Steel Memorial Yawata Hospital, Kitakyushu, Japan; [22]Department of Internal Medicine, Hanyang University College of Medicine, Guri Hospital, Guri, Korea; [23]Department of Internal Medicine, Hallym University Dongtan Sacred Heart Hospital, Hwaseong-si, Korea; [24]Liver Clinic, Kajiwara Clinic, Kitakyushu, Japan; [25]Medicine and Biosystemic Science, Kyushu University Graduate School of Medical Sciences, Fukuoka, Japan; [26]Department of Medicine, Kitakyushu Municipal Medical Center, Kitakyushu, Japan; [27]Department of Medicine, Fukuoka City Hospital, Fukuoka, Japan; [28]Department of Internal Medicine, Inje University Haeundae Paik Hospital, Busan, Korea; [29]Center for Liver Disease, National Hospital Organization Kokura Medical Center, Kitakyushu, Japan; [30]Department of Internal Medicine, The Catholic University of Korea College of Medicine, Seoul, Korea; [31]Department of Medicine, Hamanomachi Hospital, Fukuoka, Japan; [32]Department of Internal Medicine, Inje University Sanggye Paik Hospital, Seoul Korea

Aims: Reports suggest HCV-HCC patients do not respond as well to the IFN-free DAAs, but background risks and confounders for treatment failures may not have been adequately controlled. Our goal was to compare SVR12 of DAAs in East Asian patients with HCV-HCC to those without HCC using PSM to balance the HCC and non-HCC groups.

Table 1: Baseline Demographics of Propensity Score Matched (PSM) Patients (n=342)

Characteristic	HCC (n = 171)	No HCC (n = 171)	*P-value*
Male	89 (52.1)	81 (47.4)	0.39
Age	69.5 ± 9.4	68.9 ± 10.0	0.60
Body mass index (BMI)	23.3 ± 3.2	23.1 ± 3.8	0.65
Hypertension	38 (53.5)	17 (40.5)	0.18
Hyperlipidemia	6 (4.7)	7 (4.9)	0.96
Coronary artery disease	1 (1.5)	2 (5.1)	0.28
Chronic renal insufficiency	20 (14.8)	14 (9.3)	0.15
Other cancer			0.69
Prostate	1 (1.9)	1 (3.5)	
Colon	1 (1.9)	1 (3.5)	
Gastric	0 (0.0)	1 (3.5)	
Other	2 (3.9)	1 (3.5)	
Cirrhosis	106 (62.0)	103 (60.2)	0.67
Decompensation	8 (8.5)	7 (6.9)	
Hepatitis C virus (HCV) genotype and subtype			0.078
Genotype 1	**127 (74.2)**	**123 (71.9)**	
Untyped	14 (8.2)	3 (1.8)	
1a	3 (1.8)	1 (0.6)	
1b	110 (64.3)	119 (69.6)	
Genotype 2	**42 (24.6)**	**46 (26.9)**	
Untyped	13 (7.6)	9 (5.3)	
2a	15 (8.9)	26 (15.2)	
2b	11 (6.4)	11 (6.4)	
2a/2c	3 (1.8)	1 (0.6)	
Genotype 6	**2 (1.2)**	**2 (1.2)**	
Prior treatment	72 (42.1)	70 (40.9)	0.36
Prior direct acting antiviral (DAA)	5 (2.9)	10 (5.9)	
Prior non-DAA	67 (39.2)	60 (35.1)	
log10 HCV RNA, mean (SD)	5.6 ± 1.3	5.5 ± 1.3	0.55
Platelets, median (range)	115 (79 - 161)	116 (84 - 161)	0.58
Total bilirubin, mean (SD)	1.1 ± 0.9	1.0 ± 0.7	0.20
ALT, median (range)	46 (33 - 65)	47 (29 - 75)	0.55
DAA treatment initiated			0.53
SOF+RBV (sofosbuvir+ribavirin)	33 (19.3)	45 (26.3)	
LDV/SOF (ledipasvir/SOF)	94 (55.0)	87 (50.9)	
3D (paritaprevir/ritonavir, ombitasvir + dasabuvir)	12 (7.0)	13 (7.6)	
2D (paritaprevir/ritonavir, ombitasvir)	1 (0.6)	0 (0.0)	
DCV+ASV (daclatasvir+asunaprevir)	19 (11.1)	21 (12.3)	
SOF+DCV	3 (1.8)	1 (0.6)	
EBR/GZR (elbasvir+grazoprevir)	1 (0.6)	1 (0.6)	
Other	8 (4.7)	3 (1.8)	

