treatment with peginterferon alfa-2a (40KD) (PEGASYS®, Roche, Welwyn Garden City, United Kingdom) plus RBV (Copegus®, Roche, Grenzach-Wyhlen, Germany). As this study was non-interventional, dosing and treatment duration of therapy were not controlled but reflected the clinical practice of the physician and/or treatment centre at the time the patient was treated. Patient selection for submission to the database was entirely at the clinician’s discretion; no restrictive parameters were set other than diagnosis of CHC; absence of child B/C cirrhosis and age ≥18 years. Data collection was performed via online eCRF. Baseline parameters included sex, age, race, weight/body mass index (BMI), duration and mode of infection, stage of fibrosis, treatment history of CHC infection; weight loss during therapy; virological parameters included HCV genotype and viral load at different times on treatment and in the follow-up phase. Following definitions were used: rapid virological response (RVR): HCV RNA ≤50 IU/mL and/or HCV RNA qualitatively undetectable at week 4; early virological response (EVR): ≥2-log10 drop in HCV RNA and/or HCV RNA ≤50 IU/mL and/or HCV RNA qualitatively undetectable at week 12; end of treatment response (EOT), and sustained virological response (SVR): HCV RNA ≤50 IU/mL and/or HCV RNA undetectable after 24 weeks of follow-up. The study was approved by public health authority and ethics committees.

The statistical analysis was descriptive to reflect the clinical routine as intended by the clinicians. Summary statistics (mean, median, standard deviation, 25th percentile, 75th percentile, minimum, maximum, number of values) or frequencies and proportions were assessed for all collected parameters. For continuous variables as age and weight loss, receiver operating characteristic analyses estimated the best cut-off point for sustained virological response. The cut-off points found for the continuous variables were used for generating the corresponding categorical variables. Associations of various factors with sustained virological response were analyzed with Fisher’s exact test.

Only those variables which were significant in the univariate analysis were included in the multivariate analysis. Analyses were calculated with SPSS for Windows Release 12.0.1 and Testimate Version 6.4.27.

RESULTS

Baseline characteristics

The Demographics of cohort are shown in table 1.

In both genotype groups the majority of patients were males. 44% of patients got HCV infected by intravenous drug use, 16.3%

Table 1 Baseline characteristics.

<table>
<thead>
<tr>
<th>Genotype 1, 4, 5, 6</th>
<th>Genotype 2, 3</th>
<th>All genotypes</th>
</tr>
</thead>
<tbody>
<tr>
<td>N</td>
<td>717</td>
<td>263</td>
</tr>
<tr>
<td>sex (male/female, %)</td>
<td>57.5/42.5</td>
<td>64.2/35.8</td>
</tr>
<tr>
<td>Age (mean ± SD in yrs)</td>
<td>46.4 ± 11.7</td>
<td>39.6 ± 10.1</td>
</tr>
<tr>
<td>Weight (mean ± SD in kg)</td>
<td>76.3 ± 14.6</td>
<td>75.7 ± 14.5</td>
</tr>
<tr>
<td>BMI (mean ± SD in kg/m²)</td>
<td>25.7 ± 4.3</td>
<td>25.1 ± 4.1</td>
</tr>
<tr>
<td>Duration of infection (yrs)</td>
<td>15.3</td>
<td>11.3</td>
</tr>
<tr>
<td>Viral load (IU/mL) (≤ 400,000 IU/mL)</td>
<td>31.6</td>
<td>49.4</td>
</tr>
<tr>
<td>HCV genotype (n)</td>
<td>700/341/1/2</td>
<td>29/22/14/3/1</td>
</tr>
<tr>
<td>Ethnical background</td>
<td>Caucasian</td>
<td>711</td>
</tr>
<tr>
<td>African</td>
<td>10</td>
<td>4</td>
</tr>
<tr>
<td>Asian</td>
<td>13</td>
<td>14</td>
</tr>
<tr>
<td>Other</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

†Thrombophlebitis, heparin, warfarin, or other anticoagulants.

