Safety and Efficacy of Direct Antiviral Agents for Hepatitis C in Patients with Malignancies Other Than Liver Cancer: A Case Series

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Abstract: (1) Background: direct-acting antivirals (DAA) are the current standard of care for chronic hepatitis C. Oncologic patients remain among the most difficult-to-treat subgroups of hepatitis C virus (HCV)-infected patients due to their clinical frailty and complex therapeutic protocols received. (2) Methods: we retrospectively collected and analysed clinical data of 30 consecutive patients treated with DAA, between 2015 and 2022, for chronic HCV infection in the context of oncologic disease. (3) Results: most patients were females (63.3%), median age was 67 years, HCV genotype 1 was prevalent (60%), and median HCV RNA levels were 2.2 \times 10^6 IU/mL. The most common malignancy was breast cancer (37%), and the chief oncologic drugs co-administered with DAAs were tamoxifen, platinum derivatives, cyclophosphamide, paclitaxel, rituximab and doxorubicin. Overall, 50% of patients had chronic hepatitis. A total of 76.7% underwent a sofosbuvir-based treatment. Sustained virological response 12 weeks after the end of therapy (SVR12) was reached in all patients. After SVR12, two patients died. DAA treatment was well tolerated; no patients had to stop DAA treatment or showed any adverse event or drug-drug interaction specifically attributable to DAAs. (4) Conclusions: DAA treatment should be promptly offered to oncologic patients with chronic hepatitis C in order to achieve aminotransferase normalization and viremia control, making antineoplastic therapy feasible and safe.

Keywords: HCV; DAA; cancer; chemotherapy; radiotherapy; cirrhosis

1. Introduction

Hepatitis C virus (HCV) infection is still prevalent in the general population and is often detected by chance in patients unaware of their infection [1,2]. This often occurs in the context of the diagnostic workup for other diseases, including malignancies [3,4].

Direct-acting antiviral (DAA) agents are the current standard of care for chronic hepatitis C and are safe and effective in more than 95% of treated patients [1,5–7]. Drug–drug interactions represent a common issue requiring particular attention during treatment [8–10]. DAAs have been largely employed in the oncology setting and were shown to be safe and effective mostly in patients with hepatocellular carcinoma and lymphomas [10–14]. Indeed, chronic hepatitis C can complicate cancer treatment in patients with solid or hematological malignancies that are not related to HCV infection. High viral load, often a sign of immunosuppression, and/or high alanine aminotransferase (ALT) levels, possibly due to
liver toxicity, can preclude the completion of full antineoplastic protocols. Specifically, HCV infection often causes serious liver disease with advanced inflammation and/or severe fibrosis [15–18].

Accordingly, oncologic patients remain among the most difficult-to-treat subgroups of HCV-infected patients due to their clinical frailty and the complex therapeutic protocols received. Further experience is sorely needed in this setting.

We retrospectively collected and analysed clinical data of patients treated for chronic HCV infection in the context of oncologic disease. Our aim was to assess the safety and efficacy of DAA therapy and patients’ tolerability in this special subgroup and in the presence or absence of antineoplastic therapy.

2. Patients and Methods
2.1. Patients
Data of consecutive 30 patients with chronic hepatitis C and a malignancy, followed at the Unit of Infectious or Transplant Medicine or the Unit of Infectious Diseases, University of Campania “L. Vanvitelli”, between 2015 and January 2022, were included in this retrospective analysis. Patients made up a relatively heterogeneous group as they were referred by oncologists of different hospitals in our region and in different moments of their oncologic history and treatment.

Upon the first observation, patients underwent a complete physical exam, full liver function tests, blood cell count, assessment hepatitis B virus (HBV) and human immunodeficiency virus (HIV) markers, quantitative HCV-RNA, HCV genotype and liver ultrasound scan. For each patient, the body mass index (BMI: kg/m\(^2\)) was calculated.

Liver fibrosis was evaluated by transient elastography (TE, FibroScan, EchoSens, Paris, France), while clinical cirrhosis was diagnosed in patients with a clear clinical presentation (i.e., splenomegaly, esophageal varices, ascites).

2.2. Antiviral Treatment
Patients were treated according to the ongoing modifications of EASL guidelines and DAA availability in Italy [1,19–21].

A careful drug history was taken, and a consonant adjustment of treatments was carried out, when needed and possible, according to the European Association for the Study of the Liver (EASL) guidelines and HEP Drug Interactions, University of Liverpool website (http://hepdruginteractions.org).

Laboratory tests were performed by hospital standard procedures. Patients were followed up once a month for the duration of DAA treatment and at least 3 months after the end of treatment.

