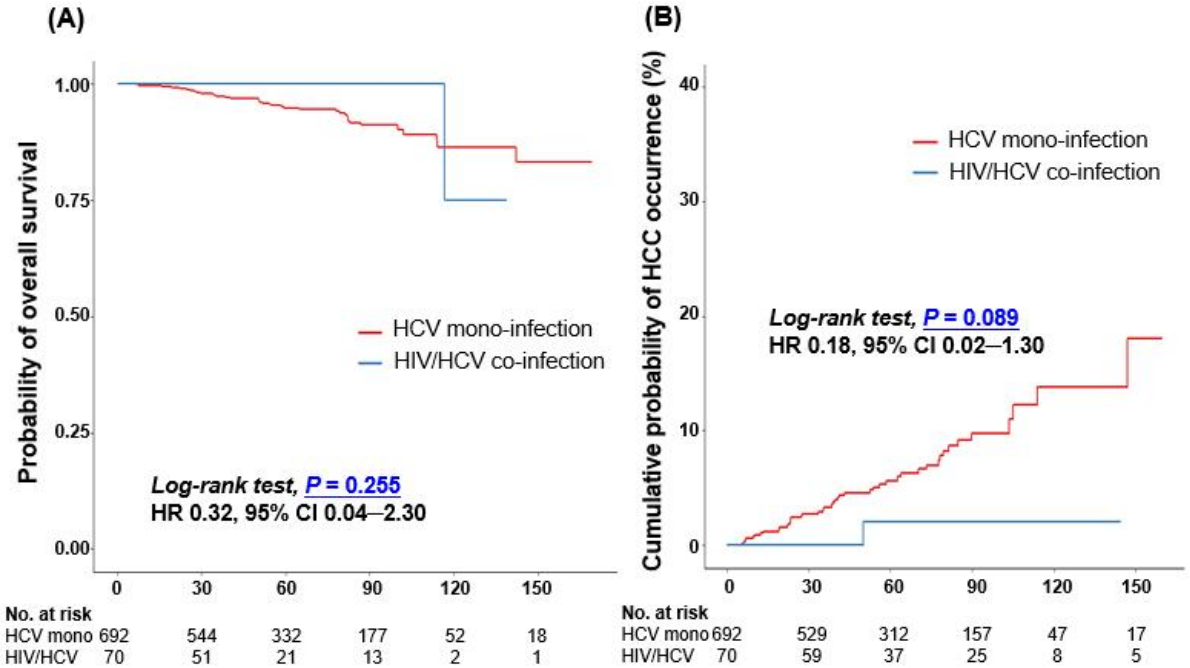
Supplementary materials

Participating centers

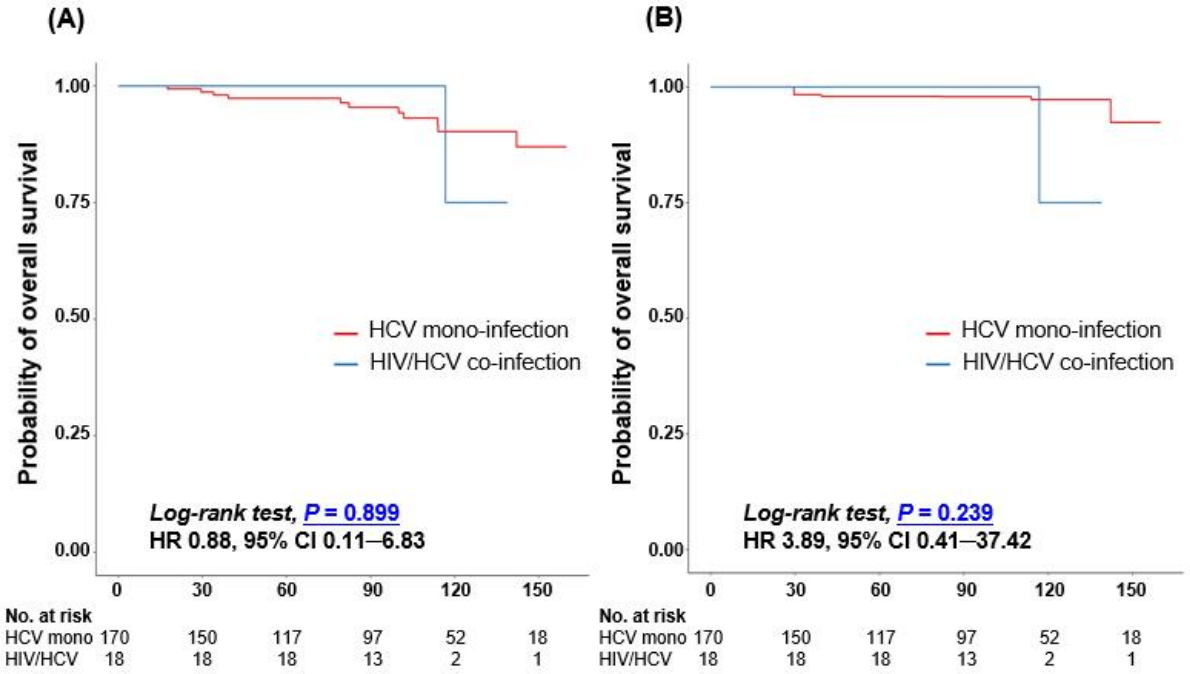
HIV/HCV co-infection: Seoul National University Hospital (Seoul, Republic of Korea); National Medical Center (Seoul, Republic of Korea); Ajou University Hospital (Suwon, Republic of Korea); Chonnam National University Hospital (Gwangju, Republic of Korea); Chungnam National University Hospital (Daejeon, Republic of Korea); Asan Medical Center (Seoul, Republic of Korea; Korea University Ansan Hospital (Ansan, Republic of Korea); Kyungpook National University