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HDV at EASL 2026

An Evolving Clinical Landscape



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Clinical Research in HDV: A Major Turning Point in Patient Management

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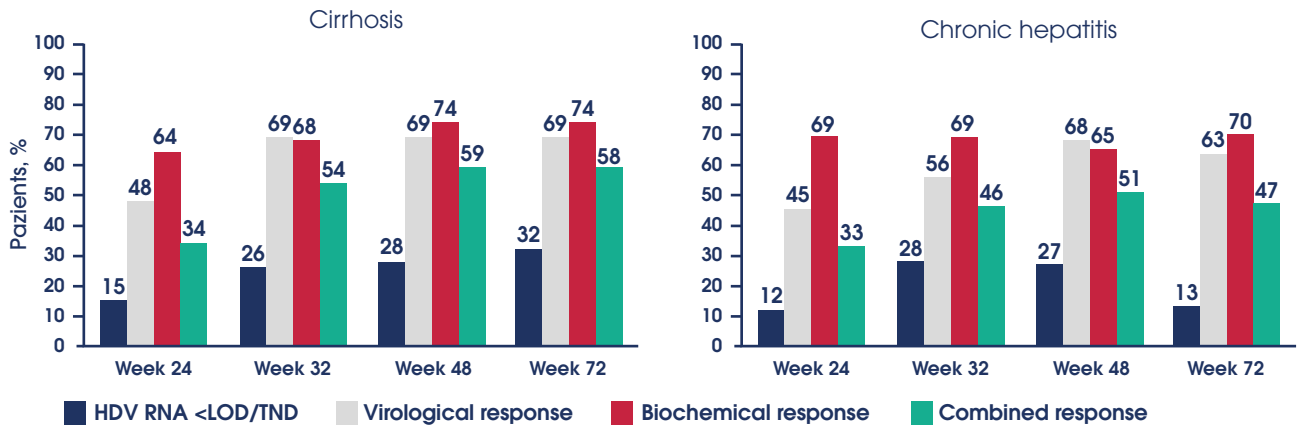
Introduction

At the EASL Congress held in late May 2026, several important new studies on hepatitis D virus (HDV) infection were presented. In particular, 4-year real-world efficacy and safety data on bulevirtide (BLV) monotherapy were reported for the first time, as well as data on the effects of treatment on long-term outcomes, including hepatic decompensation and hepatocellular carcinoma (HCC). In addition, validation studies assessing potential stopping rules in patients who achieved undetectable HDV RNA during prolonged BLV treatment were discussed. New evidence on BLV 10 mg monotherapy reinforces its long-term efficacy and consistent safety profile.

Efficacy and safety of BLV monotherapy in patients with and without cirrhosis

A total of 494 patients with chronic hepatitis D (CHD), with or without cirrhosis, who were treated with BLV 2 mg monotherapy were enrolled in the prospective, multicenter Italian D-SHIELD study (1).
The proportions of patients achieving virological, biochemical, and combined responses, as well as undetectable HDV RNA, progressively increased over time, with no significant differences between patients with and without cirrhosis (*Figure 1*).

Figure 1 D-SHIELD study: virological, biochemical, combined responses and HDV RNA negativity in patients with and without HDV-related cirrhosis



LOD/TND, Limite di rilevabilità/HDV negatività

Anolli MP, et al. J Hepatol 2026;84S1:S938.

Rates of virological breakthrough (VBK), occurring in approximately 13–16% of patients, were also similar in the two groups. No significant differences in the safety profile of BLV were observed between patients with and without cirrhosis, except for higher bile acid levels in patients with cirrhosis both before and during treatment.

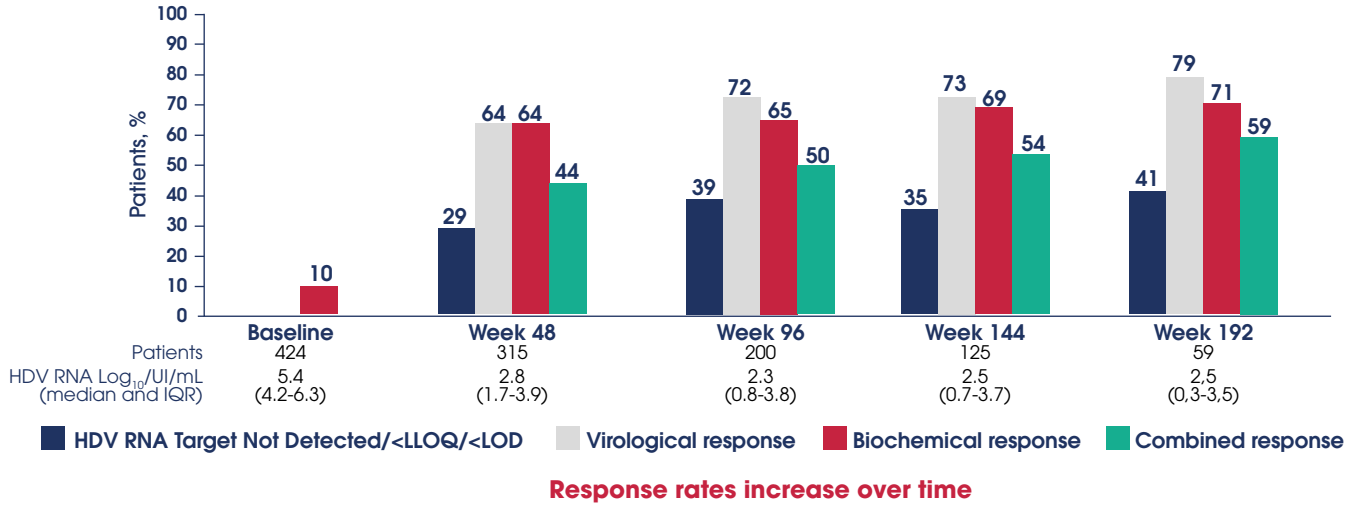
Efficacy and safety of BLV monotherapy administered for up to 4 years

A total of 424 patients with compensated cirrhosis enrolled in the retrospective multicenter SAVE-D study were treated with BLV monotherapy for up to 4 years (2). Virological and biochemical response rates progressively increased over time, including between the third and fourth year of treatment (*Figures 2 & 3*).