Response to antiviral treatment was defined as the sustained virological response (SVR = HCV RNA undetectable) 12 weeks (SVR12) after the end of therapy. Relapse was defined as the reappearance of serum HCV RNA after the end of DAA treatment.

Personal and clinical data were managed in agreement with the Declaration of Helsinki and the General Data Protection Regulation (679/2016) and were approved by our Ethical Committee of Università della Campania L. Vanvitelli (protocol code 21399/2021). Informed consent was obtained from all subjects involved in the study.

Data analyses were largely descriptive and included the presentation of categorical data as number and percent and numerical data as median and range.

3. Results
3.1. Baseline
The general clinical features of each patient included in the study are reported in Table 1. Most patients were females (63.3%), had a median age of 67 years (range 44–87) and a median BMI of 25.5 (range 21–34). Eighty percent of patients presented with solid cancer, while 20% were affected by a hematological malignancy. The most common malignancy was breast cancer (11 cases, 37%), and the chief oncologic drugs co-administered with
Table 1. General characteristics of the study group.

| Patient | Age | Sex | CCI | Malignancy | Oncologic Treatment during DAA | Radiotherapy during DAA | HCV-RNA Baseline | HCV Genotype | ALT Baseline | Fibrosis* | Antiviral Treatment | Oncologic Treatment | Virologic Outcome |
|---------|-----|-----|-----|------------|--------------------------------|-------------------------|------------------|-------------|--------------|-----------|---------------------|-------------------|-------------------|
| 1       | 71  | M   | 6   | Prostate cancer | LEU | No | 16700000 | 2 | 10 | F1 | SOF/VEL | SD | SVR 12 |
| 2       | 81  | M   | 8   | CLL | No | No | 1065210 | 2 | 24 | F3 | SOF/VEL | SD | SVR 12 |
| 3       | 46  | M   | 7   | Lung cancer | No | Yes | 142000 | 2 | 98 | F3 | SOF/VEL | PD | SVR 12 |
| 4       | 49  | M   | 5   | Lung cancer | CAR-PAC-PF | No | 880000 | 2 | 103 | F3 | SOF/VEL | SD | SVR 12 |
| 5       | 74  | M   | 8   | Prostate cancer | No | Yes | 1200000 | 3 | 118 | F4 | GLE/PB | SD | SVR 12 |
| 6       | 78  | F   | 10  | Colorectal cancer | OXA-CAP | No | 1000000 | 2 | 200 | F4 | SOF/VEL | PD | SVR 12 |
| 7       | 78  | F   | 11  | Breast cancer | PAC, TRA, PER | Yes | 4500000 | 1b | 183 | F4 | SOF/VEL | PD | SVR 12 |
| 8       | 63  | F   | 6   | Breast cancer | CYC-EPI | Yes | 800000 | 1a | 71 | F3 | SOF/VEL | SD | SVR 12 |
| 9       | 59  | M   | 8   | Metastatic melanoma | NIV | No | 3000000 | 1a | 65 | F2 | GLE/PB | SD | SVR 12 |
| 10      | 65  | F   | 7   | Breast cancer | EPI, CYC, PAC, TRA | No | 6750000 | 2 | 104 | Cl. Cirr | SOF/VEL | SD | SVR 12 |
| 11      | 77  | F   | 10  | Breast cancer | CYC | Yes | 7200000 | 2 | 12 | FO-FI | EBL/PIB | SD | SVR 12 |
| 12      | 65  | F   | 6   | Colorectal cancer | OXA-CAP | Yes | 1600000 | 1 | 103 | F3 | 3D | SD | SVR 12 |
| 13      | 63  | M   | 5   | Non-Hodgkin Lymphoma | R-CHOP | Yes | 1640000 | 1 | 48 | FO-FI | SOF/LDV | SD | SVR 12 |
| 14      | 64  | F   | 8   | Breast cancer | TAM | Yes | 1200000 | 1 | 23 | FO-FI | EBL/CR | SD | SVR 12 |
| 15      | 55  | F   | 6   | Non-Hodgkin Lymphoma | HSCT | No | 9360000 | 1b | 35 | Cl. Cirr | SOF/VEL | SD | SVR 12 |
| 16      | 79  | M   | 8   | Multiple myeloma | BOR | No | 460000 | 1b | 42 | F4 | SOF/LDV | PD | SVR 12 |
| 17      | 87  | M   | 9   | Badder cancer | No | Yes | 302000 | 2a | 144 | Cl. Cirr | SOF/VEL | SD | SVR 12 |
| 18      | 57  | F   | 4   | Breast cancer | TAM | No | 9620000 | 2a | 40 | F3 | SOF/RELV | PD | SVR 12 |
| 19      | 63  | F   | 5   | Breast cancer | CYC | Yes | 450000 | 2a/2c | 22 | F2 | SOF/VEL | PD | SVR 12 |
| 20      | 78  | F   | 8   | Breast cancer | TAM | Yes | 1000000 | 1b | 47 | Cl. Cirr | SOF/LDV | SD | SVR 12 |
| 21      | 77  | F   | 8   | Breast cancer | TAM | Yes | 3000 | 1b | 37 | Cl. Cirr | SD + RBV | PD | SVR 12 |
| 22      | 69  | F   | 7   | Colorectal cancer | OXA-CAP | No | 245000 | 1b | 96 | Cl. Cirr | SOF/LDV | SD | SVR 12 |
| 23      | 68  | F   | 4   | Lymphoma | R-CHOP | No | 230000 | 4 | 21 | FO-FI | SOF/LDV | SD | SVR 12 |
| 24      | 70  | M   | 7   | Prostate cancer | LEU | Yes | 512000 | 1b | 67 | Cl. Cirr | SD + RBV | SD | SVR 12 |
| 25      | 46  | F   | 4   | Lymphoma | R-CHOP | No | 520000 | 2a | 32 | FO-FI | SOF/VEL | SD | SVR 12 |
| 26      | 80  | M   | 9   | Lung cancer | CS-ETO | No | 420000 | 1b | 104 | Cl. Cirr | SOF+DAA-RELV | PD | SVR 12 |
| 27      | 71  | F   | 8   | Breast cancer | TAM | No | 2100000 | 1b | 56 | Cl. Cirr | SOF/LDV | PD | SVR 12 |
| 28      | 61  | F   | 10  | Uterine cancer | No | Yes | 230000 | 1b | 65 | Cl. Cirr | SOF/LDV | PD | SVR 12 |
| 29      | 60  | F   | 7   | Gastric cancer | DMA | No | 1200000 | 1b | 56 | F4 | SOF/LDV | SD | SVR 12 |
| 30      | 44  | F   | 2   | Breast cancer | TAM | No | 1200000 | 1b | 30 | F1 | SOF/VEL | SD | SVR 12 |

