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# Long-term outcomes of patients with hepatitis C virus–related liver transplantation in a high-volume transplant center

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# Long-term outcomes of patients with hepatitis C virus–related liver transplantation in a high-volume transplant center

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## Abstract

### Background and aim:

Hepatitis C virus (HCV) infection remains a major indication for liver transplantation (LT) worldwide despite the availability of highly effective direct-acting antivirals (DAAs). Reinfection of the graft is almost universal in viremic patients at the time of LT; however, long-term real-world data from predominantly living-donor programs in the DAA era are limited. We evaluated long-term patient and graft outcomes, identified predictors of post-transplant HCV recurrence, and assessed the impact of pre- and post-transplant antiviral strategies in a high-volume transplant center.

### Methods:

In this single-center retrospective cohort study, 98 adults underwent primary LT for HCV-related cirrhosis and/or hepatocellular carcinoma between 2007 and 2022. Demographic, clinical, virological, histological and therapeutic data were obtained from electronic records. Outcomes included overall survival, **HCV recurrence-free survival**, HCV recurrence, fibrosing cholestatic hepatitis (FCH), graft failure, and sustained virological response (SVR). Predictors of recurrence were examined using univariate and multivariable logistic regression, and predictors of mortality were evaluated using Cox proportional-hazards models. Model discrimination for recurrence was assessed using receiver-operating characteristic (ROC) analysis. An exploratory propensity score–matched analysis assessing pre-transplant DAA exposure was performed and is reported in the Supplementary Appendix due to the very small treated subgroup (n=8).

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**Results:**

The cohort included 59 women (60.2%) and 39 men; 90.8% received living-donor grafts. Genotype 1b predominated (80.4%). Pre-transplant DAA therapy was administered to 8 patients (8.2%), whereas 78 (85.7%) received no antiviral treatment prior to LT and the remainder received interferon-based regimens. Mean overall survival after LT was 145.0 ± 6.8 months. One-, 3-, 5- and 10-year overall survival rates were 95.7%, 88.3%, 83.5% and 75.0%, respectively; HCV recurrence-free survival rates were 78.7%, 54.3% and 39.6% at 1, 3 and 5 years. HCV recurrence occurred in 49 patients (50.0%) after a mean of 18.3 months. In multivariable logistic regression, pre-transplant HCV RNA positivity (OR 7.9; 95% CI 2.8–22.3; $p<0.001$) and absence of pre-transplant DAA therapy (OR 5.4; 95% CI 1.5–20.3; $p=0.009$) were independent predictors of recurrence. The recurrence model yielded an AUC of 0.832 with 78.2% sensitivity and 80.5% specificity. Among recurrent cases, 44/49 (89.7%) achieved SVR24; regimen-specific SVR24 was 100% with glecaprevir/pibrentasvir and 96.9% with ledipasvir/sofosbuvir. In multivariable Cox regression, FCH was independently associated with mortality (HR 2.9; 95% CI 1.1–7.8; $p=0.03$). Overall survival did not differ significantly between patients with and without recurrence.

**Conclusion:**

In this long-term, predominantly living-donor cohort, HCV-related LT provided excellent survival, exceeding 80% at 5 years and 75% at 10 years. Although virological recurrence remained common, the combination of pre-transplant DAA therapy and highly effective post-transplant regimens minimized its clinical consequences, yielding survival comparable to that of non-recurrent patients. Pre-transplant viremia and lack of DAA exposure were powerful predictors of recurrence, whereas FCH was the main determinant of late mortality. Systematic use of DAA therapy before and after LT should be considered standard of care in HCV-infected transplant candidates.

**Keywords:** hepatitis C virus; liver transplantation; recurrence; direct-acting antivirals; fibrosing cholestatic hepatitis; survival.

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**What is already known**

- HCV remains a major global indication for LT and causes substantial post-transplant morbidity and mortality. (1–5)
- Graft reinfection is virtually universal in viremic patients at LT and can progress rapidly to cirrhosis, graft failure or FCH. (5–10)
- DAA therapy has dramatically improved SVR rates and outcomes after LT, but most published data derive from deceased-donor cohorts with relatively short follow-up. (11–18)

**What this study adds**

- In a predominantly living-donor cohort with more than a decade of follow-up, overall survival after HCV-related LT exceeded 80% at 5 years and 75% at 10 years despite a 50% recurrence rate.

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- Pre-transplant HCV RNA positivity and absence of pre-transplant DAA therapy independently predicted recurrence, and PSM demonstrated 0% recurrence among patients who received DAA therapy before LT.
- FCH—not recurrence per se—was the principal determinant of late mortality, emphasizing the need to identify and aggressively manage this high-risk phenotype.

**Introduction**

Hepatitis C virus (HCV) infection is a leading cause of chronic liver disease worldwide. It can progress from asymptomatic infection to chronic hepatitis, advanced fibrosis, cirrhosis and hepatocellular carcinoma (HCC). (1–3) HCC, frequently driven by chronic viral hepatitis, ranks among the most common malignancies and one of the leading causes of cancer-related death globally. (2)

In 2016, the World Health Organization (WHO) set the ambitious goal of eliminating viral hepatitis as a public health threat by 2030. Despite substantial progress, an estimated 57 million individuals were still living with chronic HCV infection in 2020, and approximately 300,000 HCV-related deaths occurred that year. (3) Although antiviral therapy has improved dramatically with the advent of DAAs, many patients still present with advanced liver disease or HCC and ultimately require LT.

Historically, HCV has been the main indication for LT in Europe, North America, Australia and Japan. (4,5) Recurrent HCV following LT is almost universal among patients with detectable viremia at the time of transplantation. Without effective antiviral treatment, up to one-third of these patients progress to cirrhosis within five years, and long-term patient and graft survival are significantly reduced. (5–10) Several donor- and recipient-related risk factors for rapid progression have been described, including advanced donor age, high pre-transplant viral load, intense immunosuppression, and graft-related complications such as acute rejection and FCH. (5–10)

The introduction of DAAs has dramatically changed this landscape. Compared with interferon-based regimens, DAAs yield higher SVR rates, shorter treatment courses and improved tolerability both before and after transplantation. (11–18) Pre-transplant DAA therapy can prevent recurrence by achieving SVR prior to LT or, if transplantation occurs while on therapy, by markedly reducing viral inoculum at the time of graft reperfusion. (11–13,19–21) Post-transplant DAA regimens are also highly effective in eradicating recurrent HCV and improving graft function. (14–18,22–25) However, most published series originate from deceased-donor programs and focus on short- to medium-term outcomes. Long-term data from high-volume centers performing predominantly living-donor LT are scarce, and the relative contributions of pre-transplant viremia, DAA exposure and FCH to long-term survival in the DAA era are not fully defined.