The study also evaluated the prevalence of VBK, defined as an increase in viremia of at least 1 log compared with the previous time point. Approximately 20% of patients experienced VBK during the first 3 years of treatment;

Figure 2 SAVE-D study: BLV 2 mg monotherapy for up to 4 years in compensated cirrhosis

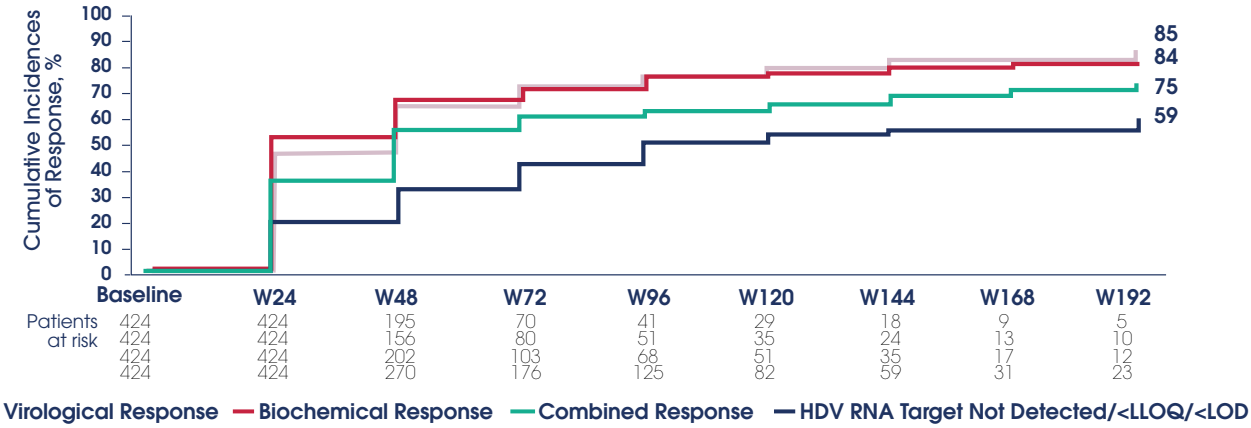
Retrospective multicenter EU/Canada study in 424 compensated cirrhotics



Degasperi E, et al. J Hepatol 2026;84S1:S43.

Figure 3 SAVE-D study: response rates to BLV monotherapy for up to 4 years

4-year cumulative incidences of responses to BLV monotherapy



Degasperi E, et al. J Hepatol 2026;84S1:S43.

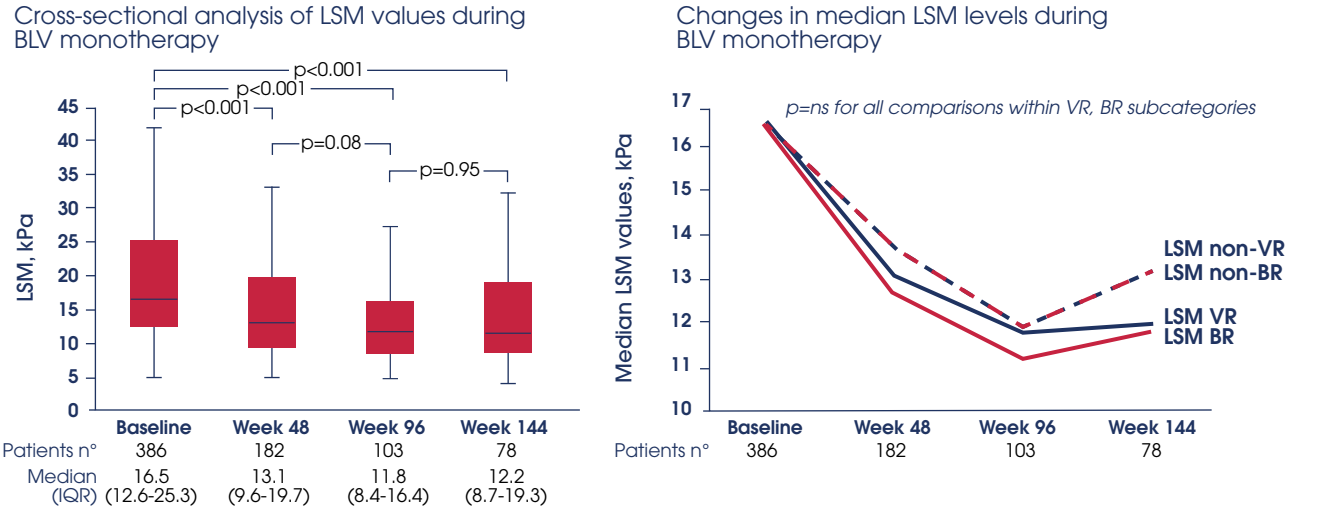
however, in many cases, this virological event was transient and was not associated with an increase in transaminase levels. The safety profile remained favourable, with no serious or unexpected adverse events (AEs).

Non-invasive markers of fibrosis in patients with cirrhosis treated with BLV monotherapy

The SAVE-D study also assessed changes over time in non-invasive markers of liver fibrosis (2).

Liver stiffness measured by transient elastography (FibroScan) progressively and significantly decreased during the first 3 years of BLV monotherapy, irrespective of virological or biochemical response (*Figure 4*). The figure also summarizes changes over time in clinically significant portal hypertension (CSPH) categories.Overall, approxima-

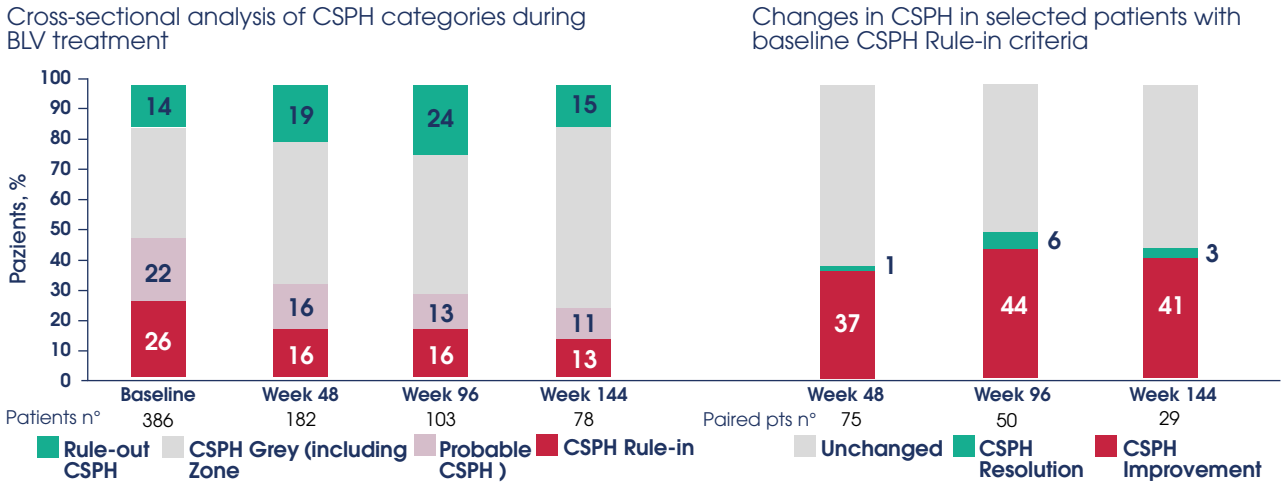
Figure 4 SAVE-D study: liver stiffness changes during treatment



LSM: Liver Stiffness Measurement; VR: Virological Response; BR: Biochemical Response

Degasperi E, et al. J Hepatol 2026;84S1:S43.

