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Liver Stiffness–Based Strategies Predict Absence of Variceal Bleeding in Cirrhotic Hepatitis C Virus–Infected Patients With and Without Human Immunodeficiency Virus Coinfection After Sustained Virological Response

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Background. In the setting of hepatitis C virus (HCV) active infection, liver stiffness (LS)–based strategies identify patients with low risk of developing esophageal variceal bleeding (VB) episodes, in whom unnecessary upper esophagogastroduodenoscopy (UGE) screening can be safely avoided. However, after sustained virological response (SVR), data on the accuracy of the criteria predicting this outcome in HCV-infected patients with cirrhosis, with or without human immunodeficiency virus (HIV) coinfection, are very limited.

Methods. This was a multicenter prospective cohort study, where HCV-monoinfected patients and HIV/HCV-coinfected individuals were included if they had (1) SVR with direct-acting antiviral–based therapy; (2) LS ≥9.5 kPa previous to treatment; and (3) LS measurement at the SVR time-point ≥14 kPa. Diagnostic accuracy of HEPAVIR, expanded Baveno VI, and HIV cirrhosis criteria, at the time of SVR, was evaluated. Missed VB episodes, negative predictive values (NPVs), and number of spared UGEs were specifically assessed.

Results. Four hundred thirty-five patients were included, 284 (65%) coinfected with HIV. Seven (1.6%) patients developed a first episode of VB after SVR. In patients without a previous VB episode, HEPAVIR, expanded Baveno VI and HIV cirrhosis criteria achieved NPV for first VB episode after SVR of 99.5% (95% confidence interval [CI], 97.1%–100%), 100% (95% CI 97.8%–100%), and 100% (95% CI 98%–100%) while sparing 45%, 39%, and 44% of UGEs, respectively. When considering HIV coinfection, the performance of the 3 criteria was similar, both in HCV-monoinfected and HIV/HCV-coinfected individuals.

Conclusions. After SVR, predictive LS-based strategies accurately identify HCV-infected patients, HIV coinfected or not, with low risk of developing VB during follow-up. In these specific patients, using HIV cirrhosis criteria maximize the number of spared UGEs while missing no VB episode.

Keywords. HCV infection; sustained virological response; direct-acting antivirals; liver stiffness; variceal bleeding.

Owing to the strong correlation between liver stiffness (LS), measured by vibration-controlled transient elastography (VCTE), and hepatic venous pressure gradient (HPVG) [1–3], noninvasively LS-based strategies identify candidates in whom upper esophagogastroduodenoscopy (UGE) for esophageal varices (EV) needing treatment screening can be safely spared, in the setting of hepatitis C virus (HCV) active infection. In people living with human immunodeficiency virus (HIV) coinfection, HEPAVIR criteria has turned out to be an accurate strategy to avoid up to one-third of UGEs for EV screening in patients with cirrhosis [4, 5]. The Baveno VI criteria have been validated in patients with compensated advanced chronic diseases from different etiologies. Consequently, individuals with LS <20 kPa and a platelet

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count >150 000/mm^3 can be withdrawn from EV surveillance [6–8]. To spare a higher proportion of unnecessary UGEs, Baveno VI expanded criteria have been proposed [9]. Recently, new HIV cirrhosis criteria have been validated in people living with HIV bearing advanced chronic liver disease [10].

It is well known that achieving sustained virological response (SVR) leads to a reduction of the HPVG [11]. Nevertheless, even after SVR, clinically significant portal hypertension may persist, especially in patients with a certain degree of portal hypertension prior to viral eradication [12]. Therefore, the risk for variceal bleeding (VB) still remains [11]. Data on the accuracy of currently criteria regarding screening and surveillance of EV in HCV-infected patients with cirrhosis, after SVR are scarce. In a recent study conducted in the ANRS CO12 CirVir cohort, the clinical value of Baveno VI criteria was validated among patients with viral cirrhosis, after SVR [12]. However, <5% of HCV-infected individuals were coinfected with HIV. Since HEPAVIR and HIV cirrhosis criteria have shown excellent performance in ruling out EV needing treatment in this specific subset [5, 10], it is reasonable to speculate that, after SVR, the predictive value of these criteria may still perform better in this subpopulation.

Therefore, the aim of the present study was to validate the currently LS-based scores, assessed at the time of SVR, for VB in HCV-infected patients, both HIV coinfected and HIV uninfected, with cirrhosis, after attaining SVR with direct-acting antiviral (DAA)–based therapy.