Hospital (Daegu, Republic of Korea); Severance Hospital (Seoul, Republic of Korea); Korea University Guro Hospital (Seoul, Republic of Korea); Inha University Hospital (Incheon, Republic of Korea); Seoul St. Mary's Hospital (Seoul, Republic of Korea).

HCV mono-infection: Seoul National University Hospital (Seoul, Republic of Korea); National Medical Center (Seoul, Republic of Korea)



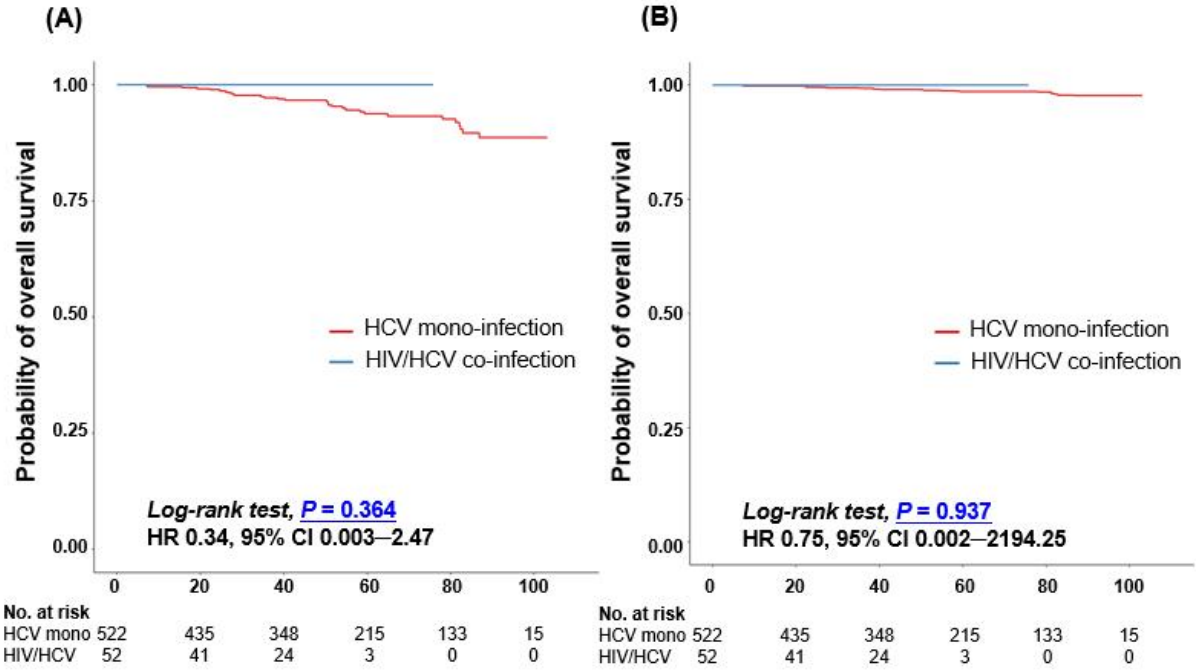
Supplementary Figure 1 Clinical outcomes in hepatitis C virus patients achieving sustained virologic response stratified by human immunodeficiency virus co-