Figure 5 SAVE-D study: changes in CSPH categories during treatment according to non-invasive criteria



CSPH Rule in: LSM >25 kPa; CSPH Rule-out: LSM ≤15 kPa + PLT ≥150; Probable CSPH: LSM >20 to ≤25 kPa + PLT <150 or LSM >15 to ≤20 + PLT <110
CSPH Improvement: PLT >150 if thrombocytopenia at baseline (PLT <150) or LSM decrease ≥30% from baseline or LSM decrease to <20 kPa if baseline LSM >25 kPa
CSPH Resolution: LSM decrease <15 kPa and PLT normalization (>150) or LSM decrease <10 kPa regardless of PLT
CPSH: Clinically Significant Portal Hypertension

Degasperi E, et al. J Hepatol 2026;84S1:S43.

tely 35% of patients with a pre-treatment non-invasive diagnosis of CSPH based on liver stiffness ± platelet count showed an improvement according to Baveno criteria (*Figure 5*). However, most of these improvements occurred during the first year of treatment.

Incidence of hepatic decompensation and HCC in patients with cirrhosis treated with BLV monotherapy

A case-control study assessed the 3-year cumulative incidence of hepatic decompensation and HCC in a group of approximately 300 patients with cirrhosis treated with BLV monotherapy and in a control group of untreated patients with cirrhosis (3). The control group was matched to the treated group for a substantial number of variables, using advanced statistical methods (*IPTW-adjusted Cox regression analysis and an IPTW-adjusted competing-risks regression model*) to minimize baseline differences between the two groups.

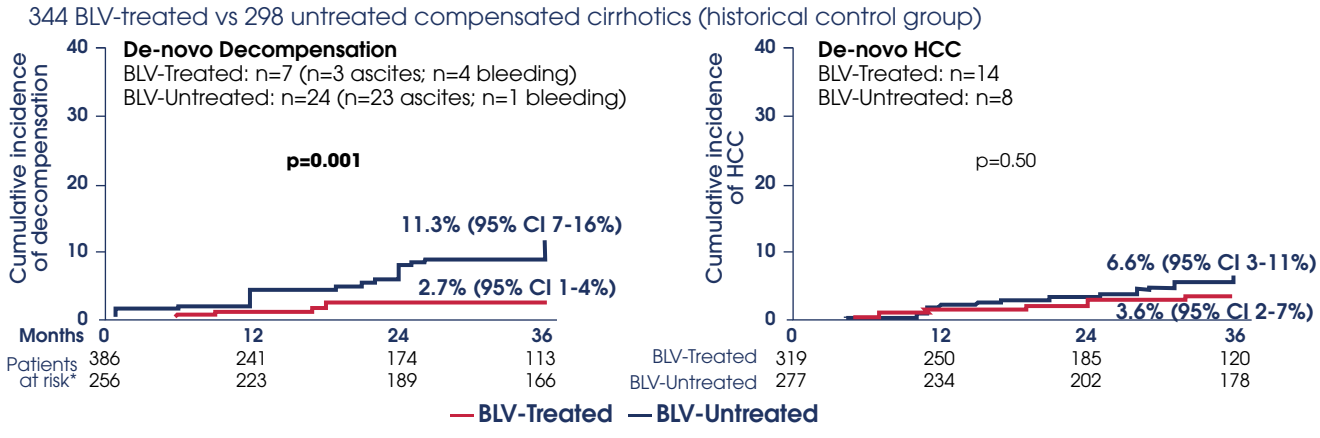
The 3-year cumulative incidence of hepatic decompensation was 2.7% in treated patients and 11.3% in controls (p=0.001) (*Figure 6*). By contrast, HCC rates were similar between the two groups (6.6% vs 3.6%, p=0.50). BLV monotherapy reduced the risk of hepatic decompensation at 3 years by 84% (95% CI, 60%–94%) but did not reduce the risk of HCC.

The effect of BLV treatment on hepatic decompensation and HCC was also evaluated at 4 years in the SAVE-D study, although a control group of untreated patients is not yet available for this analysis (2). During 4 years of BLV treatment, 29 liver-related events were observed, including 9 episodes of hepatic decompensation and 20 cases of HCC (*Figure 7*).

The 4-year cumulative incidence rates of hepatic decompensation and HCC were 2.3% and 6.8%, respectively. Notably, no episodes of hepatic decompensation occurred after week 72 of treatment.

Independent pre-treatment predictors were baseline albumin levels for hepatic decompensation and baseline albumin levels, body mass index (BMI), and age for HCC. Eleven patients underwent liver transplantation or died

Figure 6 SAVE-D study: decompensation and HCC in cirrhotic patients treated with BLV monotherapy for up to 3 years

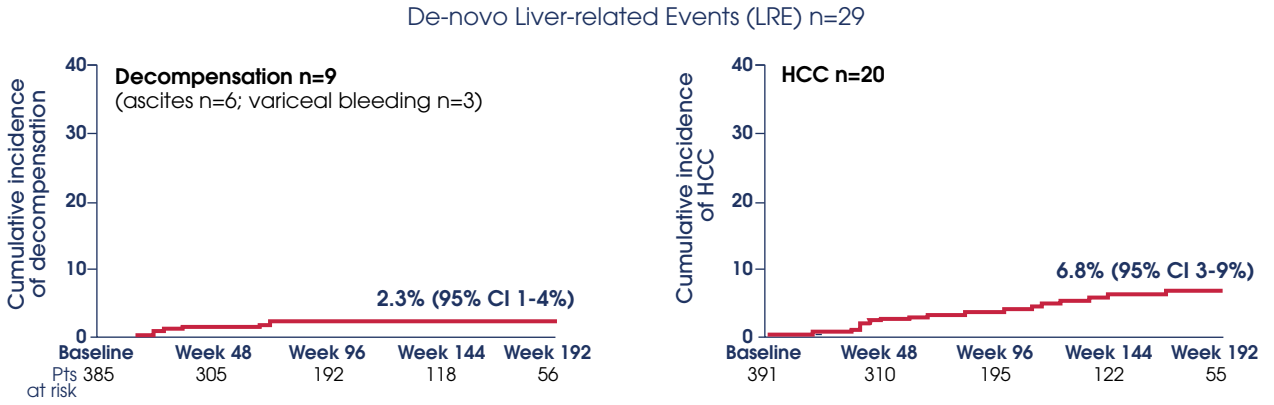


Low rates of decompensation (but not HCC) in BLV-treated cirrhotics

**Patients with previous LREs (decompensation or HCC) and HCC occurring within 6 months from baseline excluded from the analysis.*

Degasperi E, et al. J Hepatol 2026;84S1:S934.