METHODS

Patients and Follow-up

This is a multicenter prospective study that included HIV/HCV-coinfected individuals and HCV-monoinfected patients from the GEHEP-011 Cohort (ClinicalTrials.gov identifier NCT04460157), followed at 18 infectious disease units throughout Spain since October 2011. The inclusion criteria in the cohort were (1) LS value ≥9.5 kPa before starting treatment; (2) SVR with regimens containing at least 1 DAA; and (3) LS measurement available at the time of SVR. Patients with positive hepatitis B surface antigen were excluded. For this study, only patients with LS values at SVR ≥14 kPa were included.

The date of SVR was considered as the baseline time-point. All patients were evaluated, under a common protocol, at least every 6 months until death, liver transplant, HCV reinfection, or the censoring date (30 November 2019). At every visit, clinical and routine laboratory examinations were completed. Patients with cirrhosis were managed according to a specific protocol reported elsewhere [13]. Hepatocellular carcinoma screening was carried out biannually, based on plasma α-fetoprotein determinations and liver ultrasound examination, conducted by an experienced examiner at each participating center. In patients with LS ≥21 kPa, surveillance of gastroesophageal varices was performed with serial UGEs [5].

VCTE Examinations

LS was assessed by VCTE (FibroScan, Echosens, Paris, France), according to a standardized procedure, using the M probe. At each participating institution, examinations were performed by a trained operator. To considered LS determinations reliable, evaluations had to include at least 10 measurements, with a success rate ≥60% and an interquartile range <30% of the median.

EV Screening and Prevention of Variceal Bleeding

At cohort entry with LS ≥21 kPa, all patients had undergone UGE for the screening of EV, which were staged following the Japanese Research Society for Portal Hypertension staging system [14]. Thus, according to their shape and their size, EV were classified in the following way: F0, no EV; F1, straight, small-caliber varices; F2, moderately enlarged, beady varices; and F3, markedly enlarged, nodular or tumor-shaped varices. Likewise, the presence of red signs (red wale markings, cherry red spots, or hematocystic spots) was also considered. Patients with initial LS <21 kPa were spared from UGE. VCTE was performed every year. If LS showed a subsequent progression >21 kPa, a UGE was performed. Finally, surveillance UGE was repeated after 2–3 years in patients with LS at least 21 kPa if there were no EV at the initial screening examination. According to Baveno VI [6] and the American Association for the Study of Liver Diseases [15], primary prophylaxis for VB was recommended in individuals with high-risk varices (F2, F3 or F1 with red wale signs or Child-Pugh-Turcotte class C) with nonselective β-blockers (NSBBs) or endoscopic band ligation (EBL). In all patients starting NSBB therapy, blood pressure and heart rate were closely monitored. NSBB dose was adjusted to achieve a heart rate from 55–60 beats per minute, without hypotension (mean arterial pressure >85 mm Hg). In case of intolerance or contraindication to NSBBs, EBL was performed. Secondary prophylaxis with NSBBs or EBL was indicated in patients who developed a VB during follow-up.

Endpoint and Other Definitions

The primary endpoint of the study was the emergence of a gastroesophageal VB first episode after SVR. For these analyses, bleeding episodes from portal hypertension gastropathy were not considered. The diagnostic accuracy of HEPAVIR (favorable status LS <21 kPa), expanded Baveno VI (favorable status LS <25 kPa and platelet count >110 000/mm^3) and HIV cirrhosis (favorable status LS <30 kPa and platelet count >110 000/mm^3) criteria, at the time of SVR, was validated in patients without previous VB. The missed VB episodes and the number of spared UGEs spared were evaluated. The number of spared UGEs was estimated on the basis of the number of patients in whom UGE could be avoided because they showed a favorable status in each of the criteria studied, that is, those with LS of 14–20.9 kPa for HEPAVIR; LS 14–24.9 kPa and platelet

count >110 000 for expanded Baveno VI; and LS 14–29.9 kPa and platelet count >110 000 for HIV cirrhosis criteria.

SVR was defined as undetectable HCV RNA 12 weeks after the end of HCV therapy. In line with previous studies, individuals with LS ≥14 kPa were considered to have cirrhosis [13, 16, 17].

Statistical Analysis

Categorical variables are expressed as numbers (percentage) and continuous variables as median (quartile 1–quartile 3 [Q1–Q3]). Incidence rate of VB were estimated and they are provided along with 95% confidence intervals (95% CIs). Diagnostic accuracy of the currently criteria was assessed by sensitivity, specificity, positive predictive value, negative predictive value (NPV), percentage of missed VB, and percentage of spared UGEs, along with "exact" Clopper-Pearson CIs.