Furthermore, several earlier reports relied mainly on univariate analyses. Without multivariable models or propensity score methods, it is difficult to disentangle the independent effect of pre-transplant antiviral therapy from confounding clinical variables such as age, MELD score, donor type and comorbidities.

Therefore, this study aimed to provide long-term, real-world data from a high-volume transplant center in which living-donor procedures predominate. Specifically, we sought to:

(i) describe long-term overall and disease-free survival after HCV-related LT.

(ii) quantify the incidence and timing of HCV recurrence, cirrhosis, graft failure and FCH.

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(iii) identify independent predictors of recurrence and mortality using multivariable logistic and Cox regression.

(iv) evaluate the discriminative performance of a multivariable recurrence model using ROC analysis; and

(v) assess the impact of pre-transplant DAA therapy on recurrence using propensity score matching.

We hypothesized that pre-transplant DAA therapy would significantly reduce the risk of post-transplant HCV recurrence and that FCH would be the main determinant of mortality in the DAA era.

## Materials and Methods

### Study design and population

We conducted a single-center retrospective cohort study at the Liver Transplantation Institute of Inonu University, a high-volume tertiary referral center in Turkey. All consecutive adult recipients (≥18 years) who underwent primary LT for HCV-related liver disease between January 2007 and December 2022 were screened for eligibility.

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### Study population

Inclusion criteria were:

(i) chronic HCV infection documented by serology and/or HCV RNA.

(ii) decompensated cirrhosis and/or HCC in which HCV was the primary etiologic factor; and

(iii) at least three months of post-transplant follow-up.

Exclusion criteria were:

(i) multi-organ transplantation.

(ii) re-transplantation for causes other than recurrent HCV; and

(iii) missing essential data regarding pre-transplant HCV status, antiviral therapy or survival.

### Pre-transplant assessment and listing

All LT candidates underwent a standardized evaluation including clinical assessment, laboratory testing (complete blood count, liver and kidney function tests, coagulation profile), viral serology, and cross-sectional imaging (abdominal ultrasonography and contrast-enhanced computed tomography or magnetic resonance imaging). Upper gastrointestinal endoscopy was performed when indicated to assess portal hypertension and varices.

Candidacy for LT was determined according to international guidelines, considering MELD score, presence and extent of HCC, comorbidities and performance status. (2,5) Most procedures involved living-donor right-lobe grafts, reflecting national organ-availability patterns.

### Antiviral therapy before transplantation

Pre-transplant antiviral strategies evolved over the study period.

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- Interferon era: In the early years, pegylated interferon with or without ribavirin was used selectively in patients with relatively preserved liver function.
- DAA era: After the introduction of DAAs, sofosbuvir-based and ledipasvir/sofosbuvir combinations became available.

In our cohort, 78 patients (85.7%) received no antiviral treatment prior to LT. Interferon-based regimens were used in 5 patients (pegylated interferon and/or interleukin plus ribavirin). True DAA-based therapy before LT was administered to 8 patients (8.2%): ledipasvir/sofosbuvir in 5, dasabuvir-containing regimens in 2 and sofosbuvir monotherapy in 1. These patients constituted the "pre-LT DAA" group in subsequent analyses. For era-based analyses, the study period was classified as pre-DAA era (transplants performed before 2015) and DAA era (transplants performed in/after 2015), reflecting the introduction and routine availability of direct-acting antivirals in clinical practice at our center.

**Transplant procedure and immunosuppression**

LT was performed using standard techniques for living-donor or deceased-donor transplantation. Vascular and biliary anastomoses followed institutional protocols.

Post-transplant immunosuppression consisted primarily of tacrolimus-based regimens, often combined with mycophenolate mofetil and corticosteroids. A minority of patients received cyclosporine or everolimus. Steroids were tapered gradually according to clinical course, rejection episodes and comorbidities.

**Post-transplant follow-up and definitions**

Recipients were followed at regular intervals with clinical evaluation, biochemical tests and imaging. HCV RNA was measured periodically and whenever graft dysfunction was suspected. Liver biopsy was performed in cases of unexplained aminotransferase elevation, suspected rejection, FCH or fibrosis progression.

- HCV recurrence was defined as re-appearance of HCV RNA and/or histological features of recurrent hepatitis in a previously HCV-negative or SVR patient after LT.
- Fibrosing cholestatic hepatitis (FCH) was defined as a severe cholestatic form of recurrent HCV characterized by (i) a cholestatic biochemical profile (predominant hyperbilirubinemia and cholestatic enzyme elevation), (ii) high-level HCV viremia, (iii) early post-transplant onset, and (iv) compatible histopathology. All FCH diagnoses were biopsy-confirmed, with histological features including marked hepatocellular ballooning/swelling, prominent cholestasis, periportal or pericellular fibrosis, and ductular reaction.
- Graft cirrhosis or failure was defined by a combination of clinical signs of decompensation, imaging evidence of cirrhosis and portal hypertension and, when available, histological confirmation.

**Antiviral therapy after transplantation**

Post-transplant antiviral management reflected the transition from interferon to DAA therapy. Overall, 48 patients (48.9%) did not require antiviral treatment after LT, while 49 patients were treated for recurrent infection. Regimens included:

- glecaprevir/pibrentasvir (Maviret) in 7 patients (14.3%);
- ledipasvir/sofosbuvir (Harvoni) in 32 (65.3%);
- sofosbuvir plus ribavirin in 5 (10.2%);
- ribavirin plus interferon-based combinations in 5 (10.2%).

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SVR at week 12 and 24 and at 3- and 6-month time points was recorded for each regimen.

**Outcomes**

Primary outcomes were:

1. **Overall survival:** time from LT to death from any cause or last follow-up;
2. **HCV recurrence** as defined above.

Secondary outcomes included HCV recurrence-free survival (time from LT to first documented post-transplant HCV recurrence), fibrosing cholestatic hepatitis (FCH), graft failure, need for re-transplantation, and sustained virological response (SVR) rates.