infection status. A and B: Kaplan–Meier estimates of overall survival (A) and time to the occurrence (B) of hepatocellular carcinoma in hepatitis C virus (HCV) patients who achieved sustained virologic response after treatment, stratified by human immunodeficiency virus co-infection status, before inverse probability of treatment weighting adjustment. Propensity scores of inverse probability of treatment weighting were computed using the age, sex, diabetes mellitus status, HCV treatment method, categorized scores for the fibrosis-4 index, the albumin–bilirubin score, and alpha-fetoprotein levels, presence of liver cirrhosis, Child-Pugh score, and HCV genotype. HCV: Hepatitis C virus; HIV: Human immunodeficiency virus.

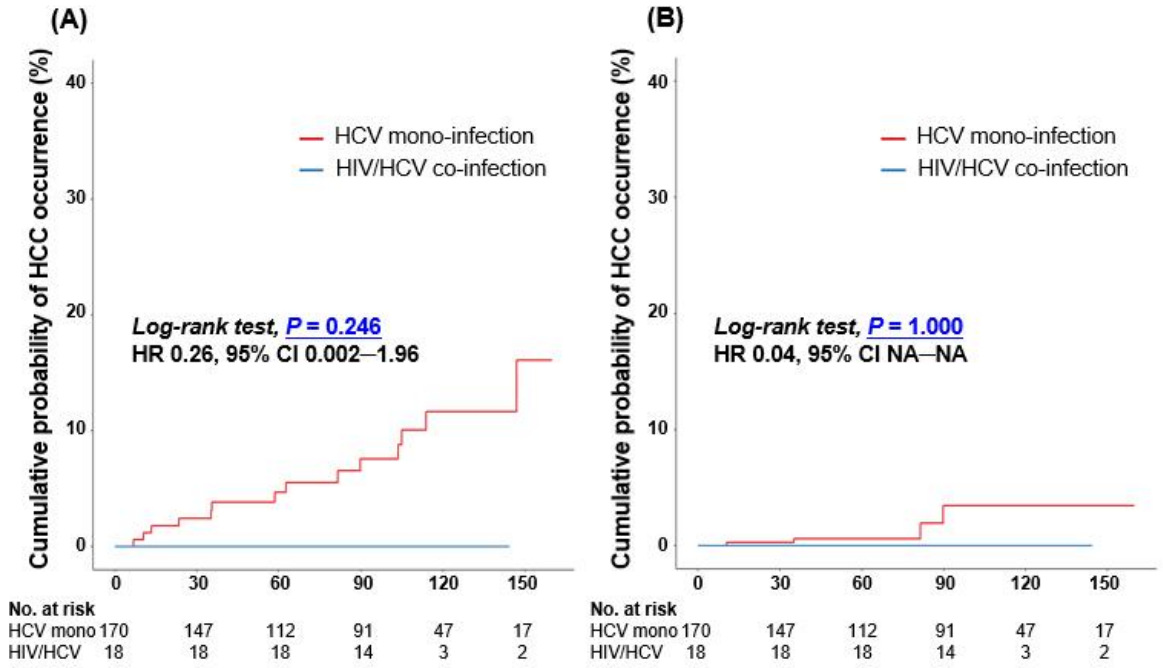


Supplementary Figure 2 Overall survival in hepatitis C virus patients achieving sustained virologic response stratified by human immunodeficiency virus co-infection status. A and B: Kaplan–Meier estimates of overall survival in hepatitis C virus (HCV) patients who achieved sustained virologic response after peginterferon plus ribavirin treatment, stratified by human immunodeficiency virus co-infection status, before (A) and after (B) inverse probability of treatment weighting adjustment. Propensity scores of inverse probability of treatment weighting were computed