Ethical Considerations

This study was conducted according to the Helsinki declaration and was approved by the Ethics Committee of the Hospital Universitario de Valme. All patients gave written informed consent before being recruited in the cohorts.

RESULTS

Patients' Characteristics

Four hundred thirty-five patients were included in this study. Nearly two-thirds of individuals were coinfected with HIV. Thirteen (3.0%) individuals had developed a VB episode before SVR. Main features of the study population are displayed in Table 1. At the time of SVR, all HIV/HCV-coinfected participants were receiving antiretroviral therapy and 235 (93%) had a plasma HIV RNA load <50 copies/mL. The median CD4 cell count was 472 (Q1–Q3, 271–690) cells/mL.

The global median follow-up time was 44 (Q1–Q3, 30–49) months. During the study, 10 (2.3%) patients were lost to follow-up, 2 (0.5%) suffered from an HCV reinfection, and 9 (2.1%) underwent a liver transplant. Overall, 35 (8.0%) patients died.

Variceal Bleeding During the Follow-up

During the study period, 10 (2.3%) individuals developed a VB episode after SVR, accounting for an overall incidence rate of 0.8 (95% CI, .4–1.4) per 100 person-years. Three (0.7%) of them had experienced a VB episode before SVR. Thus, the incidence rate of this event in patients with no VB prior to SVR was 0.6 (95% CI, .3–1.2) per 100 person-years.

The main features of the 7 patients with a VB first episode during follow-up are summarized in Table 2. Six (1.4%) were coinfected with HIV. In 4 of the 7 individuals, VB was the first hepatic decompensation of cirrhosis (patients 1–4). Patient 7 refused to undergo UGE for EV screening. Two years later, he developed simultaneously a VB episode along with ascites, hepatic encephalopathy, and acute-on-chronic disease.

Table 1. Main Characteristics of the Study Population (N = 435)

Parameter	Value
Age, y, median (Q1–Q3)	53 (49–57)
Male sex	352 (81)
Injecting drug user	305 (70)
HCV/HIV coinfection	284 (65)
LS value before treatment, kPa	
<14	26 (6)
<21	111 (26)
<25	164 (38)
<30	243 (56)
High-risk esophageal varices needing treatment in patients with LS before treatment ≥21.0 kPa	62 (14)
Primary prophylaxis of variceal bleeding in patients with high-risk varices needing treatment	
NSBBs	56 (90)
EBL	6 (10)
CPT class A prior to treatment[a]	368 (85)
MELD score prior to treatment, median (Q1–Q3)[b]	7 (6–9)
Liver complication before SVR	53 (6)
LS value at SVR, kPa	
<21	195 (45)
<25	257 (59)
<30	312 (72)
CPT class A at SVR[c]	388 (94)
MELD score at SVR, median (Q1–Q3)[d]	7 (6–9)
Platelets count, $\times 10^9/mm^3$, at SVR, median (Q1–Q3)[e]	116 (83–162)
Alcohol intake at SVR <50 g/day[f]	331 (76)

Data are presented as No. (%) of participants unless otherwise indicated.

Abbreviations: CPT, Child-Pugh-Turcotte; EBL, endoscopic band ligation; HCV, hepatitis C virus; HIV, human immunodeficiency virus; LS, liver stiffness; MELD, Model for End-Stage Liver Disease; NSBBs, nonselective β-blockers; Q1, quartile 1; Q3, quartile 3; SVR, sustained virological response.

[a]Available in 409 patients.

[b]Available in 403 individuals.

[c]Available in 413 patients.

[d]Available in 409 individuals.

[e]Available in 434 patients.

[f]Available in 349 individuals.

Performance of LS-Based Criteria for Variceal Bleeding

Considering patients without a previous VB episode, 190 (45.0%) had a favorable status according to HEPAVIR criteria, of whom 1 (0.5%) developed a VB after SVR. Taking into account expanded Baveno VI criteria, 165 (39.1%) individuals had a favorable status, none of whom experienced a subsequent VB episode. Finally, 186 (44.1%) patients had a favorable status according to HIV cirrhosis criteria and none of them developed a VB. Consequently, NPV for first VB episode after SVR was 99.5% (95% CI, 97.1%–99.0%), 100% (95% CI, 97.8%–100%) and 100% (95% CI, 98.0%–100%) for HEPAVIR, expanded Baveno VI, and HIV cirrhosis criteria, respectively. Using HEPAVIR and HIV cirrhosis criteria would have allowed a reduction of 13.2% and 11.3% of UGEs comparing to expanded Baveno VI. Table 3 summarizes diagnostic accuracy for first VB event after SVR of HEPAVIR, expanded Baveno VI, and HIV cirrhosis criteria, by favorable status.