Mode of infection (n)

Transfusion | 150 | 29 | 179 |
Intravenous Drug Use | 263 | 221 | 484 |
Sexually | 26 | 15 | 41 |
Tattoo, piercing | 15 | 6 | 21 |
Needle stick | 8 | 3 | 11 |
Surgery | 52 | 12 | 64 |
Medical personal | 12 | 2 | 14 |
Dialysis | 0 | 0 | 3 |
Other | 7 | 7 | 14 |
Unknown | 244 | 80 | 324 |
after receiving blood products and 3.7% as a sexual transmitted disease. However, in 30.3% of patients the mode of infection was unclear or the patients have not been asked. Almost 63.6% of patients presented genotype 1 at baseline, followed by 28.2% with genotype 3, 4.8% with genotype 2 and 3.1% with genotype 4. As expected patients with genotype 5 and 6 are underrepresented. In GT 1/4/5/6 patients 68.4% had a high viral load (HCV-RNA > 400,000 IU/mL), and in GT 2/3 50.6%. Weight and BMI at baseline were comparable between genotype groups. The duration of infection varied between the two groups between 11 and 15 years. Since the study was as a non-interventional study no histological criteria have been set in the first place. Only 213 out of 1,100 patients underwent liver biopsy prior to treatment of them 16.5% were categorized F3/4 according to Desmet-Scheuer. 54.5% of patients had no fibrosis (F0-1), 40.4% showed a F2-3 fibrosis and only 5.2% of patients presented histologically proven cirrhosis prior to therapy.

Almost 89% of patients in this cohort were treatment naïve. 6.8% showed a non-response to and 4.2% a relapse after prior therapy. Only 0.4% of patients got reinfected after prior successful treatment.

**Treatment outcome and factors associated with SVR**

Overall, 524 (47.6%) out of 1,100 patients achieved SVR, 39.3% of patients with genotype 1/4/5/6 and 64.5% of patients with genotype 2/3.

RVR was achieved in 20.6% of GT-1/4/5/6 and 82.1% of GT-2/3 patients, EVR in 64.7% of GT-1/4/5/6 and 75.8% of GT-2/3 patients. In week 4 positive predictive value (PPV) of patients with GT-1/4/5/6 with RVR was 69.1% in regard to achieving SVR. Patients with non-RVR resulted in a negative predictive value (NPV) in 67.8% of patients. For patients with GT-2/3 the PPV was also 69.1% and the NPV was 50.0%.

Week 12 demonstrated a PPV for EVR concerning SVR in patients with GT-1/4/5/6 and 102 (28.1%) patients to GT-2/3 HCV infection.

During the complete observation time 457 (41.5%) patients discontinued treatment, 355 (48.2%) belonged to the group of GT-1/4/5/6 and 102 (28.1%) patients to GT-2/3 HCV infection.

Until week 4 the discontinuation rate was very low with 11 GT-1/4/5/6 infected patients and 9 patients with GT-2/3 (main reasons were lack of tolerance and desire by the patients). Until week 12, 193 (17.5%) of all patients interrupted treatment, 121 (16.4%) of GT-1/4/5/6 and 72 (19.8%) with GT-2/3. The main reasons of these 193 treatment discontinuations were virological non-response (38.9%), lack of tolerability (9.8%) and wish of the patients to discontinue the treatment (43.5%).

More detailed analysis showed that there were different discontinuation rates in certain main categories concerning both weight loss groups: Patients with weight loss < 2 kg vs ≥ 2 kg abort in 35.9% vs 40.9% caused to virological non-response, in 16.7% vs 5.2% caused to lack of tolerability, in 9.0% vs 3.5% caused to compliance issues and in 37.2% vs 47.8% caused to desire of patients.

**Univariate and multivariate regression analysis**

Table 3 shows the results of univariate and multivariate regression analyses of factors described above. Factors in the univariate analysis were initial body weight, age, ethnicity (Caucasians vs others), genotypes (1,4,5,6 vs 2,3), stage of fibrosis (0-2 vs ≥ 3-4) according to Desmet-Scheuer, RVR, EVR and ≥ 2 kg of weight loss during treatment. The univariate analysis showed ages (<45 vs ≥ 45 years), GT (1/4/5/6 vs 2,3), RVR, EVR and weight loss (<2 vs ≥ 2 kg) as significant predictors for SVR.

Multivariate analysis included all significant parameters of univariate analysis. EVR [p=0.001, OR=4.816 (CI95 2.503-9.621)], RVR [p=0.001, OR = 3.343 (CI95 2.523-4.430)] age [p=0.001, OR=0.472 (CI95 0.358-0.623)] and weight loss [p=0.003, OR=1.601 (CI95 1.173-2.184)] were significant predictors, but not genotypes.