**Statistical analysis**

Continuous variables were summarized as mean ± standard deviation (SD) or median (interquartile range), and categorical variables as counts and percentages. Group comparisons used $\chi^2$ or Fisher's exact tests for categorical variables and Student's t-test or Mann–Whitney U test for continuous variables, as appropriate.

**Survival analysis:** Overall survival and HCV recurrence-free survival probabilities were estimated with the Kaplan–Meier method and compared using the log-rank test. Univariate Cox proportional-hazards models were fitted to explore associations between candidate predictors (age, sex, MELD score, pre-transplant HCC, comorbidities, FCH, rejection, pre-transplant HCV RNA positivity and pre-transplant DAA therapy) and mortality. Variables with $p < 0.10$ or strong clinical relevance were entered into a multivariable Cox model.

**Predictors of recurrence:** Univariate logistic regression evaluated potential predictors including pre-transplant HCV RNA status, age, sex, MELD score, pre- and post-transplant HCC, pre- and post-transplant antiviral therapy, FCH, rejection and comorbidities. Significant variables were incorporated into a multivariable logistic model to identify independent predictors of recurrence. Sensitivity analysis was performed restricting the cohort to the DAA era (transplants performed in/after 2015) to reduce historical confounding and differential follow-up.

**ROC analysis:** Discriminative performance of the multivariable recurrence model was assessed by constructing a ROC curve and calculating the AUC, sensitivity and specificity.

To evaluate factors associated with post-transplant HCV recurrence, we prioritized multivariable logistic regression as the primary inferential framework. Because the number of patients who received pre-transplant DAA therapy was very small (n = 8), any matched or small-sample comparative analysis would be statistically fragile; therefore, propensity score matching (PSM) was performed only as an exploratory, hypothesis-generating analysis and is reported in the Supplementary Appendix (Supplementary Methods and Supplementary Table S1).

**Results**

**Baseline characteristics**

The study included 98 patients, 59 (60.2%) female and 39 (39.8%) male. Donors were predominantly male (61.8%). Living-donor LT accounted for 89 cases (90.8%), with only 9 deceased-donor grafts (9.2%). Among patients with available genotype data, genotype 1b was most common (41/51; 80.4%), followed by genotypes 1a (5.9%), 3a (7.8%), 2a (2.0%), 4d (2.0%) and 5a (2.0%). Before transplantation, 30 patients (30.6%) had hepatocellular carcinoma (HCC), while no de novo HCC occurred after LT. Pre-transplant HCV RNA was positive in 37 of 72 tested patients (51.4%). Comorbidities were frequent: 9.2% had diabetes mellitus, 11.2% hypertension, 9.2% hypothyroidism and 6.1% hepatitis B virus co-infection.

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No de novo HCC was observed after LT in this cohort. However, given the retrospective design and the primary focus of the present analysis on HCV virological recurrence, HCC recurrence events and HCC-attributable deaths were not analyzed as prespecified endpoints in the current dataset. Accordingly, the "recurrence-free" endpoint reported in this study refers specifically to HCV recurrence-free survival (i.e., freedom from post-transplant HCV recurrence) and should not be interpreted as oncologic disease-free survival.

Regarding antiviral treatment history, 78 patients (85.7%) had never received HCV therapy before LT. Eight patients (8.2%) had been treated with DAAs (ledipasvir/sofosbuvir, dasabuvir-based regimens or sofosbuvir), while a small number received interferon-based regimens. After transplantation, 48 patients (48.9%) did not require antiviral therapy, whereas 49 received one or more regimens for recurrent infection, most commonly ledipasvir/sofosbuvir (32 patients, 32.6%) or glecaprevir/pibrentasvir (7 patients, 7.1%). Baseline demographic and clinical data for the entire cohort are summarized in Table 1.

Because pre-transplant DAA therapy became available only in the later years of the study period, the pre-LT DAA subgroup had shorter potential follow-up than historical controls. We therefore accounted for transplant timing in adjusted models and performed sensitivity analyses restricted to the DAA era.

**Table 1: Patient demographic and clinical data.**
**Insert Table 1 About Here**

**Table 1.** Patient demographic and clinical characteristics of the study cohort (N = 98). Data are presented as number (n) and percentage (%). Variables include recipient sex, donor sex, HCV genotype, transplantation type, HCV treatment before and after transplantation, hepatocellular carcinoma (HCC) status, HCV RNA status before and after transplantation, HCV recurrence, comorbidities, sustained virologic response (SVR; overall and at 3 and 6 months), re-transplantation, cirrhosis/graft failure, fibrosing cholestatic hepatitis (FCH), cytomegalovirus (CMV) status, immunosuppressive regimen, rituximab use, and rejection-related treatments.

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Because pre-transplant DAA therapy became available only in the later years of the study period, the pre-LT DAA subgroup had shorter potential follow-up than historical controls. We therefore accounted for transplant timing in adjusted models and performed sensitivity analyses restricted to the DAA era.

**Long-term survival**

The mean post-transplant survival time for the entire cohort was 145.0 ± 6.8 months (approximately 12.1 years). One-, 3-, 5- and 10-year overall survival rates were 95.7%, 88.3%, 83.5% and 75.0%, respectively. HCV recurrence-free survival at 1, 3 and 5 years was 78.7%, 54.3% and 39.6%. Patients with recurrence who eventually achieved SVR had excellent outcomes, with a mean survival of 162.4 ± 7.5 months and 10-year survival approaching 88.7%. Kaplan–Meier estimates for overall survival, HCV recurrence-free survival, and survival among patients with recurrence are presented in Table 2.

**Table 2: Kaplan-Meier analysis (survival>3 months)**

**Table 2 Insert About Here**

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Kaplan–Meier analysis is presented for overall survival (n = 98), HCV recurrence-free survival (n = 98), and survival among patients with HCV recurrence (n = 49).

Univariate and multivariable Cox proportional hazards regression analyses for overall survival are summarized in Table 3. In the multivariable model, fibrosing cholestatic hepatitis (FCH) remained independently associated with mortality (adjusted HR 2.9, 95% CI 1.1–7.8; p=0.03), whereas rejection, MELD score, age, sex, and pre-transplant DAA exposure were not statistically significant. Cox regression analyses restricted to patients with recurrence are provided in Supplementary Table S3.

