A real-world experience of SARS-CoV-2 infection in a tertiary referral centre of Montréal: Unexpected low prevalence and low mortality

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ABSTRACT

BACKGROUND: The impact of severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection in patients with chronic liver disease (CLD) and liver transplant (LT) recipients remains a concern. The aim of this study was to report the impact of coronavirus disease 2019 (COVID-19) infection among patients at the tertiary health care centre Centre hospitalier de l’Université de Montréal (CHUM) during the first wave of the SARS-CoV-2 pandemic. METHODS: This real-world, retrospective cohort included all patients admitted to our liver unit and/or seen as an outpatient with CLD with or without cirrhosis and/or LT recipients who tested positive to SARS-CoV-2 infection. Cases were considered positive as defined by the detection of SARS-CoV-2 by reverse-transcription polymerase chain reaction (RT-PCR) on nasopharyngeal swabs. RESULTS: Between April 1 and July 31, 2020, 5,637 were admitted to our liver unit and/or seen as outpatient. Among them, 42 were positive for SARS-CoV-2. Twenty-two patients had CLD without cirrhosis while 16 patients had cirrhosis at the time of the infection (13, 2, and 1 with Child–Pugh A, B, and C scores, respectively). Four were LT recipients. Overall, 15 of 42 patients (35.7%) were hospitalized; among them, 7 of 42 (16.7%) required respiratory support and 4 of 42 (9.5%) were transferred to the intensive care unit. Only 4 of 42 (9.5%) patients died: 2 with CLD without cirrhosis and 2 with CLD with cirrhosis. Overall survival was 90.5%. CONCLUSION: This real-world study demonstrates an unexpectedly low prevalence and low mortality in the context of SARS-CoV-2 infection among patients with CLD with or without cirrhosis and LT recipients.

KEYWORDS: chronic liver disease; cirrhosis; COVID-19; liver transplantation; SARS-CoV-2

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INTRODUCTION

Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) is responsible for the coronavirus disease 2019 (COVID-19) (1). The impact of this infection in special populations, such as patients with chronic liver disease (CLD) and liver transplant (LT) recipients, is still a matter of discussion (2). International studies are limited by a selection bias because they only included patients requiring hospitalization at the time of infection, thus reporting the most severe cases (3–5). In addition, multicentric or monocentric studies showed that geographical and management differences affected patient outcome (6–7). More studies are needed, especially real-world experience, to better understand the prognostic factors and thus better modulate public health policies.

As of July 2021, more than 1,400,000 cases of COVID-19 infection had been confirmed in Canada (https://coronavirus.jhu.edu/about/how-to-use-our-data; Johns Hopkins Center for Systems Science and Engineering [CSSE]; up to date July 14, 2021). The province of Québec was the most affected, with almost 380,000 cases (https://www.inspq.qc.ca/covid-19; up to date July 14, 2021). In Québec, Montréal was the epicentre of the pandemic. Here, we report the results from almost the entire cohort of patients being followed as outpatients or inpatients in the largest liver unit in Montréal. The objective of this real-world report is to describe the characteristics of all patients with CLD with or without cirrhosis as well as LT recipients during the first wave of the COVID-19 pandemic.

PATIENTS AND METHODS

Study design

This retrospective cohort study included all cases with laboratory-confirmed SARS-CoV-2 infection between April 1 and July 31, 2020 in patients admitted and/or seen as outpatients in our liver unit at the Centre hospitalier de l’Université de Montréal (CHUM). Inclusion criteria were patients aged ≥18 years old with any COVID-19 symptom profile or disease severity, with CLD with or without cirrhosis, and LT recipients. Positive cases were defined as detection of SARS-CoV-2 by reverse-transcription polymerase chain reaction (RT-PCR) on nasopharyngeal swabs. All positive cases in all epidemiological contexts (screening, contact cases, etc.) were included. Cases were excluded if any of the following conditions were met: SARS-CoV-2 infection was not laboratory-confirmed, or hospitalization status or mortality outcome was not known. The current study included 11 patients reported in a previously published registry (4–5).

Variables and definitions

Demographic, clinical and laboratory data were collected from physical and electronic records, de-personalized data were extracted from Oacis (BDM IT Solutions, Saskatoon, Saskatchewan, Canada). Diagnosis of cirrhosis was confirmed by documentation of fibrosis by computed tomography scan, magnetic resonance, transient elastography, or liver biopsy. Diagnosis of cirrhosis was ascertained in some patients by detailed chart review for clinical, radiologic, or biochemical evidence of liver cirrhosis. Alcohol use was defined as no drinking, social drinking (up to 2 drinks/d for men and to 1 drink/d for women), or current daily drinking (consuming more than social drinking limits on a daily basis). Overweight and obesity were defined as a given body mass index (BMI) of ≥25 or ≥30 kg/m², respectively. When data on BMI was unavailable and obesity was not mentioned in medical records, obesity was assumed to be absent. Data on decompensation or hepatocellular carcinoma (HCC) were collected from chart review. The presence and severity of ascites, encephalopathy, variceal bleeding, and other major decompensating events at baseline and during COVID-19 were collected. If patients developed worsening of ascites, hepatic encephalopathy, or variceal bleeding during COVID-19, they were deemed to have decompensated during COVID-19.