In summary, in addition to well-known predictive factors of virological response after PEG/RBV treatment, weight loss should be considered as an easy to measure and reliable positive predictive factor.

**DISCUSSION**

This large cohort study of patients treated with PEG-IFN alfa-2a plus RBV in clinical practice confirmed and extended existing data on the characterization of independent positive predictors of SVR including age, RVR, EVR and weight loss under therapy. Besides weight loss, these predictive factors have been identified previously under study

---

**Table 2** Response rate (RVR, EVR and SVR) in regard to genotype and weight loss.

<table>
<thead>
<tr>
<th>Weight loss</th>
<th>GT 1/4/5/6</th>
<th>GT 2/3</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;2 kg</td>
<td>&gt;2 kg</td>
<td>&lt;2 kg</td>
</tr>
<tr>
<td>RVR</td>
<td>12.4%</td>
<td>23.6%</td>
</tr>
<tr>
<td>EVR</td>
<td>51.5%</td>
<td>69.4%</td>
</tr>
<tr>
<td>SVR</td>
<td>25.3%</td>
<td>44.4%</td>
</tr>
</tbody>
</table>

**Withthöft T et al. Weight loss as a predictor for SVR in HCV treated patients**

© 2014 ACT. All rights reserved.
conditions with strict in- and exclusion criteria. Although weight loss is easy to measure its value as a predictive factor is rarely described in literature. Most studies are retrospective and results are partly inconsistent. Lindsay et al conducted a retrospective analysis of the HALT-C trial and showed that besides other factors less reduction in body weight was associated with null response. In a small retrospective study with 45 patients, Suwantarit et al found that after multivariate analysis genotype non-1, weight loss >5kg during therapy and leucocytes<3000/µL at EOT were significant predictive factors of SVR.

In a retrospective study with 126 patients with the aim to identify predictors of weight loss on treatment at week 24 and of week 24 after end of treatment no significant correlation of increased weight loss and improved antiviral response to treatment outcome (SVR) was found.

The approach chosen in this study was different to our study where predictive factors for SVR were evaluated. Additionally it was not differentiated between genotypes due to the smaller sample size.

Our data set was gathered prospectively and represents information of CHC treatment under “real world”-conditions of a very large cohort.

Data from randomised clinical trials form the basis of treatment guidelines and inform clinicians and healthcare workers on individual patient management. However, clinical trial populations by necessity are defined and restricted and the trial process itself involves the use of clear protocols, for example concerning dose reductions or treatment. The support and monitoring of patients during a clinical trial is also likely to improve compliance with treatment, which is an important contributor to a successful outcome. As such, the clinical trial situation may not fully reflect real-life clinical practice with its more diverse, complex patient population, variability in access and support mechanisms, and possibly less well defined protocols in routine practice. Cohort studies such as the current study are therefore important to assess how well clinical trial data transfers into routine practice. In the current study, 47.6% of patients overall achieved SVR, 39.3% of patients with genotypes 1,4,5,6 and 64.5% of patients with genotypes 2/3. This compares favourably with pivotal clinical trials which reported rates of 42-46% in genotype 1-infected patients and 76-82% in genotype 2/3-infected patients. The findings of this study are also in line with those from other retrospective analyses of HCV-infected patients treated in routine clinical practice where overall SVR rates of 49-66% have been reported, with rates of 37-61% in genotype 1-infected and approximately 70% of genotype 2-infected patients (20-23%), GT 1-4. Treatment with PEG plus RBV was well tolerated in the current study. Rates of discontinuation of therapy were similar to that reported in pivotal clinical trials (14-21%) and within the range reported by other cohort studies of PEG plus RBV in clinical practice (11-33%).

Table 3 Regression analysis for the predictors of SVR.