Methods: Data were from 10 study centers comprising of 30 clinical sites in Hong Kong, Japan, Korea, and Taiwan representing the Real-World Evidence from the Asia Liver Consortium for Chronic Hepatitis C (REAL-C) - a registry of patients treated with IFN-free DAAs in routine practice (n=3702). 1:1 PSM matching on cirrhosis, prior treatment, baseline platelet, age, sex, baseline HCV RNA, treatment regimen, baseline ALT, HCV genotype, and BMI was used to balance the groups at baseline.

Results: In our cohort, there were 195 patients with HCC at baseline or prior to DAA initiation and 3507 patients who did not have HCC at baseline. Prior to PSM, HCC patients were significantly older, more likely male, more likely to have renal insufficiency, cirrhosis, and decompensation (all $P<0.004$). After PSM, there were 171 HCC and N=171 non-HCC patients for analysis. As shown in Table 1, there were no significant differences in the baseline characteristics between the matched HCC and non-HCC cohorts. The majority (51-55%) of both groups received LDV/SOF; eight (three HCC, five non-HCC) stopped treatment before completion while ~10-12% had an adverse reaction (most common: anemia [>~5-6%] and fatigue [~3-5%]). There were seven deaths: five in the HCC group (four were liver-related) and two in the non-HCC group (both were non-liver-related). Overall, SVR12 rate was >96% for both groups with no significant differences. (Table 2)

Table 2: Tolerability and Outcomes for PSM Patients with and without HCC

	HCC	No HCC	*P*-value
Death	**5**	**2**	0.11
Non-liver related	1 (0.6)	2 (1.2)	
Liver-related	4 (2.3)	0 (0.0)	
Adverse events	21 (12.9)	17 (10.1)	0.43
Headache	0 (0.0)	2 (3.3)	0.14
Fatigue	3 (4.6)	2 (3.3)	0.71
Insomnia	3 (4.6)	1 (1.6)	0.35
Nausea	0 (0.0)	1 (1.6)	0.30
Arthralgia	1 (1.5)	0 (0.0)	0.33
Myalgia	0 (0.0)	1 (1.6)	0.30
Rash	3 (2.5)	2 (1.5)	0.57
Pruritus	2 (3.0)	0 (0.0)	0.17
Anemia	7 (6.0)	7 (5.2)	0.78
Dyspnea	1 (1.5)	0 (0.0)	0.33
Study discontinuation Non-compliance Psychological side effects Other	**3** 1 (0.8) 0 (0.0) 2 (1.5)	**5** 1 (0.7) 1 (0.7) 3 (2.0)	0.59
SVR-12 rates	**96.8** **95% CI: 93-99**	**96.3** **95% CI: 92-99**	**0.83**

Conclusions: This PSM study compared treatment for HCV patients with/without HCC, finding no difference in treatment tolerability, completion, and cure rates.

Keywords: HCV, HCC, DAA

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Hepatocellular Carcinoma Recurrence after Direct-Acting Antiviral Therapy in Patients with Chronic Hepatitis C: A Korean Multi-Center Retrospective Study

Soon Sun Kim[1], Sun Hyuk Hwang[1], Hyo Jung Cho[1], Do Young Kim[2], Hye Won Lee[2], Su Jong Yu[3], Young Youn Cho[8], Jeong Won Jang[4], Byoung Kuk Jang[5], Chang Wook Kim[6], Hee Yeon Kim[6], Hana Park[7], So Young Yoon[1], Gil Ho Lee[1], Sung Won Cho[1], Jae Youn Cheong[1]

[1]Department of Gastroenterology, Ajou University School of Medicine, Suwon; [2]Department of Internal Medicine, Yonsei University College of Medicine, Seoul; [3]Department of Internal Medicine and Liver Research Institute, Seoul National University College of Medicine; [4]De-

partment of Internal Medicine, College of Medicine, The Catholic University of Korea, Seoul; [5]Department of Internal Medicine, Keimyung University School of Medicine, Daegu; [6]Division of Hepatology, Department of Internal Medicine, Uijeongbu St. Mary's Hospital, College of Medicine, The Catholic University of Korea, Seoul; [7]Department of Internal Medicine, CHA Bundang Medical Center, CHA University, Seongnam, Korea; [8]Department of Internal Medicine, Chung-Ang University Hospital, Seoul, Korea

Aims: The risk factor of hepatocellular carcinoma (HCC) recurrence following direct-acting antiviral (DAA) therapy remains unclear. The aims: of this study were to estimate the rate of HCC recurrence following DAA therapy in Korean patients with chronic hepatitis C (CHC), and to evaluate the risk factors for HCC recurrence after DAA therapy.