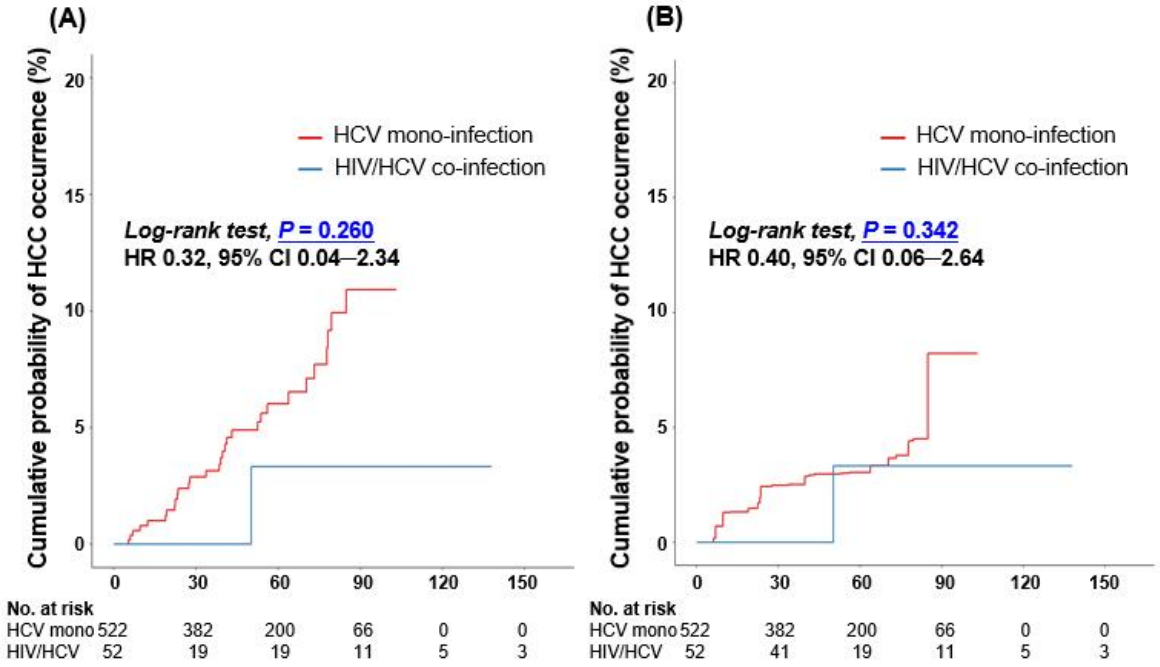
using the age, sex, diabetes mellitus status, HCV treatment method, categorized scores for the fibrosis-4 index, the albumin-bilirubin score, and alpha-fetoprotein levels, presence of liver cirrhosis, Child-Pugh score, and HCV genotype. HCV: Hepatitis C virus; HIV: Human immunodeficiency virus.