Figure 7 SAVE-D study: decompensation and HCC in cirrhotic patients treated with BLV monotherapy for up to 4 years



Baseline predictors of LRE by Multivariate Cox-regression analysis;
Decompensation: albumin levels**; HCC:** age, BMI, albumin levels

Liver Transplants: n=8 (**HCC in 6; ESLD in 2**); Deaths: n=5 (liver-related in 3)

ESLD, End-state liver disease

Degasperi E, et al. J Hepatol 2026;84S1:S934.

from liver-related causes: HCC was responsible for 7 of these cases and hepatic decompensation for 4.
For the first time, in contrast to all studies published over the past 40 years in untreated patients with HDV, a change in the pattern of complications has been demonstrated in BLV-treated patients: most complications, and consequently most indications for liver transplantation, were related to the development of HCC rather than hepatic decompensation.

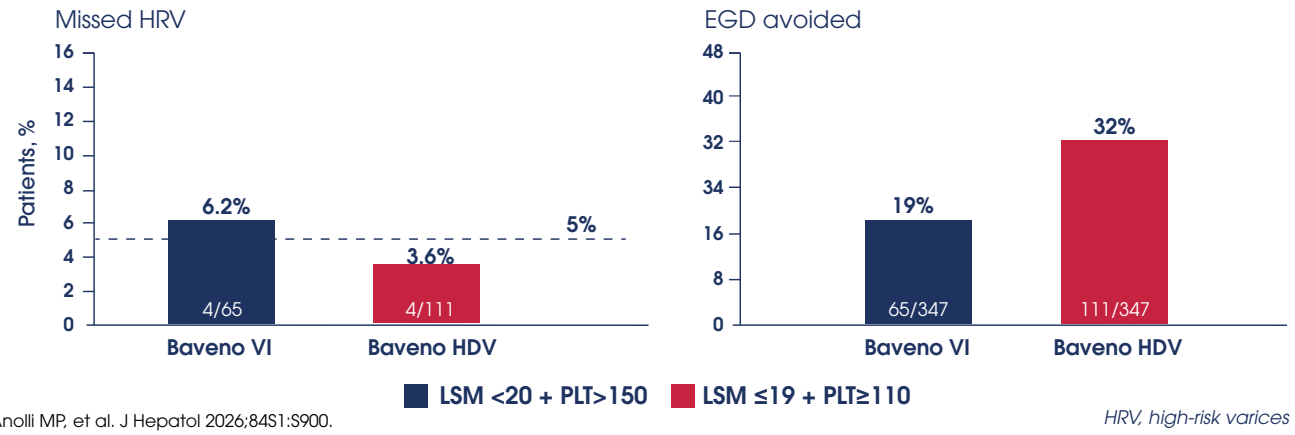
Artificial Intelligence to investigate HDV and rare events

These two cohorts were also used to develop and validate an Artificial Intelligence (AI) model based on the generation of synthetic patient data (4).
Synthetic cohorts of BLV-treated and untreated patients showed high fidelity to real-world clinical data and a high level of privacy protection (*CSF, clinical synthetic fidelity*, 90%/94%; *NNDR, nearest neighbor distance ratio*, 0.71/0.85). Propensity score matching identified 624 matched pairs from 3,000 synthetic data samples per group while maintaining consistent SAFE metrics. The 3-year cumulative incidences of hepatic decompensation and HCC were similar in real and synthetic patients in both the BLV-treated and untreated groups: hepatic decompensation 2.7% vs 4.2% in real vs synthetic BLV-treated patients, and 11.3% vs 10.0% in real vs synthetic untreated patients; HCC 6.6% vs 8.5% in real vs synthetic BLV-treated patients, and 3.6% vs 4.8% in real vs synthetic untreated patients. For hepatic decompensation, rates were 2.7% versus 4.2% in BLV-treated patients and 11.3% versus 10.0% in untreated patients.
Overall, high-fidelity individual-level synthetic data accurately reproduced the clinical outcomes of patients with CHD and cirrhosis, both in those treated with BLV and in untreated patients. This approach could provide a means of overcoming some of the challenges associated with clinical research in rare diseases.

Validation of Baveno criteria for avoiding EGD in untreated patients with cirrhosis

A total of 347 untreated patients with HDV-related cirrhosis, for whom both liver stiffness measurements (LSM) and esophagogastroduodenoscopy (EGD) data were available, were included from the SAVE-D and D-SHIELD studies (5). The Baveno VI–VII criteria for avoiding EGDs in patients with LSM <20 kPa and platelet count >150,000 x 10^9/L were suboptimal in this population because of the high proportion of patients excluded from EGD despite having high-risk varices (6.2%) and a low proportion of EGDs that could be avoided (19%) (*Figure 8*).
Modification of the Baveno criteria using HDV-specific cutoffs (LSM ≤19 kPa and platelet count ≥110,000 x 10^9/L; Baveno-HDV) not only reduced the number of misclassified patients (only 3.6%) but also increased the number of EGDs that could be avoided (32%).

Figure 8 Identification of patients with cirrhosis in whom EGD can be avoided: performance of the Baveno VI criteria and the new Baveno-HDV criterion



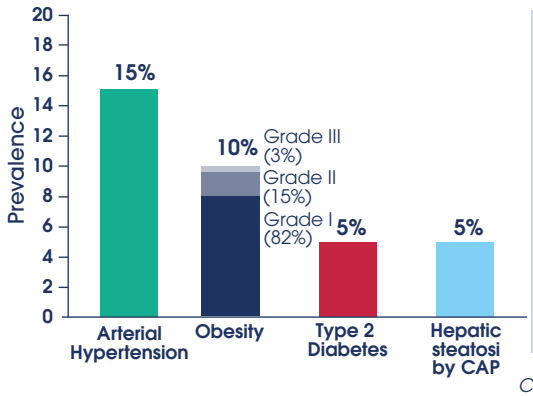
Anolli MP, et al. J Hepatol 2026;84S1:S900.

HRV, high-risk varices

Prevalence of metabolic liver disease in patients with CHD

This study assessed, for the first time, the impact of cardiovascular risk factors and metabolic liver disease (MetHDV) in a large cohort of untreated patients with HDV-related chronic hepatitis or cirrhosis (6) (*Figure 9*).
A total of 828 patients enrolled in the SAVE-D and D-SHIELD studies were evaluated, 84% of whom had cirrhosis. Hypertension, obesity, type 2 diabetes and hepatic steatosis (CAP > 275db) were observed in only 15%, 10%, 5% and 5% of patients, respectively. Overall, the prevalence of cardiovascular risk factors and fatty liver disease in this European large cohort of patients with CHD is lower than that reported for other types of chronic viral diseases.