3D, ombitasvir + paritaprevir + ritonavir + dasabuvir; ALT, alanine aminotransferase; BOR, borotemizib; CAP, capcitabine; CAR, carboplatin; CCI, Charlson Comorbidity Index; CIS, cisplatin; Cl. Cirr, clinical cirrhosis; CLL, chronic lymphocytic leukemia; CYC, cyclophosphamide; DAA, direct antiviral agents; EBL, ebivir; EPL, epoxidoxorubicin; ETO, etoposide; GLE, glecaprevir; GR, grazoprevir; HCV, hepatitis C virus; HSCT, hematopoietic stem cell transplantation; IMA, imatinib; LDV, ledipasvir; LEU, leuprolide acetate; NIV, nivolumab; OXA, oxaliplatin; PAC, paclitaxel; PD, progressive disease; PEM, pembrolizumab; PER, pertuzumab; PIB, pibrentasvir; RBV, ribavirina; R-CHOP, rituximab+ cyclophosphamide + doxorubicin + vincristine + prednisone; SD, stable disease; SIM, simprevir; SOF, sofosbuvir; SVR, sustained virological response; TAM, tamoxifen; TRA, trastuzumab; VEL, velipastavir. * Fibrosis by fibroscan or clinical cirrhosis. ˆ Oncologic outcome was evaluated 12 weeks after the end of DAA treatment.

The median estimated glomerular filtration rate (by Modification of Diet in Renal Disease, MDRD study equation) was 83 mL/min × 1.73 m² (range 46–190). Many comorbidities were also present, with a median Charlson Comorbidity Index (CCI) of 7 (range 2–11).

In terms of liver disease stage, 50% of patients had chronic hepatitis, and 50% had cirrhosis. Only one patient had an HBV co-infection already on specific antiviral treatment. HCV genotype 1 was prevalent (60%) and median HCV RNA levels were 2.2 × 10⁶ IU/mL (range 3.9 × 10⁶–96.1 × 10⁶).

At baseline, 53.3% of patients showed increased ALT levels (median 57 U/L, range 10–200 U/L). In addition, 90% of patients were naïve to antiviral treatment, and 10% were previously non-responder to peg-interferon plus ribavirin.