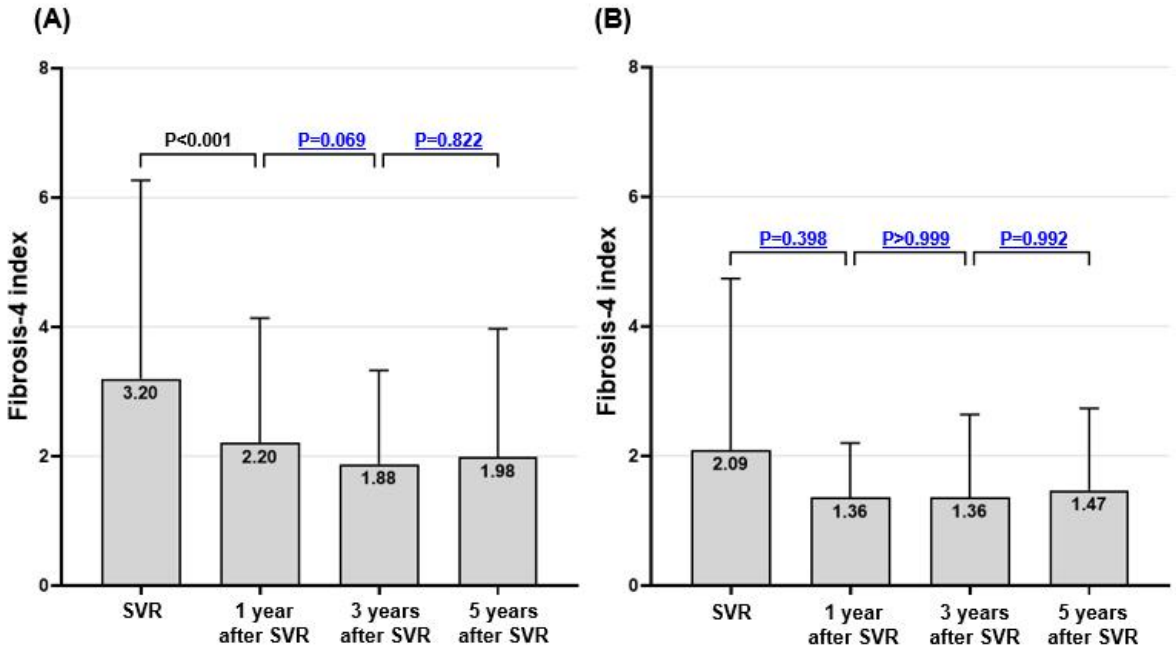
Supplementary Figure 3 Overall survival in hepatitis C virus patients treated with direct-acting antivirals. A and B: Kaplan-Meier estimates of overall survival in hepatitis C virus (HCV) patients who achieved sustained virologic response after direct-acting antiviral treatment, stratified by human immunodeficiency virus co-infection status, before (A) and after (B) inverse probability of treatment weighting adjustment. Propensity scores of inverse probability of treatment weighting were computed using the age, sex, diabetes mellitus status, HCV treatment method, categorized scores for the fibrosis-4 index, the albumin–bilirubin score, and alpha-fetoprotein levels, presence of liver cirrhosis, Child-Pugh score, and HCV genotype.HCV: Hepatitis C virus; HIV: Human immunodeficiency virus.



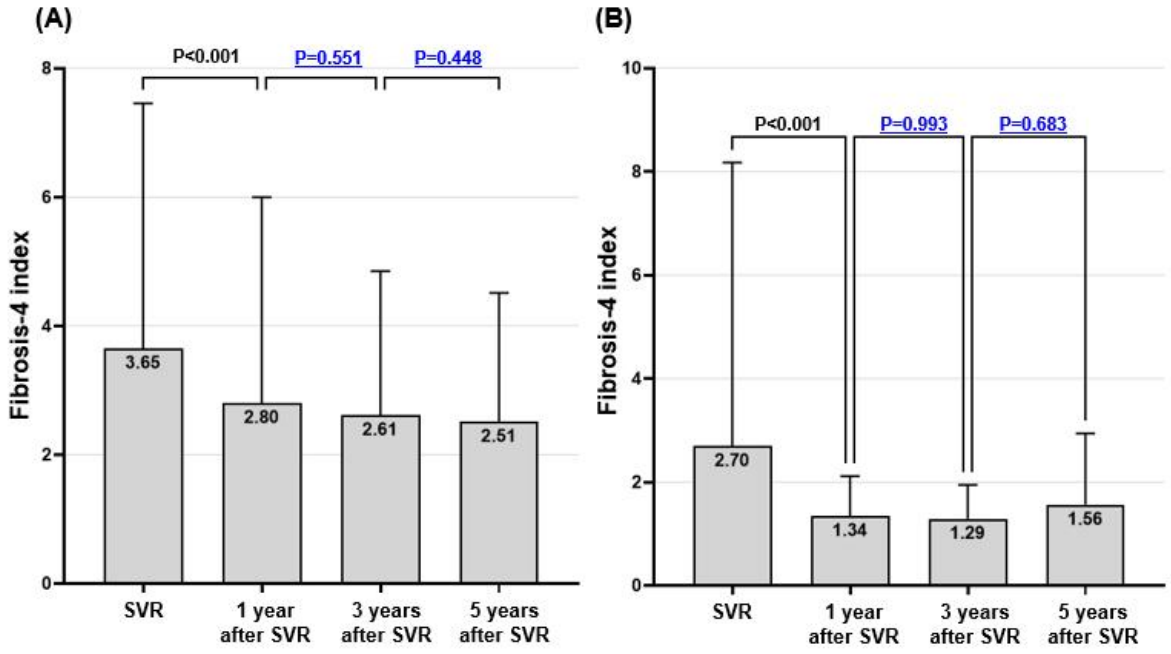
Supplementary Figure 4 Cumulative incidence of hepatocellular carcinoma. A and B: Kaplan–Meier estimates of the time-to-occurrence of hepatocellular carcinoma in hepatitis C virus (HCV) patients who achieved sustained virologic response after peginterferon plus ribavirin treatment, stratified by human immunodeficiency virus co-infection status, before (A) and after (B) inverse probability of treatment weighting adjustment. Propensity scores of inverse probability of treatment weighting were computed using the age, sex, diabetes mellitus status, HCV treatment method, categorized scores for the fibrosis-4 index, the albumin–bilirubin score, and alpha-fetoprotein levels, presence of liver cirrhosis, Child-Pugh score, and HCV genotype. HCC: Hepatocellular carcinoma; HCV: Hepatitis C virus; HIV: Human immunodeficiency virus.