**Predictors of post-transplant HCV recurrence and model performance**

Univariate and multivariable logistic regression analyses for predictors of post-transplant HCV recurrence are presented in Table 4. Pre-transplant HCV RNA positivity (adjusted OR 7.9, 95% CI 2.8–22.3; p<0.001) and absence of pre-transplant DAA therapy (adjusted OR 5.4, 95% CI 1.5–20.3; p=0.009) were independently associated with recurrence.

Subgroup analyses of recurrence rates by genotype, age category, and donor type are provided in Supplementary Table S1. Given the very small number of patients receiving pre-transplant DAAs (n=8), propensity score matching was performed only as an exploratory analysis and is reported in Supplementary Table S2.

Model discrimination for recurrence was good, with an AUC of 0.832 (sensitivity 78.2%, specificity 80.5%) as shown in Figure 1; corresponding ROC summary metrics are provided in Supplementary Table S4.

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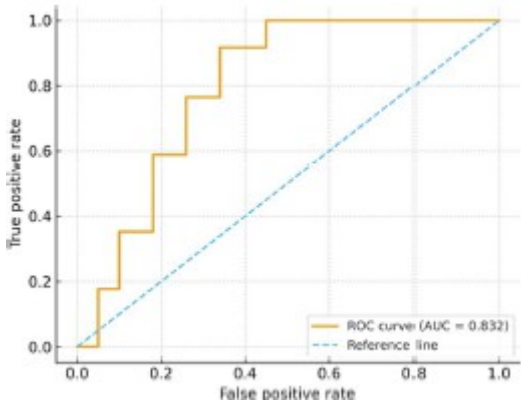
**Table 4: Logistic regression for predictors of post-transplant HCV recurrence (univariate and multivariable).**
**Insert Table 4 About Here**

**Figure 1: ROC curve for prediction of post-transplant HCV recurrence.**
**Insert Figure 1 About Here**



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### Antiviral therapy and SVR in recurrent disease

Among the 49 patients with recurrence, 44 (89.7%) achieved SVR24. Regimen-specific outcomes were: glecaprevir/pibrentasvir SVR24 100% (7/7), ledipasvir/sofosbuvir SVR24 96.9% (31/32), sofosbuvir plus ribavirin SVR24 80.0% (4/5), and interferon-based triple therapy SVR24 0% (0/2). Regimen-specific SVR outcomes among recurrent patients are summarized in Table 5.

**Table 5: SVR rates among patients with post-transplant HCV recurrence (n=49), by regimen.**
**Insert Table 5 About Here**

### HCV recurrence and clinical outcomes

Overall, 49 patients (50.0%) developed HCV recurrence during follow-up. Recurrence typically occurred within the first two years after LT, with a mean time of 18.3 months. Key adverse clinical outcomes among patients with recurrence (re-transplantation, graft failure/cirrhosis, FCH, and acute rejection) are summarized in Table 6.

Among recurrent patients, re-transplantation occurred in 2 (4.1%), graft cirrhosis or failure in 3 (6.1%), FCH in 5 (10.2%) and acute rejection in 4 (8.2%). Among the five patients who developed FCH, the diagnosis occurred in the early post-transplant period and was characterized by a cholestatic biochemical profile with high-level viremia. After FCH diagnosis, clinical deterioration was typically rapid, culminating in death and/or graft failure in a subset of cases. Post-FCH antiviral management was individualized: DAA therapy was initiated promptly when clinically feasible using contemporary regimens available at our center; however, in cases with advanced graft dysfunction or late presentation, treatment response was limited and outcomes remained poor.

**Table 6: Clinical outcomes among patients with post-transplant HCV recurrence (n=49).**
**Insert Table 6 About Here**

## Discussion

### Principal findings

In this long-term, real-world cohort from a high-volume center where living-donor procedures predominate, we observed three main findings. First, overall survival after HCV-related LT was excellent, exceeding 80% in 5 years and 75% in 10 years, despite a 50% recurrence rate. Second, pre-transplant HCV RNA positivity and absence of pre-LT DAA therapy were strong, independent predictors of recurrence, with a multivariable model showing good discriminative performance (AUC 0.832). In adjusted Cox regression, FCH showed a strong association with mortality and remained independently associated with adverse survival. However, because FCH events were infrequent in this cohort (n = 5), the precision of hazard estimates is limited, and the findings should be interpreted cautiously. Clinically, these results underscore that while most recurrent HCV can be effectively managed in the DAA era, the subset who develop FCH represents a distinct high-risk phenotype requiring early recognition (prompt cholestasis-focused evaluation and timely biopsy) and rapid initiation of potent antiviral therapy.

These findings expand on previous reports by combining robust multivariable modelling, ROC analysis and PSM with long-term follow-up in a predominantly living-donor context.

### High recurrence but preserved survival in the DAA era

The 50% recurrence rate in our cohort is comparable to earlier series in which nearly all viremic patients developed recurrent infection after LT. (6–10,15–17) Historically, such

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recurrence translated into rapid fibrosis progression, early cirrhosis and inferior survival compared with LT for other etiologies. (6–10)

In contrast, we observed no significant difference in overall survival between patients with and without recurrence, and recurrent patients who achieved SVR had excellent 10-year survival approaching 90%. This shift reflects the profound impact of DAA therapy. Multiple DAA-era studies have reported SVR rates above 90% and improved graft outcomes in LT recipients. (11–18,19–25) Our regimen-specific SVR24 rates—100% with glecaprevir/pibrentasvir and 96.9% with ledipasvir/sofosbuvir—are consistent with these data and demonstrate that recurrent HCV is now a largely controllable complication, provided that potent therapy is available and initiated promptly.

Thus, in contemporary practice, recurrence should no longer be equated with poor prognosis in centers where DAA therapy is widely accessible and used aggressively.

**Mechanistic interpretation: why pre-transplant viremia and DAA exposure matter**

Our analyses highlight the central importance of pre-transplant viral status and DAA therapy. Pre-LT HCV RNA positivity was associated with nearly eightfold higher odds of recurrence, even after adjustment, while pre-LT DAA therapy was strongly protective in both multivariable and PSM analyses.