Figure 9 MetHDV study: prevalence of cardiometabolic risk factors



39 (5%) patients had type 2 diabetes (T2DM), and 126 (15%) patients had arterial hypertension (AHTN), 85 (10%) patients had obesity (defined as BMI ≥30), of whom 13 (15%) grade II obesity (BMI 35-39) and 2 (3%) grade III obesity (BMI≥40).

Analyzing the impact of cardiometabolic risk factors (AHTN, T2DM), BMI and CAP values on responses to BLV, only CAP predicted the achievement of a biochemical response at on-treatment week 48 (OR 1.01 95% CI 1.00-1.02, p=0.02).

CAP, controlled attenuation parameter

Anolli MP, et al. J Hepatol 2026;84S1:S901.

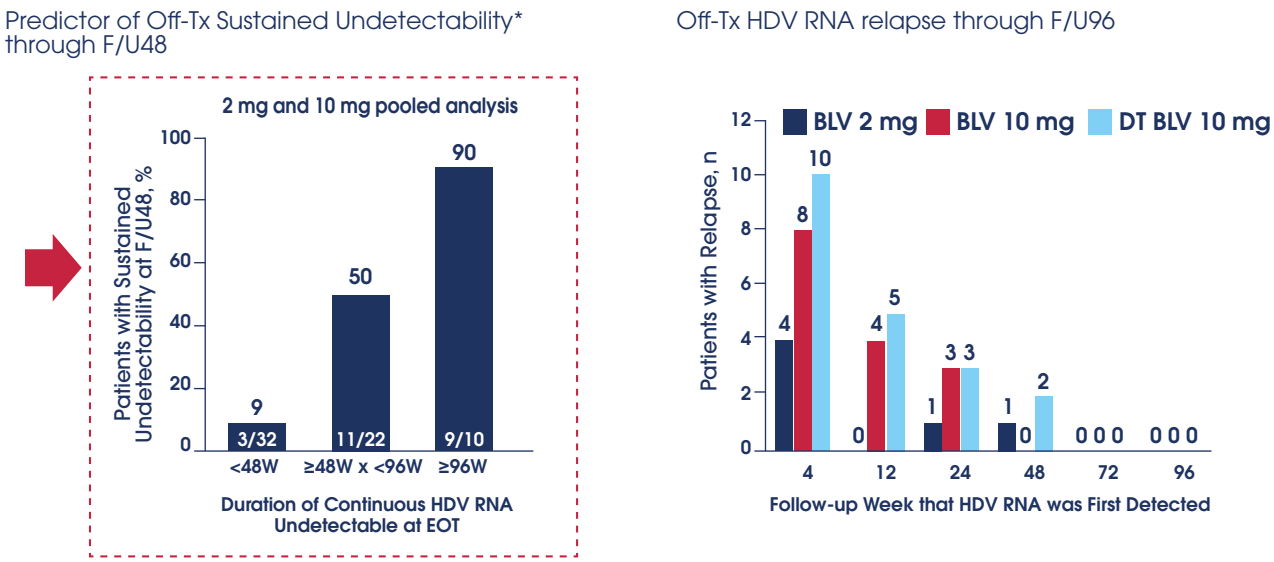
Stopping rules during BLV monotherapy

The possibility of discontinuing BLV monotherapy in selected patients based on virological response was first suggested in 2023 (7). In the reported patient, the discontinuation of BLV monotherapy after 2 years of HDV RNA negativity (i.e. target not detected, TND, as measured by Robogne 2.0) out of a total of a 3-year treatment proved to be effective and sustained over time. Despite staying HBsAg-positive while receiving tenofovir (TDF), the patient remained HDV RNA-negative throughout the subsequent 4 years after BLV discontinuation.

In the MYR301 study (8), the probability of remaining HDV RNA-negative after discontinuation of BLV monotherapy was shown to be directly related to the duration of HDV RNA negativity during treatment (*Figure 10*): 90% of patients who had been HDV RNA-negative for 2 years, 50% among those with suppressed viremia for only 1 year, and only 9% among patients who had been HDV RNA-negative for less than 1 year before treatment discontinuation. In other words, only patients who achieve an early complete virological response (TND), within the first 48 weeks of treatment, and maintain this response through week 144 (the third year of therapy) have a high probability of achieving a sustained complete virological response. However, fewer than 10% of patients treated with BLV 2 mg for 144 weeks achieved an early virological response.

Two important studies addressing this issue were presented at EASL 2026. In a French retrospective study (9), among five patients treated with BLV monotherapy who discontinued treatment after at least 96 weeks, only one (20%) experienced relapse. The second study was the prospective, multicenter BUL-STOP study involving centers in Germany and Italy (10) (*Figure 11*). Among the 21 patients who discontinued BLV monotherapy, five of the six patients who had achieved early HDV RNA negativity, within the first 25 weeks of treatment, maintained a sustained complete virological response after BLV discontinuation. In contrast, most patients (4/6) who

Figure 10 MYR 301 study: undetectable (TND) HDV RNA during off-treatment follow-up



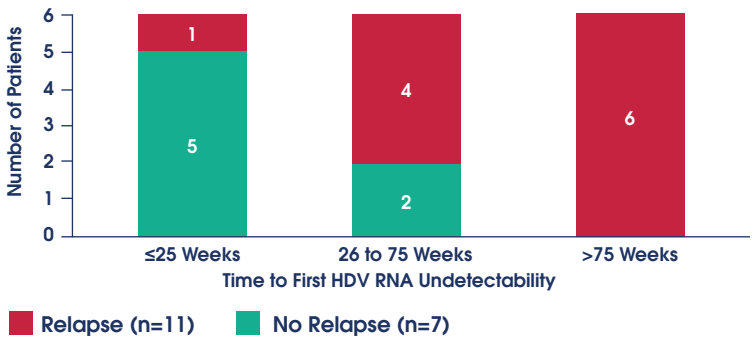
Patients with more than 96 weeks of undetectable HDV RNA on-treatment had the highest rates of sustained, off-treatment undetectable HDV RNA; no patients relapsed after F/U48

2 years HDV RNA=TND on therapy as a potential stopping rule !!

HDV RNA relapse was defined as HDV RNA undetectable at EOT and ≥1 HDV RNA sample during the follow-up period.

Wedemeyer H e Aleman S, et al. EASL 2025. Oral LBO-004

Figure 11 BUL-STOP study: relapse by time to first HDV RNA undetectability



Wedemeyer H, et al. J Hepatol 2026;84S1:S935.

achieved HDV RNA negativity between weeks 26 and 76 experienced relapse.

These data confirm, for the first time in a prospective study of guided BLV discontinuation, that the subgroup of patients achieving a complete virological response within the first year of BLV monotherapy may be able to discontinue treatment after at least 2 years of consolidation therapy.