Table 2. Main Features of Patients Who Developed Gastroesophageal Variceal Bleeding After Sustained Virological Response (n = 7)

ID	SVR Date[a]	CPT Score at SVR	Platelet Count at SVR, $\times 10^9/mm^3$	LS at SVR, kPa	Date of UGE Prior to VB	EV in UGE Prior to VB	PP	VB Date	UGE Findings at VB	Closest LS to VB, kPa	Closest Platelet Count to VB, $\times 10^9/mm^3$	VB-Related Death
1	5 Jan 2017	7	56	34.8	27 Sep 2016	F3	NSBBs	23 Jan 2018	F3	48.0 8 Jan 2018	58	No
2	27 Oct 2015	5	34	30.0	2 Jun 2014	F0	No	11 Jul 2017	F3	38.9 18 Sep 2017	45	No
3	17 Nov 2015	5	74	32.0	1 Feb 2017	F2	Yes[a]	19 Jan 2018	F3	24.2 24 Nov 2016	72	No
4	3 Feb 2016	5	91	33.5	1 Aug 2017	F3	EBL	20 Apr 2019	F3	30.0 27 Mar 2017	95	No
5	21 Dec 2015	6	50	38.5	12 Jul 2017	F1	No	18 Oct 2018	F3	65.2 20 Oct 2016	37	No
6	1 Jul 2016	5	51	173	30 May 2016	F2	NSBBs	1 Oct 2018	F3	21.3 22 May 2018	67	No
7	2 Dec 2016	5	43	21.0	No	…[b]	No	25 Sep 2018	F3	19.4 10 Apr 2018	71	Yes 30 Oct 2018

Abbreviations: CPT, Child-Pugh-Turcotte; EBL, endoscopic band ligation; EV, esophageal varices; ID, identification; LS, liver stiffness; NSBBs, nonselective β-blockers; PP, primary prophylaxis; SVR, sustained virological response; UGE, upper esophagogastroduodenoscopy; VB, variceal bleeding.

[a]NSBBs were prescribed; however, patient was not adherent to PP.

[b]UGE for EV needing treatment screening was not performed because of patient refusal. Thus, no PP was initiated.

Conducting sensitivity analyses by HIV coinfection, the 3 criteria performed similarly both in HCV-monoinfected and HIV/HCV-coinfected patients. Regarding HCV-monoinfected individuals, none of the 3 criteria missed a VB event, achieving NPV of 100%. The application of either HEPAVIR or HIV cirrhosis criteria would spare 12.7% of UGEs compared to expanded Baveno VI. In the HIV/HCV coinfection group, HEPAVIR criteria missed 1 VB episode (0.8%), showing NPV of 99.2% (95% CI, 95.4%–99.9%). Expanded Baveno VI and HIV cirrhosis missed no bleeding events, maintaining NPV at 100%. Finally, HEPAVIR and HIV cirrhosis criteria employment would have attained an absolute reduction of 13.5% and 10.5% of UGEs more than expanded Baveno VI, respectively. Diagnostic accuracy for a first episode of VB after SVR of the 3 criteria, according to HIV coinfection, is displayed in Table 4.

When considering the whole population, HEPAVIR criteria classified 195 (44.8%) patients in the favorable status group while missing 3 (1.5%) VB events. According to expanded Baveno VI and HIV cirrhosis criteria, 166 (38.2%) and 188 (43.2%) individuals had a favorable status, respectively. None of them developed a VB episode. Thus, NPV for a VB event after SVR, irrespective of previous VB episode, was 98.5% (95% CI, 95.6%–99.7%), 100% (95% CI, 97.8%–100%), and 100% (95% CI, 98.1%–100%) for HEPAVIR, expanded Baveno VI and HIV cirrhosis criteria, respectively. Use of HIV cirrhosis and HEPAVIR criteria would have attained an absolute reduction of 11.7% and 14.1% of UGEs more than expanded Baveno VI criteria.