Mechanistically, these findings are plausible. At the moment of graft reperfusion, viremic patients expose the new liver to a large inoculum of infectious virions in the context of intense immunosuppression. High baseline viremia has been linked to more rapid fibrosis progression, FCH and graft failure in previous work. (5–10,15,16) By achieving SVR or at least markedly suppressing viral load before LT, DAA therapy reduces or eliminates this inoculum, thereby lowering the probability of successful graft colonization. (11–13,19–21,27,28)

Our findings support the biological plausibility that reducing or eliminating viremia before graft reperfusion lowers the probability of graft reinfection under intense immunosuppression. Importantly, however, the number of patients who received pre-transplant DAAs in our cohort was very small (n = 8), reflecting real-world access and timing constraints over the long study period. Therefore, while the adjusted association observed in multivariable modeling is consistent with prior literature, any small-sample comparative analyses (including matching approaches) should be interpreted as exploratory and hypothesis-generating rather than definitive evidence of causal effects.

**Fibrosing cholestatic hepatitis as a driver of mortality**

Although only 5 of 49 recurrent patients developed FCH, this complication emerged as the only independent predictor of mortality in multivariable Cox regression. This finding aligns with previous reports describing FCH as a particularly aggressive form of recurrent HCV characterized by extremely high viral loads, profound cholestasis and rapid progression to graft failure. (15,29)

Our results emphasize that the clinical impact of recurrence is heterogeneous. The majority of recurrent patients, especially those treated with DAAs, maintain excellent long-term graft function. Conversely, the small subsets who develop FCH have markedly worse outcomes and require early recognition, prompt initiation of highly potent antiviral therapy and careful modulation of immunosuppression. From a clinical perspective, FCH should be considered a distinct high-risk phenotype within the spectrum of recurrent HCV after LT.

**Comparison with previous studies**

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Pre-DAA studies reported 5-year patient survival rates of 65–76% after HCV-related LT, with recurrent HCV serving as a major cause of graft loss and death. (6–10) Many of these series also identified advanced donor age, high viral load and intense immunosuppression as risk factors for aggressive recurrence. (5–10,15–17)

Our 5-year survival rate of 83.5% and 10-year rate of 75.0% compare favorably with those historical data and are in line with recent DAA-era studies showing improved outcomes. (11–18,19–25) Importantly, our cohort differs from many Western series in its high proportion of living-donor grafts (>90%), yet survival outcomes are at least comparable. This suggests that living-donor LT in experienced centers can achieve excellent long-term results in HCV-infected patients.

Few previous reports have combined long-term follow-up with multivariable models, ROC analysis and PSM. By doing so, our study provides more robust evidence that pre-LT DAA therapy has an independent protective effect against recurrence and that FCH is the principal determinant of mortality in the DAA era.

**Clinical implications**

Our findings have several practical implications for clinicians managing HCV-infected LT candidates and recipients:

1. Systematic evaluation for DAA therapy before LT should be standard practice. Whenever liver function and logistics allow, achieving SVR or at least substantial viral suppression prior to transplantation can dramatically reduce recurrence risk.
2. Early post-LT initiation of potent DAA regimens in patients with recurrence is essential and can normalize survival to that of non-recurrent patients.
3. Close monitoring for FCH, including attention to cholestatic patterns of liver biochemistry and timely biopsy, is crucial, as this subgroup carries the highest mortality risk.
4. In high-volume centers with strong expertise in living-donor procedures, HCV infection should not be considered a contraindication to LT, provided that comprehensive antiviral strategies are implemented.

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Taking together, these points suggest that structured pre- and post-transplant antiviral protocols can transform HCV-related LT from a high-risk indication into one with long-term outcomes comparable to other etiologies.

**Strengths and limitations**

Strengths of this study include:

- a relatively large single-center cohort with long-term follow-up exceeding a decade.
- a predominantly living-donor population, which is under-represented in the literature.
- application of multivariable logistic and Cox models, ROC analysis and PSM to rigorously assess predictors of recurrence and survival.

Because pre-transplant DAA therapy became available only in the later years of the study period, the pre-LT DAA subgroup had shorter potential follow-up than historical controls. We therefore accounted for transplant timing in adjusted models and performed sensitivity analyses restricted to the DAA era.

A key limitation is the very small number of patients who received pre-transplant DAA therapy (n = 8), which limits power and precision for comparisons involving this subgroup and increases the risk of model instability in small-sample methods. Accordingly, we

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emphasize multivariable regression as the primary analytic approach and present any matched analyses only as exploratory, hypothesis-generating results in the Supplementary Appendix.

Finally, findings reflect the experience of a single high-volume center and may not be directly generalized to smaller programs or those with different donor profiles and immunosuppression strategies.

**Conclusion**

In this long-term cohort from a high-volume, predominantly living-donor transplant center, LT for HCV-related liver disease achieved excellent outcomes, with 5-year survival exceeding 80% and 10-year survival reaching 75%. Although HCV recurrence remained frequent, pre-transplant viremia and the absence of DAA therapy were the main determinants of recurrence, whereas FCH was the principal determinant of mortality. Importantly, when recurrent infection was treated promptly with potent DAAs, survival did not differ from that of patients without recurrence.

These findings support a management strategy in which pre-LT DAA therapy is pursued whenever feasible and post-LT recurrence is treated aggressively, with particular vigilance for FCH. Such an approach can deliver long-term outcomes for HCV-infected patients that are comparable to those of other LT indications and contribute meaningfully to the broader goal of HCV elimination.

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**Abbreviations**

DAAs :direct-acting antivirals

LT: liver transplantation

HCC:hepatocellular carcinoma

ALT :alanine transaminase

SVR:sustained virological response

**Author contributions**

EA participated in data analysis and interpretation and article drafting. EA, MH, YB participated in data analysis and interpretation and article drafting. SY, VI, EA participated in data analysis and interpretation and article drafting. MA, HGG participated in data analysis and interpretation and in critically revising the manuscript for important intellectual content. EA, MH participated in the study design, data analysis and interpretation and gave final approval for publication. All authors read and approved of the final manuscript.

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## Availability of data and materials

The datasets used and/or analysed during the current study are available from the corresponding author on reasonable request.

## Declarations

### Ethics approval and consent to participate

This study was conducted according to the principles expressed in the Declaration of Helsinki and approved by the Ethics Committee of Inonu University in line with the Declaration of Helsinki (19-03-2024**2024/5697). Information an Informed consent was obtained from all patients.

### Consent for publication

Not applicable.