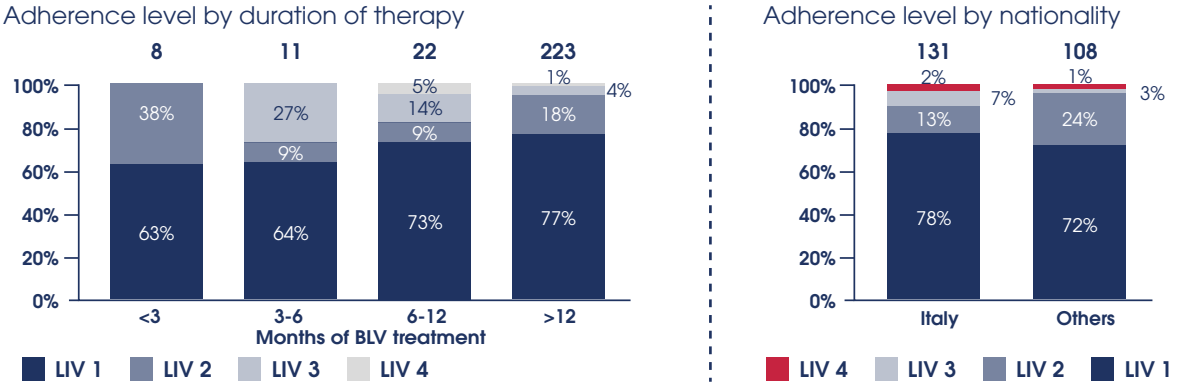
However, implementing this potential stopping rule in clinical practice is currently not recommended for at least three reasons. First, most patients currently receiving BLV have advanced liver disease, frequently cirrhosis with CSPH or Child-Pugh class B cirrhosis, and the risk of post-discontinuation virological and biochemical relapse could have serious clinical consequences. Second, the prospective study included a very limited number of patients – only six – with an early virological response. Third, this strategy must also consider the sensitivity and specificity of the assay used for HDV RNA quantification, as a recent study demonstrated that TND rates vary significantly according to the assay used (11).

Persistence and adherence to BLV therapy in real-world clinical practice

An Italian multicenter study assessed real-world adherence to long-term BLV monotherapy in 273 Italian patients (12). Most patients reported being adherent or highly adherent to BLV treatment, regardless of treatment duration (<1 vs >1 year) and country of birth (Italy vs other countries) (*Figure 12*).

Only around 6% of patients reported suboptimal adherence. These findings extend data recently reported from Germany and Greece at the AASLD 2025 Congress.

Figure 12 Adherence to long-term BLV monotherapy in clinical practice in Italy



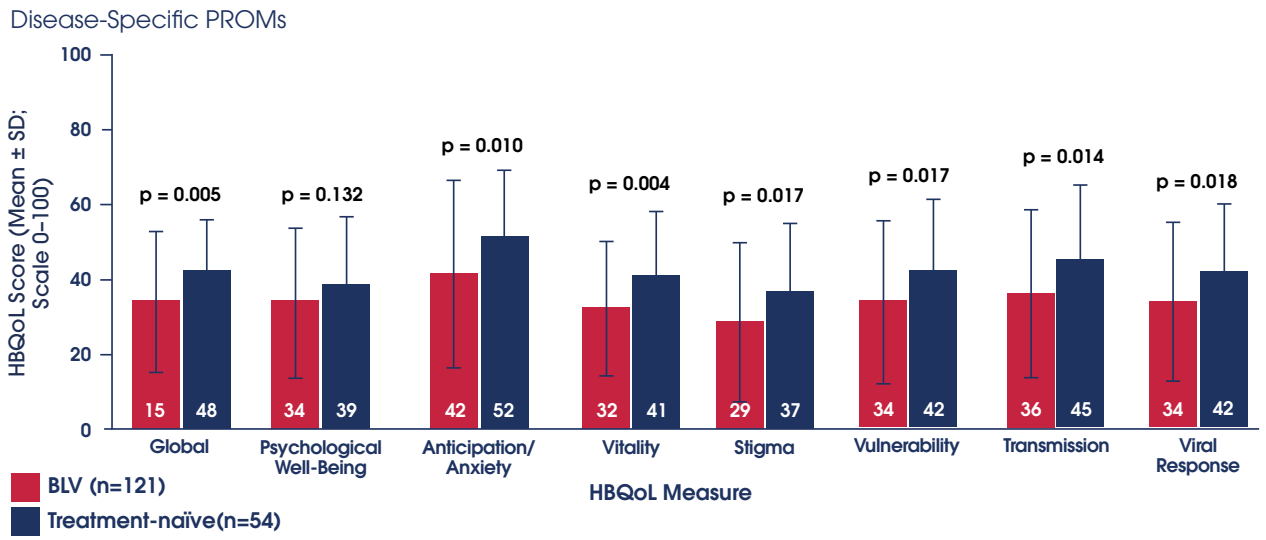
LIV 1: highly adherent; LIV 2: adherent; LIV 3: moderately adherent; LIV 4: occasionally adherent

Badia L, et al. J Hepatol 2026;84S1:S947.

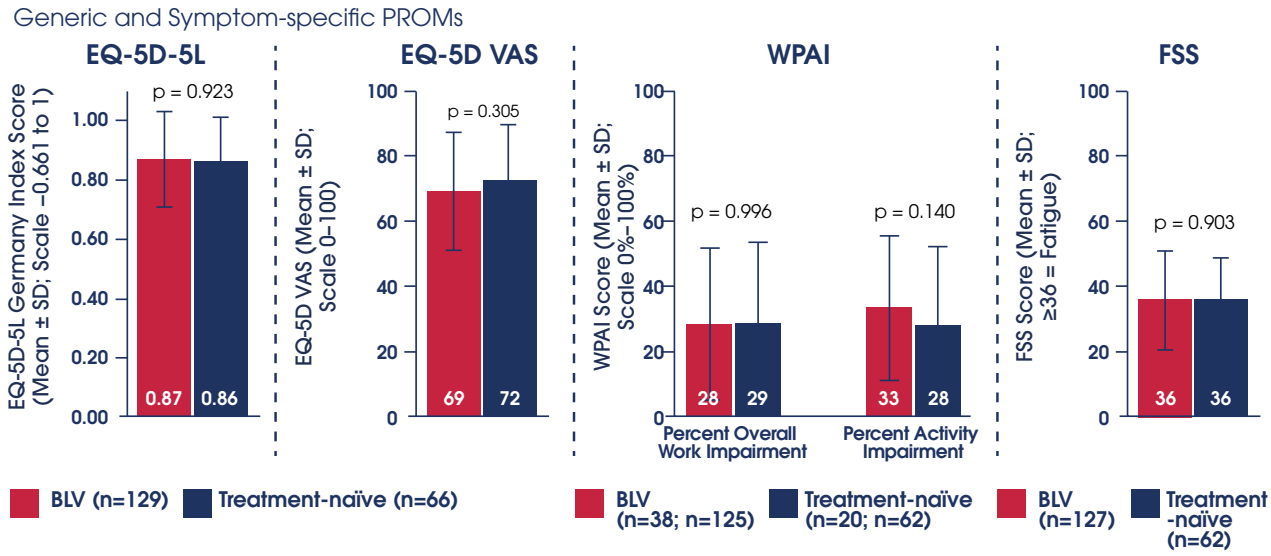
Patient-Reported Outcomes in patients treated with BLV