Regarding DAA treatment, 76.7% underwent a sofosbuvir-based treatment, and 23.3% underwent a non-sofosbuvir-based treatment; in only 4 patients, ribavirin was added to the treatment regimen. During DAA treatment, 80% of patients were exposed to chemotherapy, and as many as 33.3% were exposed to radiotherapy.

3.2. Treatment Results

ALT levels became normal in 93.3% of patients (median 21.5 U/L range 10–93.1 U/L) after 1 month of DAA treatment and in 97% of patients after 2 months of DAA treatment.

All patients obtained a documented SVR12. A total of 19 (63%) patients were followed up for a longer time and were assessed at 24 weeks after DAA treatment; of these, 1 patient relapsed, whilst 18 maintained the SVR. Two patients (7%) died, and seven patients (23%)
were lost to follow-up after reaching SVR12; furthermore, two patients (7%) had not yet reached 24 weeks after the end of therapy. Patients died for reasons unrelated to DAA treatment but for a rapid worsening of oncologic disease.

No HBV reactivation was observed in the HBV-HCV coinfected patient, who continued his HBV-directed antivirals.

DAA treatment was well tolerated; no patients had to stop DAA treatment or showed any adverse event specifically attributable to DAA. In particular, no specific adverse event could be identified when DAA was added to ongoing oncologic therapy. Only patients treated with ribavirin ($n = 4$) developed mild anemia, with a median hemoglobin 1 month after starting treatment of 12.2 g/dL. Furthermore, no drug-drug interactions were observed. There were no differences in safety and efficacy between the different antiviral regimens.

As regards the oncologic outcome, among patients with ALT above the UNL at baseline, 11 (68.8%) had stable disease, 3 (18.7%) had progressive disease, and 2 (12.5%) died. Among patients with normal ALT values at baseline, 12 (85.7%) had stable disease, and 2 (14.3%) had progressive disease.

4. Discussion

Chronic hepatitis C remains highly prevalent in specific geographic areas of the world, including the Campania region of southern Italy [1,22].

Patients with malignancy and HCV infection represent a special population with a greater risk of oncologic treatment delay, dose modification and longer treatment duration as previously reported in a specific cancer population [23]. At the moment, no specific treatment indications are provided in the current EASL guidelines [1]. In real life, hepatologists often face patients who become aware of HCV infection during the diagnostic workup for malignancy, but few data are available regarding the outcomes of antiviral treatment in this subgroup.

DAA treatment is generally safe, effective and well tolerated, and only a limited life expectancy for non-liver-related co-morbidities is now considered a contraindication to therapy [1]. In oncologic patients, this issue is very important, as short life expectancy is possible in advanced disease. Thus, a careful prognostic stratification of the malignancy is necessary to appropriately consider DAA treatment as already reported in patients with hematological cancers [24].

In many cases, HCV-related elevation of liver enzymes contraindicates or complicates cancer chemotherapy [25]; antiviral treatment, with consequent fast ALT normalization, allows for higher dose regimens and access to investigational cancer therapies, possibly increasing the chances of an oncologic cure [26,27].

DAA treatment was safe and effective in our 30 oncologic patients. They were referred to us during a period of 6 years and were followed up and treated with different protocols according to the ongoing modifications of EASL guidelines and DAA availability in Italy. However, all treatments and protocols were well tolerated and largely achieved a sustained virological response, allowing for oncologic treatment to be carried out without specific modifications. A careful evaluation of drug-drug interactions before starting antiviral therapy allowed us to proceed with oncologic treatment without adverse events or the need to stop treatment. Previous experience also showed good tolerability of DAA treatment during chemotherapy [10,26].

Half of our patients were treated with newer DAA schedules (sofosbuvir/velpatasvir and pribentasvir/glecaprevir) with a good antiviral response and no adverse drug reactions. These regimens are simpler, shorter and with fewer drug-drug interactions than previous ones, encouraging antiviral treatment start.

Despite the low number of patients and the not homogeneous follow-up, our findings suggest oncologists should not be concerned when treating patients with HCV infection. However, a longer follow-up could be useful in this category of patients to highlight possible late viral reactivations or clinical flares related to the immunosuppressive state.
In conclusion, in the absence of specific contraindications or limited life expectancy, DAA treatment should be promptly offered to oncologic patients with chronic hepatitis C in order to achieve ALT normalization and viremia control, making antineoplastic therapy feasible and safe.

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Informed Consent Statement: Informed consent was obtained from all subjects involved in the study.

Data Availability Statement: The data presented in this study are available on request from the corresponding author. The data are not publicly available due to patient privacy protection.

Conflicts of Interest: The authors declare no conflict of interest.

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