Methods: A total of 103 participants with CHC who obtained complete response after HCC treatment were treated with DAA between August 2015 and December 2016 and were followed up until January 2018. HCC treatments were classified as potentially curative included liver resection, radiofrequency ablation, percutaneous ethanol injection, cryoablation and liver transplantation.

Results: Among 103 patients, 82 patients had cirrhosis (79.2%) and 98 patients had Child-Pugh class A (95.1%). HCC stage of the patients was 49.6%, 38.9%, 9.7%, and 2.0% at stage I, II, III, and IV, respectively, according to modified UICC classification. Total duration of HCC treatment was median 2.3 (range, 0.03–143.0) months, and interval from last HCC treatment to start of DAA therapy was median 12.6 (range, 1.5–28.6) months. During the median 15.7 (range, 4.3–29.9) months follow-up, 38 patients (36.9%) experienced tumor recurrence. The median time to recurrence was 22.8 months. The univariate analyses showed that lower platelet count, non-curative HCC treatment, shorter interval from HCC treatment to DAA therapy and longer total HCC treatment duration could be the risk factors for HCC recurrence. In multivariate analysis, shorter interval from HCC treatment to DAA therapy (<12 months) and longer total HCC treatment duration (≥18 months) were found to be the independent risk factors for HCC recurrence (hazard ratio [HR], 2.76; 95% confidence interval [CI], 1.21–6.30; *P*=0.016 and HR, 1.96; 95% CI, 1.00–3.83; *P*=0.049, respectively).

Conclusions: In CHC patients with HCC, DAA therapy should be cautiously performed after sufficient interval without recurrence after complete treatment response.

Keywords: Carcinoma, Hepatocellular, Hepatitis C, Chronic, Direct-acting antiviral agent, Neoplasm recurrence, Risk factors

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Pretreatment NAFLD Activity Score Significantly Predicts Fibrosis Regression in Hepatitis C Patients Receiving Antiviral Therapy

Dong Hyeon Lee, Won Kim, Sae Kyung Joo, Yong Jin Jung, Byeong Gwan Kim, Kook Lae Lee

Department of Internal Medicine, Seoul Metropolitan Government Seoul National University Boramae Medical Center, Seoul, Korea

Aims: Previous studies have reported that antiviral therapy engenders histological improvement and fibrosis regression in patients with hepatitis C. However, the effect of non-alcoholic fatty liver disease (NAFLD) accompanying hepatitis C on histological improvement and fibrosis regression during antiviral therapy remains unclear.

Methods: In this prospective cohort study, a total of 55 patients with hepatitis C underwent paired liver biopsy during antiviral therapy. Among them, 44 patients achieved sustained virologic response (SVR) after antiviral therapy. The NAFLD activity score (NAS) assessment and the quantification of a fibrotic surface area (FSA) were done by a single liver pathologist using paired Masson's trichrome-stained liver tissues for each patient. The relative changes of FSA between before and after antiviral treatment was defined as "[(% of FSA after treatment)-(% of FSA before treatment)]/(% of FSA before treatment)".

Results: Baseline fibrotic surface area was significantly related to Metavir fibrosis stage in only patients attaining SVR (*P*<0.05) but not in all patients with hepatitis C. In univariable analysis, age, serum protein level, and the NAS were significant predictors of the quantitative reduction of FSA during antiviral therapy (all *P*<0.1). In multiple linear regression analysis, serum protein level (*P*=0.07) and the NAS (*P*=0.03) were independent predictors of fibrosis regression. In patients with the NAS < 4, the quantitative reduction of FSA was more frequently observed after antiviral treatment rather than in those with the NAS > or = 4 (*P*=0.02).

Conclusions: The underlying severe NAFLD may hinder fibrosis regression in hepatitis C patients after antiviral treatment. Especially, in patients with the NAS < 4, fibrosis regression may be anticipated by antiviral treatment.

Keywords: HCV, NAFLD, NAFLD activity score, Fibrosis