<table>
<thead>
<tr>
<th>Predictors of SVR</th>
<th>Univariate analysis</th>
<th>Multivariate analysis</th>
<th>p-value</th>
<th>OR</th>
<th>95% C.I. of OR</th>
<th>p-value</th>
<th>OR</th>
<th>95% C.I. of OR</th>
<th>N</th>
</tr>
</thead>
<tbody>
<tr>
<td>Initial body weight (kg)</td>
<td>0.056</td>
<td>0.113</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age (&lt; 45 yrs vs ≥ 45 yrs)</td>
<td>0.000</td>
<td>0.000</td>
<td>0.379</td>
<td>0.297-0.483</td>
<td>0.000</td>
<td>0.472</td>
<td>0.358-0.623</td>
<td>1100</td>
<td></td>
</tr>
<tr>
<td>Caucasians vs others</td>
<td>0.169</td>
<td>0.138</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Genotypes 2, 3 vs 1, 4, 5, 6</td>
<td>0.000</td>
<td>0.181</td>
<td>2.796</td>
<td>2.154-3.629</td>
<td>0.000</td>
<td>3.343</td>
<td>2.523-4.430</td>
<td>1100</td>
<td></td>
</tr>
<tr>
<td>RVR</td>
<td>0.000</td>
<td>0.000</td>
<td>4.590</td>
<td>3.545-5.944</td>
<td>0.000</td>
<td>4.816</td>
<td>3.503-6.621</td>
<td>1100</td>
<td></td>
</tr>
<tr>
<td>EVR</td>
<td>0.000</td>
<td>0.003</td>
<td>5.776</td>
<td>4.289-7.778</td>
<td>0.000</td>
<td>1.891</td>
<td>1.443-2.477</td>
<td>1100</td>
<td></td>
</tr>
<tr>
<td>Weight loss ≥ 2 kg vs &lt; 2kg during therapy</td>
<td>0.000</td>
<td>0.003</td>
<td>1.891</td>
<td>1.443-2.477</td>
<td>0.000</td>
<td>1.601</td>
<td>1.173-2.184</td>
<td>1100</td>
<td></td>
</tr>
</tbody>
</table>

1 best cut by ROC-analysis.

They were identified in numerous clinical studies and were estimated as positive predictors for SVR.

In this study, age older than 45 years was either in a univariate as well as in a multivariate regression analysis a significant negative predictive variable for SVR. In previous studies, age older than 40-45 years has also been shown to be negative predictive factors for SVR following interferon-based therapy in clinical studies and in cohort analyses. Nevertheless weight loss can be used as a reliable predictor even though weight loss is easy to measure its value as a predictive factor is rarely described in literature. Most studies are retrospective and results are partly inconsistent. Lindsay et al conducted a retrospective analysis of the HALT-C trial and showed that besides other factors less reduction in body weight was associated with null response. In a small retrospective study with 45 patients, Suwantarit et al found that after multivariate analysis genotype non-1, weight loss >5kg during therapy and leucocytes<3000/µL at EOT were significant predictive factors of SVR.

In a retrospective study with 126 patients with the aim to identify predictors of weight loss on treatment at week 24 and of week 24 after end of treatment no significant correlation of increased weight loss and improved antiviral response to treatment outcome (SVR) was found.

The approach chosen in this study was different to our study where predictive factors for SVR were evaluated. Additionally it was not differentiated between genotypes due to the smaller sample size.

Our data set was gathered prospectively and represents information of CHC treatment under “real world”-conditions of a very large cohort.

Data from randomised clinical trials form the basis of treatment guidelines and inform clinicians and healthcare workers on individual patient management. However, clinical trial populations by necessity are defined and restricted and the trial process itself involves the use of clear protocols, for example concerning dose reductions or treatment. The support and monitoring of patients during a clinical trial is also likely to improve compliance with treatment, which is an important contributor to a successful outcome. As such, the clinical trial situation may not fully reflect real-life clinical practice with its more diverse, complex patient population, variability in access and support mechanisms, and possibly less well defined protocols in routine practice. Cohort studies such as the current study are therefore important to assess how well clinical trial data transfers into routine practice. In the current study, 47.6% of patients overall achieved SVR, 39.3% of patients with genotypes 1,4,5,6 and 64.5% of patients with genotypes 2/3. This compares favourably with pivotal clinical trials which reported rates of 42-46% in genotype 1-infected patients and 76-82% in genotype 2/3-infected patients. The findings of this study are also in line with those from other retrospective analyses of HCV-infected patients treated in routine clinical practice where overall SVR rates of 49-66% have been reported, with rates of 37-61% in genotype 1-infected and approximately 70% of genotype 2-infected patients (20-23%), GT 1-4. Treatment with PEG plus RBV was well tolerated in the current study. Rates of discontinuation of therapy were similar to that reported in pivotal clinical trials (14-21%) and within the range reported by other cohort studies of PEG plus RBV in clinical practice (11-33%).