A European multicenter study evaluated Patient-Reported Outcomes (PROs) in patients with CHD (13). Many of the items related to *Disease-Specific Patient-Reported Outcomes* showed a statistically significant improvement in the 121 patients treated with BLV compared to the 54 untreated control patients. Specifically, improvements were observed in the anticipation/anxiety and vitality domains (*Figure 13*). By contrast, generic or

Figure 13 European multicenter study on PROs in patients treated with BLV monotherapy



HBQoL scale: 0–100, where 0 represents the best possible HRQoL
Mean scores are shown with SD error bars. P-values are from t-tests; bold P-values indicate statistical significance.
HBQoL, Hepatitis B Quality of Life; PROM, patient-reported outcome measure



Mean scores are shown with SD error bars. P values are from t tests.
EQ-5D-5L Germany scale: –0.661 to 1, where 1 represents full health and below 0 represents a health state equivalent to death;
EQ-5D VAS scale: 0–100, where 100 represents the best possible HRQoL; WPAI scale: 0%–100%, where 100% indicates severe impairment;
FSS scores ≥36 indicate fatigue.; EQ-5D-5L, EuroQoL 5-Dimension 5-Level; EQ-5D VAS, EuroQoL 5-Dimension visual analogue scale; FSS, Fatigue Severity Scale; HRQoL, health-related quality of life; PROM, patient-reported outcome measure; WPAI, Work Productivity and Activity Impairment.

Buti M, et al. J Hepatol 2026;84S1:S946.

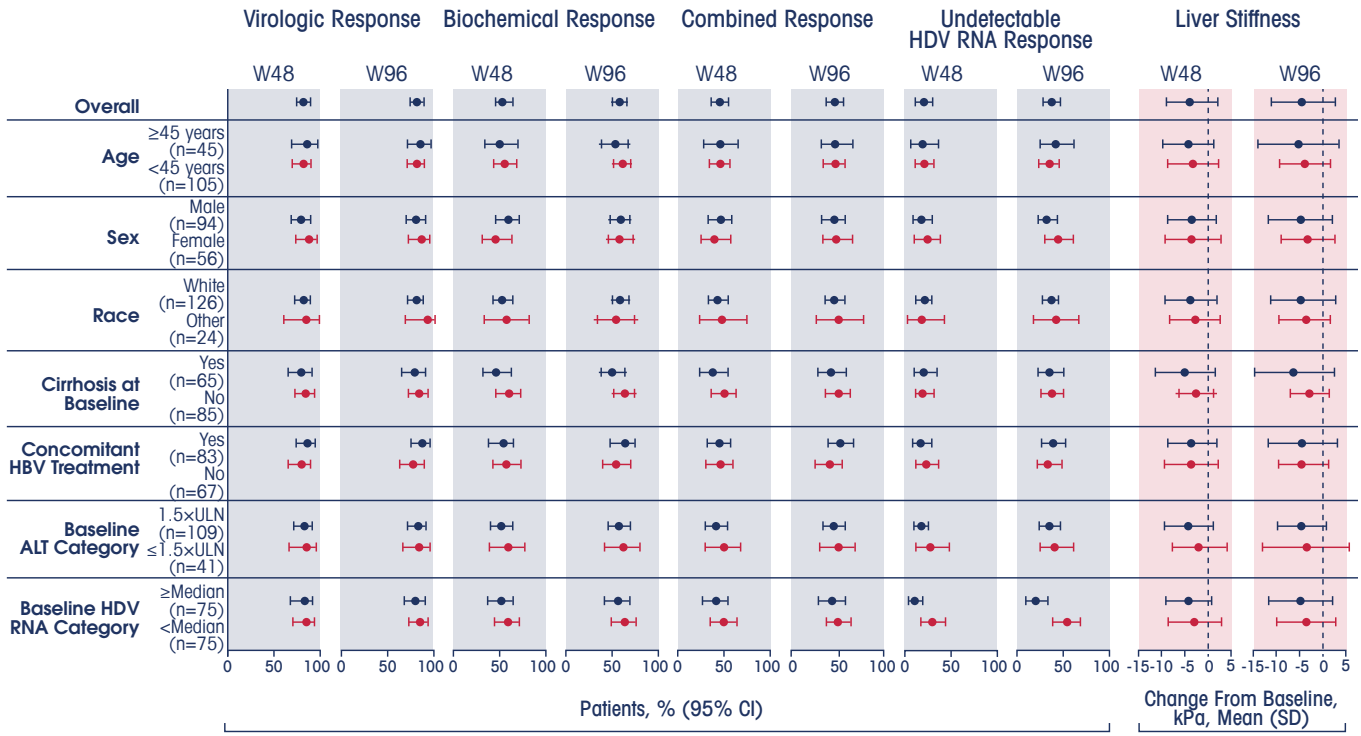
symptom-specific PROs domains assessed using EQ-5D-5L, EQ-VAS, WPAI, and FSS were similar between the two groups.

Efficacy and safety of BLV 10 mg monotherapy

The global efficacy of BLV 10 mg monotherapy and its efficacy across patient subgroups at weeks 48 and 96 were analysed by pooling data from the MYR301 and MYR204 studies (14). Overall, 150 patients were included. Virological response (VR), biochemical response, combined response, undetectable HDV RNA rates, and changes in liver stiffness at weeks 48 and 96 are shown in *Figure 14*. BLV 10 mg monotherapy was effective across subgroups with different demographic and disease characteristics. Patients with lower HDV RNA levels at baseline were more likely to achieve undetectable HDV RNA during the first 96 weeks of treatment. These findings support the efficacy of BLV 10 mg monotherapy across a broad range of patients with chronic hepatitis D and compensated liver disease.