Supplementary Figure 5 Cumulative incidence of hepatocellular carcinoma. A and B: Kaplan–Meier estimates of the time-to-occurrence of hepatocellular carcinoma in hepatitis C virus (HCV) patients who achieved sustained virologic response after direct-acting antiviral treatment, stratified by human immunodeficiency virus co-infection status, before (A) and after (B) inverse probability of treatment weighting adjustment. Propensity scores of inverse probability of treatment weighting were computed using the age, sex, diabetes mellitus status, HCV treatment method, categorized scores for the fibrosis-4 index, the albumin–bilirubin score, and alpha-fetoprotein levels, presence of liver cirrhosis, Child-Pugh score, and HCV genotype. HCC: Hepatocellular carcinoma; HCV: Hepatitis C virus; HIV: Human immunodeficiency virus.



Supplementary Figure 6 Changes in the fibrosis-4 index. A and B: Changes in the fibrosis-4 index in hepatitis C virus (HCV) mono-infected patients (A) and human immunodeficiency virus/HCV co-infected patients (B) who achieved sustained virologic response after receiving peginterferon plus ribavirin treatment. Within-patient changes in the fibrosis-4 index over time were assessed using repeated measures analysis of variance. The mean Fibrosis-4 index is presented at the top of each bar in the graph. SVR: Sustained virologic response.



Supplementary Figure 7 Changes in the fibrosis-4 index. A and B: Changes in the fibrosis-4 index in hepatitis C virus (HCV) mono-infected patients (A) and human immunodeficiency virus/HCV co-infected patients (B) who achieved sustained virologic response after direct-acting antiviral treatment. Within-patient changes in the fibrosis-4 index over time were assessed using repeated measures analysis of variance. The mean Fibrosis-4 index is presented at the top of each bar in the graph. SVR: Sustained virologic response.

Supplementary Table 1 Cox regression analysis of factors associated with ov erall survival of hepatocellular carcinoma before inverse probability of treat ment weighting in patients who achieved sustained virologic response

Variables	Crude HR (95% CI)	*P*-value	Adjusted HR (95% CI)	*P*-value
Group		0.255		0.658
HCV mono-infection	1 [Reference]		1 [Reference]	
HIV/HCV co-infection	0.32 (0.04–2.30)		0.63 (0.08–4.71)	