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## References


1. Gökahmetoğlu S, Polat C, Atalay MA, Sezgin GC, Ergör G, Aygen … doi: 10.5152/tjg.2022.21822. PMID: 36098364; PMCID: PMC9797710.
2. Akarca US, Unsal B, Sezgin O, Yalcin K, Akdogan M, Gonen C, G… doi: 10.5152/tjg.2021.201171. PMID: 34876392; PMCID: PMC8975510.
3. Martinello M, Solomon SS, Terrault NA, Dore GJ. Hepatitis C. …:1085–1096. doi: 10.1016/S0140-6736(23)01320-X. PMID: 37741678.
4. Berenguer M. Liver transplant 2002;8:S14.
5. Watt K, Veldt B, Charlton M. A practical guide to the management of HCV infection following liver transplantation. … Transplantation. 2009;87(10):1573–1581. doi: 10.1111/j.1600-6143.2009.02702.x. Epub 2009 Jun 16. PMID: 19538491.
6. Zekry A, Whiting P, Crawford DH, Angus PW, Jeffrey GP, Padbury R, et al. Impact of immunosuppression on hepatitis C recurrence after liver transplantation. J Liver Transpl Surg. 2003 Apr;9(4):339–347. doi: 10.1053/jlts.2003.50063. PMID: 12682883.
7. Bonaccorsi-Riani E, Sempoux C, Piette N, Julliard O, Kabamba B, Goffin E, et al. Fibrosing cholestatic hepatitis in liver transplantation: clinical and histological features. Acta Gastroenterol Belg. 2012 Dec;75(4):411–418. PMID: 23402084.
8. Kim JM, Lee KW, Song GW, Jung BH, Lee HW, Yi NJ, Kwon CH, Hwang S, et al. Outcome of hepatitis C virus infection after adult living donor liver transplantation: a single-center experience with interferon-based antiviral therapy. Clin Mol Hepatol. 2016;22:227–236. doi: 10.3350/cmh.2015.0031. Epub 2015 Dec 29. PMID: 26793691; PMCID: PMC4717607.

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9. Singal AK, Guturu P, Hmoud B, Kuo YF, Salameh H, Wiesner RH. Evolving frequency and outcomes of liver transplantation based on etiology of liver disease. Transplantation. 2013;95:755–760. [PubMed] [Google Scholar] [Ref list].

10. Adam R, Karam V, Delvart V, O'Grady J, Mirza D, Klempnauer J, et al. Evolution of indications and results of liver transplantation in Europe. J Hepatol. 2012;57:675–688. [PubMed] [Google Scholar] [Ref list].

11. Lim N, Singh D, Jackson S, Lake JR. Recurrence of Hepatocellular Carcinoma After Liver Transplantation in the Direct Acting Antiviral Era. Prog Transplant. 2021 Jun;31(2):155–164. doi: 10.1177/1526924820951034/000510341. Epub 2020 Sep 14. PMID: 33173777; PMCID: PMC7590751.

12. Kauffman-Ortega E, Ruiz-Manriquez J, Olivas-Martinez A, Cordero-Perez P, et al. Direct-acting antivirals and outcome of liver transplantation for hepatitis C in the MELD era. Arch Med Res. 2021;52:453–460. doi: 10.1016/j.arcmed.2021.04.003. Epub 2021 May 7. PMID: 33966917.

13. Arora SS, Axley P, Ahmed Z, Satapathy SK, Wong R, Kuo YF, et al. Hepatitis C virus infection, direct-acting antivirals, and post-liver transplant outcomes: a contemporary United States analysis. Transpl Int. 2019;32:854–864. doi: 10.1111/tri.13424. Epub 2019 Mar 26. PMID: 30866110.

14. Guillouche P, Feray C. Systematic review: anti-viral therapy of recurrent hepatitis C after liver transplantation. Aliment Pharmacol Ther. 2011;33:163–174. [PubMed] [Google Scholar] [Ref list].

15. Burra P, De Martin E, Zanetto A, Senzolo M, Russo FP, et al. Hepatitis C virus infection in liver transplantation: who, when, how? Transpl Int. 2015;28:35–52. doi: 10.1111/tri.12642. Epub 2015 Aug 11. PMID: 26199060.

16. Samonakis DN, Germani G, Burroughs AK. Immunosuppression and recurrent viral hepatitis after liver transplantation. J Hepatol. 2012;56:973–983. doi: 10.1016/j.jhep.2011.06.031. Epub 2011 Sep 29. PMID: 21963518.

17. Terrault NA, Shiffman ML, Lok AS, Saab S, Tong L, Brown RS Jr, et al. Outcomes in hepatitis C patients undergoing living versus deceased donor liver transplantation. Liver Transpl. 2007;13:122–129.

18. Çekin AH, Güner R, Çağkan İnkaya A, Oğuz D, Özdemir O, … Real-life experience with direct acting antiviral therapy for recurrent hepatitis C after liver transplantation in Turkey. Turk J Gastroenterol. 2023;34:… doi: 10.5152/tjg.2023.22749. PMID: 37565793; PMCID: PMC10645295.

19. Verna EC, Shetty K, Lukose T, Terry N, Mentore K, Olsen SK, et al. Low post-transplant survival in patients with hepatitis C virus–associated fibrosing cholestatic hepatitis. Liver Int. 2015;35:510–517. [PubMed] [Google Scholar] [Ref list].

20. Sulkowski MS, Vargas HE, Di Bisceglie AM, Kuo A, Reddy KR, Lim JK, et al. Safety and efficacy of sofosbuvir-containing regimens for hepatitis C in patients with decompensated cirrhosis or post–liver transplantation: the HCV-TARGET study. Hepatology. 2014;60(S1):660A. [Google Scholar] [Ref list].

21. Kwo PY, Mantry PS, Coakley E, Te HS, Vargas HE, Brown R Jr, et al. An interferon-free antiviral regimen for HCV after liver transplantation. N Engl J Med.

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2014;371:2375–2382. doi: 10.1056/NEJMoa1408921. Epub 2014 Nov 11. PMID: 25386767.