Safety was also assessed by pooling 96-week safety data from the MYR301 and MYR204 studies. A total of 199 patients treated with BLV 2 mg or 10 mg were included in the analysis: 49 patients received BLV 2 mg and

Figure 14 BLV 10 mg monotherapy: overview of efficacy results at weeks 48 and 96 overall and across subgroups



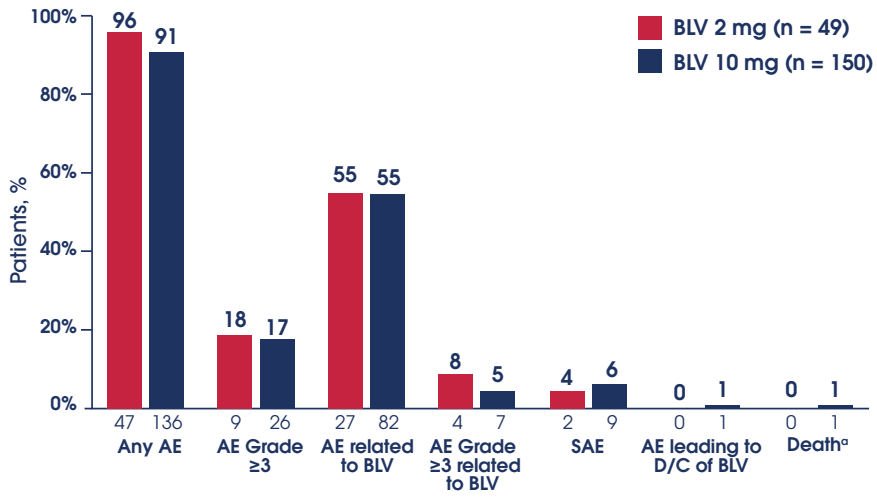
Overall, treatment with BLV 10 mg monotherapy for up to 96 weeks maintained or improved the efficacy outcomes observed at week 48, and efficacy was consistent across subgroups (Figure 2)

Undetectable HDV RNA rates (% of patients (95% CI)) were higher among patients with baseline viral loads less than median (53% (41%–65%) vs 20% (12%–31%)) at week 96

Streinu-Cercel A, et al J Hepatol 2026;84S1:S962

150 received BLV 10 mg (15) (*Figure 15*). The AE profile was generally consistent between the two dose groups through 96 weeks of treatment. Among patients receiving BLV 2 mg and BLV 10 mg, 96% and 91%, respectively, experienced an AE. The incidence of Grade ≥3 AEs was similar in the BLV 2 mg (18%) and BLV 10 mg (17%) groups. Most AEs were mild to moderate in severity. Serious adverse events (SAEs) were infrequent, and none were considered related to BLV. One patient (1%) experienced an AE leading to BLV discontinuation. This non-serious event, myalgia, was considered related to BLV. AEs previously shown to occur more frequently with BLV than without BLV treatment and reported in ≥10% of patients in either group by week 96 included injection-site reactions, headache, fatigue, pruritus, and eosinophilia. Other common AEs, including vitamin D deficiency and cytopenias, are associated with underlying liver disease. Injection-site reactions occurred at similar frequencies in the BLV 2 mg and 10 mg groups (20% and 19%, respectively). Bile salt elevations associated with the mechanism of action of BLV were asymptomatic. No association was observed between bile salt levels and AEs of interest, including pruritus.

Figure 15 BLV 10 mg monotherapy: overall summary of AEs with 96 weeks



[a]*One death due to plasma cell myeloma that was determined to be unrelated to study treatment.*
D/C, discontinuation

Asselah T, et al. J Hepatol 2026;84S1:S939

Overall, BLV monotherapy at both 2 mg and 10 mg showed a favourable safety profile and was well tolerated through 96 weeks, including in patients with compensated cirrhosis.

The safety of BLV 10 mg monotherapy was further evaluated across patient subgroups in an integrated analysis of 150 patients from the MYR204 and MYR301 studies through 96 weeks of treatment (16). Most AEs were mild to moderate in severity. Only one patient discontinued BLV because of an AE (non-serious myalgia), and no discontinuations were due to injection-site reactions. SAEs were infrequent, and none were considered related to BLV. The most common AEs were vitamin D deficiency (30%), injection-site reactions (19%), headache (17%), and leukopenia (15%). Vitamin D deficiency and cytopenias are common in patients with underlying chronic hepatitis D and occurred at similar or lower frequencies in the BLV 10 mg group compared with the delayed-treatment BLV 10 mg group at week 48. The safety profile of BLV and the frequency of AEs remained consistent across all analysed subgroups. A numerically higher incidence of SAEs was observed in patients aged ≥45 years, with 4 of 7 events (57%) related to COVID-19 and none considered related to BLV.

This finding is consistent with the literature showing a higher background rate of SAEs, including those related to COVID-19, in this age subgroup. Patients with cirrhosis at baseline had a numerically higher incidence of Grade ≥3 AEs, largely attributable to cytopenias, which commonly occur in patients with cirrhosis. On-treatment bile salt elevations were similar in patients with and without cirrhosis.

Taken together, these data indicate that long-term BLV 10 mg monotherapy was well tolerated, with a safety profile that remained consistent across patient subgroups analysed. Most treatment-emergent AEs were mild to moderate in severity, while Grade ≥3 AEs and SAEs were infrequent. These findings support the safety of long-term BLV 10 mg monotherapy in patients with chronic hepatitis D and compensated liver disease.

Conclusions

The EASL 2026 Congress marked a turning point for clinical research in HDV. Studies were presented for the first time that have, and are likely to continue to have, a significant impact on the management of patients with HDV in Italy and worldwide. These included data on the efficacy and safety of BLV monotherapy in patients with and without cirrhosis; the efficacy and safety of extending this treatment strategy for up to 4 years in patients with compensated cirrhosis; its effects on non-invasive markers of fibrosis in patients with cirrhosis; the significant reduction in liver-related complications among patients with cirrhosis; and the validation in clinical practice of potential stopping rules in a selected group of patients receiving BLV monotherapy. These studies are complemented by research placing patients at the center of scientific interest, both by assessing improvements in PROs during treatment and by investigating treatment adherence. Within this framework, emerging evidence on BLV 10 mg monotherapy adds to our understanding of its long-term clinical profile, providing key insights to optimize therapeutic strategies across diverse patient subgroups.

Although not summarized here, several new anti-HDV therapeutic approaches currently undergoing clinical development are also of considerable interest, particularly anti-HBsAg monoclonal antibodies administered as monotherapy or in combination with HBV-specific siRNAs. Results from phase IIb and phase III trials with these agents are expected to be presented in 2027 and could represent a major therapeutic innovation for HDV within the next few years.

In conclusion, 49 years after publication of the first paper on HDV (*Rizzetto M et al., Gut 1977*), the clinical landscape in the management and treatment of patients with HDV infection has changed completely. BLV monotherapy now provides a therapeutic approach capable of effectively suppressing HDV replication over the long term, with a significant clinical impact on the progression of liver disease.

For these reasons, and while awaiting new drugs that may become available in the near future, it is recommended that antiviral treatment with BLV monotherapy should be initiated in all patients with CHD to reduce disease progression to cirrhosis, hepatic decompensation, and, most importantly, HCC.

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