DISCUSSION

This study suggests that, after SVR with DAA-based regimens, HIV cirrhosis, HEPAVIR criteria, and expanded Baveno VI are useful to identify subjects at low risk of experiencing a VB episode. Using these strategies, a substantial number of patients may be spared from an invasive and inconvenient examination, such as UGE. In these specific patients, using HIV cirrhosis criteria maximizes the number of spared UGEs while missing no VB episode.

The correlation between LS and HPVG has been demonstrated in HCV-infected patients before and after achieving SVR [2, 11, 18]. Consequently, noninvasive LS measurement is commonly used for identifying individuals without clinically significant portal hypertension [19]. In the setting of HCV-active infection, LS has turned out to be a predictor of presence of EV needing treatment and has been related to the risk of developing VB. Thus, to identify patients with low risk of VB, different LS cutoffs have been validated, taking into account platelet count or not, in HCV-monoinfected patients and HIV/HCV-coinfected individuals [4–9]. Because achieving viral cure improves portal hypertension [20, 21], reduces the risk of liver complications [22], and prevents de novo development of EV [23], UGEs after SVR for surveillance of EV may be unnecessary in a significant

Table 3. Diagnostic Accuracy for a First Episode of Gastroesophageal Variceal Bleeding After Sustained Virological Response of HEPAVIR, Expanded Baveno VI, and Human Immunodeficiency Virus Cirrhosis Criteria by Favorable Status (n = 422)

Diagnostic Accuracy	HEPAVIR (LS <21 kPa)	Expanded Baveno VI (LS <25 kPa and Platelet Count >110 000/mm^3)	HIV Cirrhosis (LS <30 kPa and Platelet Count >110 000)
Sensitivity, % (95% CI)	85.7 (42.1–99.6)	100 (59.0–100)	100 (59.0–100)
Specificity, % (95% CI)	45.5 (40.7–50.5)	39.9 (35.2–44.9)	44.9 (40.1–49.8)
PPV, % (95% CI)	2.6 (.9–5.5)	2.7 (1.1–5.6)	3.0 (1.2–6.0)
NPV, % (95% CI)	99.5 (97.1–99.0)	100 (97.8–100)	100 (98.0–100)
Missed VB, No. (%)	1/190 (0.5)	0/165	0/186
Spared UGE, No. (%)	190 (45.0)	165 (39.1)	186 (44.1)

Abbreviations: CI, confidence interval; HEPAVIR, Grupo Andaluz para el Estudio de las Hepatitis Víricas; HIV, human immunodeficiency virus; LS, liver stiffness; NPV, negative predictive value; PPV, positive predictive value; UGE, upper esophagogastroduodenoscopy; VB, variceal bleeding.

number of patients. Additionally, recommendations on the interval of UGE surveillance of EV, particularly after SVR, have not been definitely established and those currently available are based upon expert opinions. Moreover, to date, data regarding the accuracy of several criteria commonly used for deciding VB surveillance after SVR are quite limited. A recent work showed that favorable Baveno VI status among individuals with viral-related cirrhosis allowed the identification of patients with no risk of bearing EV needing treatment after viral eradication [12]. However, the cohort was heterogeneous as it included cirrhosis of different etiologies. Furthermore, patients with and without viral eradication at inclusion were followed. In addition, HIV coinfection was present in a very low proportion of patients. What was more relevant in this study is that none of the 80 individuals who at the time of viral suppression had a favorable Baveno VI status showed portal hypertension progression, as did those who changed from unfavorable to favorable status during the follow-up.

Although well validated in several studies [7, 8], Baveno VI criteria are perceived as conservative, as the number of spared UGEs is rather low. With expanded Baveno VI, the number of spared UGEs can be optimized, while keeping the rate of VB missed below the 5% threshold. However, it raises some doubts as it may increase the risk of missing events [9, 24, 25]. In the present work, expanded Baveno VI achieved 100% NPV for VB while allowing us to avoid 39% of UGEs among patients with cirrhosis. HEPAVIR criteria showed practically identical NPV for VB, with a probability of developing the event far below 5%, offering a significant advantage in terms of UGEs spared. Nevertheless, HIV cirrhosis criteria did not classify any patient with VB as a false negative, achieving a 100% NPV for a first episode of VB after SVR, with an increase of 5% in spared UGEs. Although developed in patients with HIV coinfection, these criteria also seem to perform very well in patients with HCV monoinfection. Additionally, HIV cirrhosis criteria appear to be useful to identify patients with no risk of developing VB event after SVR, irrespective of VB episodes occurrence prior to viral cure. Further studies will be necessary to clarify this specific issue.