Age		<0.001		0.002
<65 years	1 [Reference]		1 [Reference]	
≥65 years	4.34 (2.28–8.25)		3.01 (1.51–6.00)	
Sex		0.272		
Male	1 [Reference]			
Female	0.71 (0.38–1.31)			
Diabetes mellitus		0.049		0.268
Absent	1 [Reference]		1 [Reference]	
Present	2.05 (1.00–4.18)		1.52 (0.72–3.22)	
Hypertension		0.146		
Absent	1 [Reference]			
Present	0.42 (0.13–1.36)			
Child-Pugh score		0.112		
A	1 [Reference]			
B	2.61 (0.80–8.53)			
Treatment		0.041		0.275
Peginterferon plus ribavirin	1 [Reference]		1 [Reference]	
DAA	2.44 (1.04–5.73)		1.64 (0.67–4.03)	
Fibrosis-4 index		<0.001		0.610
≤3.25	1 [Reference]		1 [Reference]	
>3.25	2.93 (1.55–5.54)		1.14 (0.69–1.89)	
Liver cirrhosis		<0.001		0.039
Absent	1 [Reference]		1 [Reference]	
Present	3.33 (1.80–6.12)		2.08 (1.07–4.08)	
AFP (ng/mL)		0.219		
<20	1 [Reference]			
≥20	1.73 (0.72–4.13)			

ALBI score		0.002		0.122
≤-2.60	1 [Reference]		1 [Reference]	
>-2.60	2.66 (1.43–4.93)		1.72 (0.87–3.39)	
APRI		0.057		
<1	1 [Reference]			
≥1	1.84 (0.98–3.43)			

HCV: Hepatitis C virus; HIV: Human immunodeficiency virus; DAA: Direct-acting antiviral; AFP: Alpha-fetoprotein; ALBI: Albumin-bilirubin score; APRI: Aspartate aminotransferase to platelet ratio index.

Supplementary Table 2 Cox regression analysis of factors associated with time to occurrence of hepatocellular carcinoma before inverse probability of treatment weighting in patients who achieved sustained virologic response

Variables	Crude HR (95% CI)	*P* value	Adjusted HR (95% CI)	*P* value
Group		0.089		0.212
HCV mono-infection	1 [Reference]		1 [Reference]	
HIV/HCV co-infection	0.18 (0.02–1.30)		0.28 (0.04–2.07)	
Age		0.034		
<65 years	1 [Reference]			
≥65 years	1.91 (1.05–3.48)			
Sex		0.071		
Male	1 [Reference]			
Female	0.58 (0.32–1.05)			
Diabetes mellitus		0.005		0.040
Absent	1 [Reference]		1 [Reference]	
Present	2.59 (1.34–5.02)		2.05 (1.03–3.41)	
Hypertension		0.047		0.129
Absent	1 [Reference]		1 [Reference]	
Present	1.96 (1.01–3.80)		1.71 (0.86–3.41)	
Child-Pugh score		0.756		
A	1 [Reference]			
B	1.08 (0.42–2.73)			

Treatment		0.342		
Peginterferon plus ribavirin	1 [Reference]			
DAA	1.40 (0.70–2.79)			
Fibrosis-4 index		<0.001		0.642
≤3.25	1 [Reference]		1 [Reference]	
>3.25	4.19 (2.23–7.88)		1.14 (0.66–1.94)	
Liver cirrhosis		<0.001		0.003
Absent	1 [Reference]		1 [Reference]	
Present	4.47 (2.50–8.02)		2.65 (1.40–5.02)	
AFP (ng/mL)		0.004		0.943
<20	1 [Reference]		1 [Reference]	
≥20	2.97 (1.42–6.20)		1.00(0.88–1.15)	
ALBI score		0.009		0.876
≤-2.60	1 [Reference]		1 [Reference]	
>-2.60	2.16 (1.20–3.87)		1.05 (0.55–2.01)	
APRI		<0.001		0.012
<1	1 [Reference]		1 [Reference]	
≥1	4.16 (2.15–8.06)		2.75 (1.25–6.05)	

HCV: Hepatitis C virus; HIV: Human immunodeficiency virus; DAA: Direct-acting antiviral; AFP: Alpha-fetoprotein; ALBI: Albumin-bilirubin score; APRI: Aspartate aminotransferase to platelet ratio index.