22. Choudhary NS, Saigal S, Gautam D, Saraf N, Rastogi A, Goja S, Bhangui P, Soin AS. Efficacy and Safety of Sofosbuvir-based Regimens in Treatment of Recurrent Hepatitis C Virus Infection after Living Donor Liver Transplantation. J Clin Exp Hepatol. 2018;8:3–10. doi: 10.1016/j.jceh.2017.08.004. Epub 2017 Sep 1. PMID: 29892173; PMCID: PMC5992320.
23. Qu Y, Guo Y, Li T. Efficacy and safety of sofosbuvir-based interferon-free therapies for hepatitis C in liver transplant recipients: a meta-analysis. Clin Res Hepatol Gastroenterol. 2017;32:740–748. [PubMed] [Google Scholar] [Ref list].
24. Beinhardt S, Al-Zoairy R, Kozbial K, Stättermayer AF, Maieron A, Stauber R, et al. Long-term outcomes of sofosbuvir-based therapy in liver transplant recipients with recurrent hepatitis C virus infection. Liver Int. 2018;38:1184–1197. doi: 10.1111/liv.13652. Epub 2018 Jan 31. PMID: 29197145.
25. Iacob S, Cerban R, Pietrareanu C, Ester C, Iacob R, Gheorghe C, Popescu I, Gheorghe L. 100% sustained virological response in liver transplant recipients with recurrent hepatitis C virus infection treated with direct-acting antivirals. J Gastrointestin Liver Dis. 2018;27(2):139–144. doi: 10.15403/jgld.2014.1121.272.100. PMID: 29922758.
26. Kwo PY, Mantry PS, Coaldey E, et al. An interferon-free antiviral regimen for HCV after liver transplantation. N Engl J Med. 2014;371:2375–2382. doi:10.1056/NEJMoa140892.
27. Carrai P, Morelli C, Cordone G, et al. The Italian compassionate use of sofosbuvir observational cohort study for the treatment of recurrent hepatitis C: clinical and virological outcomes. Transpl Int. 2017;30:1253–1265. doi:10.1111/tri.13018.
28. Liu A, Angirekula M, Elhawary A, Mara K, Kulai T, Leise M, Watt KD. Progression of fibrosis in liver transplant recipients with recurrent hepatitis C treated with direct-acting antivirals. Clin Transplant. 2023;37(8):e15050. doi: 10.1111/ctr.15050. Epub 2023 Jun 17. PMID: 37329298.
29. Dumortier J, Boillot O, Scoazec JY. Natural history, treatment and prevention of hepatitis C recurrence after liver transplantation. World J Gastroenterol. 2014;20(32):11069–11081. doi: 10.3748/wjg.v20.i32.11069. PMID: 25170196; PMCID: PMC4145750.

**Table 1: Patient demographic and clinical data.**

| | | n | % |
|---|---|---|---|
| GENDER | FEMALE | 59 | 60.2 |
| | MALE | 39 | 39.8 |
| DONOR GENDER | FEMALE | 34 | 38.2 |

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| | | | |
|---|---|---|---|
| | MALE | 55 | 61.8 |
| HCV GENOTYPE | 1A | 3 | 5.9 |
| | 1B | 41 | 80.4 |
| | 2A | 1 | 2.0 |
| | 3A | 4 | 7.8 |
| | 4D | 1 | 2.0 |
| | 5A | 1 | 2.0 |
| TRANSPLANTATION TYPE | LIVING DONOR | 89 | 90.8 |
| | CADAVERIC | 9 | 9.2 |
| HCV TREATMENT BEFORE TX | NO TREATMENT | 78 | 85.7 |
| | HARVONI (LEDİPASVİR-SOFOSBUVİR) | 5 | 5.5 |
| | EXVIERA (DASABUVIR) | 2 | 2.2 |
| | SOVALDI (SOFOSBUVIR | 1 | 1.1 |
| | INTERLOKIN ,RIBAVIRIN | 4 | 4.4 |
| | PEGASYS (PEGİNTERFERON ALFA-2A) | 1 | 1.1 |
| HCV TREATMENT AFTER TX | NO TREATMENT | 48 | 48.9 |
| | COPEGUS,PEGASYS( RIBAVIRIN) | 3 | 3.06 |
| | MAVIRET(100 MG GLEKAPREVİR - 40 MG PİBRENTASVİR ) | 7 | 7.1 |
| | HARVONI(LEDİPASVİR - SOFOSBUVİR) | 32 | 32.6 |
| | RIBAVIRIN+SOVOSFOBIR | 5 | 5.1 |

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| | | | |
|---|---|---|---|
| | RIBAVIRIN,INTERFERON | 3 | 3.06 |
| HCC BEFORE TX | YES | 30 | 30.6 |
| | NO | 68 | 69.4 |
| HCC AFTER TX | YES | 0 | 0.0 |
| | NO | 98 | 100.0 |
| HCV RNA BEFORE TX | NEGATIVE | 35 | 48.6 |
| | POSITIVE | 37 | 51.4 |
| HCV RNA AFTER TX | NEGATIVE | 34 | 60.7 |
| | POSITIVE | 22 | 39.3 |
| HCV RECCURENCE | YES | 49 | 50.0 |
| | NO | 49 | 50.0 |
| ADDITIONAL DISEASES | NO DISEASES | 51 | 52.0 |
| | HYPOTHYROIDISM | 9 | 9.2 |
| | DIABETES MELLITUS | 9 | 9.2 |
| | HYPERTENSION | 11 | 11.2 |
| | HEPATITIS B VIRUS | 6 | 6.1 |
| | OTHERS | 12 | 12.2 |
| SVR V/Y | YES | 56 | 90.3 |
| | NO | 6 | 9.7 |
| SVR 3AY | YES | 52 | 83.9 |
| | NO | 10 | 16.1 |
| SVR 6AY | YES | 56 | 90.3 |
| | NO | 6 | 9.7 |
| RE-TRANSPLANTATİON | YES | 2 | 2.0 |
| | NO | 96 | 98.0 |
| CIRRHOSIS OR GRAFT FAILURE | YES | 11 | 11.2 |
| | NO | 87 | 88.8 |

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| | | | |
|---|---|---|---|
| FIBROSING CHOLESTATIC HEPATITIS | YES | 11 | 11.2 |
| | NO | 87 | 88.8 |
| CMV | NEGATIVE | 95 | 99.0 |
| | POSITIVE | 1 | 1.0 |
| IMMUNSUPRESIVE TREATMENT | NO | 1 | 1.0 |
| | PROGRAF CELLCEPT | 32 | 32.7 |
| | PROGRAF(TAKROLİMUS) | 49 | 50.0 |
| | CERTAIN(EVORILIMUS) | 6 | 6.1 |
| | CELLCEPT(MİKOFENOLİK ASİT) | 7 | 7.1 |
| | SANDIMUN(SİKLOSPORİN) | 3 | 3.1 |
| RITUKSİMAB | YES | 0 | 0.0 |
| | NO | 98 | 100.0 |
| REJECTION | YES | 6 | 6.1 |
| | NO | 92 | 93.9 |
| REJ-PULSE STEROİD | YES | 4 | 4.1 |
| | NO | 94 | 95.9 |