Table 3 Regression analysis for the predictors of SVR.
in regions where it is not possible to conduct more often HCV RNA determinations.

Clinicians were fully supported in supplying data to this data set and selection bias was avoided. However, each treatment center confirmed that all patients treated within the pre-specified observational period were included in the data collection, and that there was no patient selection for documented patients. As with all observational studies, another potential bias may be caused by missing data because only data collected by routine and documented by the clinicians in the patient records were available for inclusion in the study. In spite of this, an important advantage of the present study is the large number of patients included.

CONCLUSION

Weight loss is considered as a negative consequence of PEG/ribavirin therapy. But being easy to measure it represents a suitable additional reliable on-treatment predictive factor for estimating the success of antiviral therapy. It is useful to recognize the need for eventual changing treatment protocol in patients without weight loss in very early stage of treatment.

ACKNOWLEDGEMENTS

We express our gratitude to all patients, their families, the investigators and the nurses who participated in the ongoing study.

STATEMENT OF INTEREST

Thomas Witthöft has received an honorarium for the current study, travel grants and speaker fees from MSD and Roche. Peter Buggisch has received fees for serving as a speaker, and an advisory board member for Roche, MSD, BMS and Novartis. Dietrich Hueppe has served as a speaker, a consultant and an advisory board member for Roche, MSD, Gilead, Bristol-Myers Squibb and Novartis. Christine John has received an honorarium and expenses from Roche for the current study and has received travel grants from Roche, Gilead and Bristol-Myers-Squibb. Axel Baumgarten has served as a speaker, a consultant and an advisory board member for Roche, Tibotec, Bristol-Myers Squibb, GSK, Pfizer, Gilead, and MSD and has research funding from Roche, Tibotec, GSK, Pfizer, MSD, and Abbott for clinical study projects. Thomas Lutz received fees as speaker from Roche, BMS, Gilead, Boehringer-Ingelheim, MSD, GKV and as board member from Gilead, Abbott, Boehringer-Ingelheim, Janssen-Cilag and travel grants from Roche, Gilead, Boehringer, BMS, Abbott and Janssen-Cilag. Albrecht Stoehr has served a speaker and advisory board member for Abbott, Gilead, Roche, Janssen-Cilag, MSD and GKV and as board member from Roche, Gilead, Boehringer, BMS, Abbott and Janssen-Cilag. Albrecht Stoehr has served a speaker and advisory board member for Gilead, Roche, Janssen-Cilag, MSD, and ViViV. Ulrich Alshuth is employee of Roche Pharma AG. Stefan Mauss has served as a consultant or received honoraria from Roche, BMS, AbbVie, Gilead, Janssen-Cilag, Boehringer-Ingelheim, MSD, and has received grants from Roche, BMS, Gilead, AbbVie, Janssen-Cilag, and is shareholder of Medivir. All other authors have no conflicts of interest to declare.

This study has been presented as a poster at Hepdart 2011 on Kauai, USA.

This study was funded by Roche Pharma AG. The authors had complete access to the data that supports this article. Funding for editorial support was also provided by Roche Pharma AG.

REFERENCES

1 Mc Hutchinson JG. Understanding hepatitis C. Am J Manag Care 2004; 10: S21-S29
10 Yee HS, Chang MF, Pocha C, Lim J, Ross D, Morgan TR, Monto A; Department of Veterans Affairs Hepatitis C Resource Center Program; National Hepatitis C Program Office. Management and treatment of hepatitis C virus infection: recommendations from the Department of Veterans Affairs Hepatitis C Resource Center program and the National Hepatitis C Program office. Am J Gastroenterol 2006; 101: 2360-2378
Weight loss as a predictor for SVR in HCV treated patients
Withöft T et al. Weight loss as a predictor for SVR in HCV treated patients


Peer reviewers: Kazuaki Nishio, 1-5-8, Hatanodai, Shinagawaku, Tokyo, 142-8666 Japan; Neda Svirtlih, Professor, MD, PhD, Department for Hepatitis, Medical Faculty, University of Belgrade, Clinic for Infectious and Tropical Diseases, Clinical Centre of Serbia, Bulevar oslobodjenja 16, Belgrade, 11000, Serbia.