Table 4. Diagnostic Accuracy for a First Episode of Gastroesophageal Variceal Bleeding After Sustained Virological Response of HEPAVIR, Expanded Baveno VI, and Human Immunodeficiency Virus (HIV) Cirrhosis Criteria (Favorable Status), According to HIV Infection (n = 422)

Diagnostic Accuracy	HEPAVIR (LS <21 kPa)	Expanded Baveno VI (LS <25 kPa and Platelet Count >110 000/mm^3)	HIV Cirrhosis (LS <30 kPa and Platelet Count >110 000)
HIV/HCV-coinfected individuals (n = 272)			
Sensitivity, % (95% CI)	83.3 (35.9–99.6)	100 (54.1–100)	100 (54.1–100)
Specificity, % (95% CI)	44.4 (38.3–50.6)	38.7 (32.8–44.9)	43.2 (37.2–49.4)
PPV, % (95% CI)	3.3 (1.1–7.5)	3.6 (1.3–7.6)	3.8 1.4-8.1)
NPV, % (95% CI)	99.2 (95.4–100)	100 (96.5–100)	100 (96.8–100)
Missed VB, No. (%)	1/119 (0.8)	0/103	0/115
Spared UGE, No. (%)	119 (43.8)	103 (37.9)	115 (42.3)
HCV-monoinfected individuals (n = 150)			
Sensitivity, % (95% CI)	100 (2.5–100)	100 (2.5–100)	100 (2.5–100)
Specificity, % (95% CI)	47.7 (39.4–56.0)	41.9 (33.8–50.3)	48.0 (39.7–56.3)
PPV, % (95% CI)	1.3 (.03–6.8)	1.1 (.03–6.2)	1.3 (.03–6.9)
NPV, % (95% CI)	100 (94.9–100)	100 (94.2–100)	100 (94.9–100)
Missed VB, No. (%)	0/71	0/62	0/71
Spared UGE, No. (%)	71 (47.3)	62 (41.3)	71 (47.3)

Abbreviations: CI, confidence interval; HCV, hepatitis C virus; HEPAVIR, Grupo Andaluz para el Estudio de las Hepatitis Víricas; HIV, human immunodeficiency virus; LS, liver stiffness; NPV, negative predictive value; PPV, positive predictive value; UGE, upper esophagogastroduodenoscopy; VB, variceal bleeding.

This study has some limitations. First, the analyses are restricted to HCV-related chronic liver disease. Previous studies have demonstrated that expanded Baveno VI criteria performed very well in the subset of the main etiologies of chronic liver disease, including alcoholic and nonalcoholic steatohepatitis, as well hepatitis B virus infection [9]. Thus, it can be hypothesized that the studied criteria might safely rule out for VB in individuals with chronic liver disease from other etiologies. In any case, further studies with specific data in non-HCV-infected patients cirrhosis are needed. Second, despite a median follow-up of nearly 4 years, the number of VB was quite low. However, these results from a real-life cohort reflect the currently clinical landscape of HCV-related liver cirrhosis after SVR. Despite longer follow-up, the incidence of VB in HCV-cured patients with advanced liver disease is low in this setting. Consequently, a larger number of bleeding events is not easy to achieve. In the meantime, patients and healthcare systems would have to face the overload and costs of unnecessary invasive examinations. Finally, this study is conducted in the GEHEP-011 cohort, which is largely comprised of HIV/HCV-coinfected individuals. Nevertheless, this is the study validating the performance of LS-based strategies for VB in HCV-infected patients with cirrhosis after SVR, which included the largest sample size of HIV-coinfected individuals. In addition, we provide long-term data of a large sample size of HCV-infected patients with cirrhosis, in which individuals are prospectively followed in a real-life clinical practice setting. Moreover, all patients included showed the same cause of liver disease and had achieved SVR. Focusing on patients with viral cure is critical as a great proportion of HCV-infected individuals had attained SVR in our area nowadays. Those are the strengths of this study.

In conclusion, HIV cirrhosis criteria showed a better performance than HEPAVIR and extended Baveno VI criteria as they could identify a higher proportion of patients who may benefit from avoiding UGE-based varices surveillance after SVR, with a high yield to predict the absence of VB. Thus, subjects with LS cutoff of 30 kPa and a platelet count threshold of 110 000 at SVR are not at risk of developing VB during follow-up. Consequently, nearly 44% of unnecessary UGEs could be safely spared, while missing no bleeding events, if patients with favorable HIV cirrhosis criteria do not undergo UGE-based surveillance for varices.

Notes

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