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**Table 2: The overall survival and disease free survival rates at 1,3, 5 and 10 years.**

| | | Overall survival (n=98) | Disease free survival (n=98) | Survival of patients with recurrence (n=49) |
|---|---|---|---|---|
| Survival (mo.) Mean±SE | | 145.01±6.79 | 70.46±7.44 | 162.44±7.50 |
| Cumulative survival rate | 1 year | 95.7% | 78.7% | 98.0% |
| | 3 years | 88.3% | 54.3% | 91.8% |
| | 5 years | 83.5% | 39.6% | 91.8% |

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| | | | |
|---|---|---|---|
| 10 years | 75.0% | NA | 88.7% |

**Table 3:Cox regression survival analysis (All patients)**

| | | Univariate Cox regression | |
|---|---|---|---|
| | | HR (95% CI) | HR p-value |
| Pre-TX HCV rna | Negative (n=35) | reference | |
| | Positive (n=36) | 0.998 (0.361-2.761) | 0.996 |
| RECİPİENT DM | Yes (n=15) | Reference | |
| | No (n=75) | 3.267 (0.434-24.569) | 0.250 |
| ADDITIONAL DISEASES | No (n=48) | Reference | |
| | Yes (n=46) | 1.067 (0.444-2.565) | 0.884 |
| Pre-TX HCC | No (n=68) | Reference | |
| | Yes (n=30) | 1.515 (0.601-3.818) | 0.378 |
| Pre-TX HCV treatment | Yes (n=13) | Reference | |
| | No (n=74) | 1.133 (0.257-4.987) | 0.869 |
| Pre-TX MELD | (n=98) | 0.937 (0.846-1.039) | 0.218 |
| Pre-TX ALT | (n=98) | 0.999 (0.995-1.003) | 0.693 |
| Post-TX ALT | (n=98) | 1.001 (1.000-1.003) | 0.061 |

**Table 4: Logistic regression analysis for reccurence.**

| | | Univariate Logistic regression | |
|---|---|---|---|
| | | OR (95% CI) | OR p-value |
| Pre-TX HCV RNA | Negative (n=35) | Reference | |

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| | | | |
|---|---|---|---|
| | Positive (n=37) | 12.444 (4.068-38.065) | **<0.001** |
| RECIPIENT DM | Yes (n=19) | Reference | |
| | No (n=79) | 1.233 (0.408-3.728) | 0.710 |
| ADDITIONAL DISEASES | No (n=51) | reference | |
| | Yes (n=47) | 0.475 (0.212-1.064) | 0.070 |
| Pre-TX HCC | No (n=68) | reference | |
| | Yes (n=30) | 1.212 (0.513-2.866) | 0.661 |
| Pre-TX HCV treatment | Yes (n=13) | reference | |
| | No (n=78) | 16.364 (2.026-132.148) | **0.009** |
| Pre-TX MELD | (n=98) | 0.976 (0.911-1.046) | 0.500 |
| Pre-TX ALT | (n=98) | 1.000 (0.997-1.003) | 0.951 |
| Post-TX ALT | (n=98) | 0.998 (0.995-1.000) | 0.097 |

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**Table 5:Cox regression survival analysis (recurrence patients)**

| | | Univariate Cox regression | |
|---|---|---|---|
| | | HR (95% CI) | HR p-value |
| Pre-TX HCV rna | Negative (n=7) | reference | |
| | Positive (n=28) | 27.866 (0.000-7477973) | 0.602 |
| RECIPIENT DM | Yes (n=7) | reference | |
| | No (n=41) | 25.749 (0.001-1139815) | 0.552 |
| ADDITIONAL DISEASES | No (n=30) | reference | |
| | Yes (n=19) | 0.348 (0.039-3.128) | 0.346 |
| Pre-TX HCC | No (n=33) | reference | |
| | Yes (n=16) | 1.577 (0.261-9.543) | 0.620 |
| Pre-TX HCV | Yes (n=1) | reference | |

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| | | | |
|---|---|---|---|
| treatment | No (n=45) | 0.104 (0.012-0.943) | **0.044** |
| Pre-TX MELD | (n=49) | 0.927 (0.748-1.147) | 0.484 |
| Pre-TX ALT | (n=49) | 1.000 (0.994-1.006) | 0.924 |
| Post-TX ALT | (n=49) | 0.999 (0.991-1.008) | 0.862 |

**Table 6:SVR rates in the recurrent patients.**

| | | copegus, pegasys | mavıret | harvoni | rıbavirin+ sovosfobir | pegasy, ribavirin, interferon | p-value |
|---|---|---|---|---|---|---|---|
| SVR | Yes | 2 (66.7) | 7 (100) | 31 (96.9) | 4 (80.0)[a,b] | 0 (0)[a] | 0.066 |
| | No | 1 (33.3) | 0 (0) | 1 (3.1) | 1 (20.0)[a,b] | 2 (100)[a] | |
| SVR 3 mo | Yes | 2 (66.7)[a,b] | 7 (100)[b] | 28 (87.5)[b] | 3 (60.0)[a,b] | 0 (0)[a] | 0.015 |
| | No | 1 (33.3)[a,b] | 0 (0)[b] | 4 (12.5)[b] | 2 (40.0)[a,b] | 2 (100)[a] | |
| SVR 6 mo | Yes | 2 (66.7)[a,b] | 7 (100)[b] | 31 (96.9)[b] | 4 (80.0)[a,b] | 0 (0)[a] | 0.002 |
| | No | 1 (33.3)[a,b] | 0 (0)[b] | 1 (3.1)[b] | 1 (20.0)[a,b] | 2 (100)[a] | |
| | Total | 3 (6.1) | 7 (14.3) | 32 (65.3) | 5 (10.2) | 2 (4.1) | 49 (100) |

*Superscript letters indicate statistical significance between column proportions

** Data are presented as n (%)