Statistical methods

The results were analyzed using the SPSS v.25.0 statistical software (IBM Corp., Armonk, NY, USA).

Data were presented in the form of mean ± standard deviation or percentage.

Ethical and regulatory approval

As the data collected contained no personal health identifiers, it was deemed not to require
written informed consent by our institutional ethics committee.

**RESULTS**

**Prevalence**
Between April 1 and July 31, 2020, 5,637 patients were admitted and/or seen as outpatients (telephone and in-person visits) in our liver unit at the CHUM (Figure 1). Overall, 42 patients were detected with a SARS-CoV-2 infection (prevalence of 0.7%). Of the 42 patients, 22 were diagnosed with CLD without cirrhosis, 16 with CLD and cirrhosis, and 4 had undergone LT.

CLD = Chronic liver disease; LT = Liver transplant; SARS-CoV-2 = Severe acute respiratory syndrome coronavirus 2; CP = Child–Pugh

**Demographic and clinical characteristics of patients**
Table 1 shows the demographic and clinical characteristics of patients with CLD without cirrhosis, 16/42 (38.1%) had CLD with cirrhosis, and 4/42 (9.5%) were LT recipients. Overall, 5,380 individuals were seen as outpatients, of whom 26 tested positive for SARS-CoV-2 (prevalence = 0.5%). 210 patients were admitted to the hospital for a total of 257 episodes; of those 210 patients, 16 tested positive (prevalence = 7.6%).

*Figure 1: Cohort selection*

*Between April 1 and July 31, 2020, 5,637 patients consulted and/or were admitted to the liver unit. Among them, 42 patients were diagnosed with a SARS-CoV-2 infection (prevalence of 0.7%). Of the 42 patients, 22 were diagnosed with CLD without cirrhosis, 16 with CLD and cirrhosis, and 4 had undergone LT.

CLD = Chronic liver disease; LT = Liver transplant; SARS-CoV-2 = Severe acute respiratory syndrome coronavirus 2; CP = Child–Pugh*
**Table 1:** Demographic and clinical characteristics of the 42 patients with confirmed SARS-CoV-2 infection

| Characteristics                                      | CLD without cirrhosis (n = 22) | CLD with cirrhosis (n = 16) | LT recipients (n = 4) |
|------------------------------------------------------|---------------------------------|------------------------------|-----------------------|
| Age, y, median (IQR), mean                           | 50 (26–82), 51                  | 60 (40–78), 60               | 55 (30–68), 52        |
| Sex (male), no. (%)                                  | 15 (68)                         | 12 (75)                      | 2 (50)                |
| Ethnicity, no. (%)                                   |                                 |                              |                       |
| White                                                | 9 (40)                          | 13 (81)                      | 4 (100)               |
| Black                                                | 9 (40)                          | 1 (6)                        | 0 (0)                 |
| Asian                                                | 2 (9)                           | 1 (6)                        | 0 (0)                 |
| Arabic                                               | 1 (5)                           | 1 (6)                        | 0 (0)                 |
| Hispanic                                             | 1 (5)                           | 0 (0)                        | 0 (0)                 |
| Etiology of liver disease, no. (%)                   |                                 |                              |                       |
| HBV                                                  | 8 (36)                          | 3 (19)                       | 0 (0)                 |
| NAFLD                                                 | 7 (32)                          | 3 (19)                       | 1 (25)                |
| HCV                                                   | 4 (18)                          | 5 (31)                       | 1 (25)                |
| ALD                                                   | 2 (9)                           | 4 (25)                       | 0 (0)                 |
| Other aetiologies*                                   | 1 (5)                           | 1 (6)                        | 2 (50)                |
| Comorbidities, no. (%)                               |                                 |                              |                       |
| Obesity                                               | 15 (68)                         | 11 (69)                      | 2 (50)                |
| Arterial hypertension                                | 6 (27)                          | 6 (38)                       | 2 (50)                |
| Diabetes mellitus                                     | 2 (9)                           | 6 (38)                       | 1 (25)                |
| Cardiovascular disease                                | 3 (14)                          | 2 (13)                       | 1 (25)                |
| Chronic kidney disease                                | 3 (14)                          | 2 (13)                       | 1 (25)                |
| Chronic obstructive pulmonary disease                 | 1 (5)                           | 5 (31)                       | 0 (0)                 |
| History of stroke                                     | 1 (5)                           | 0 (0)                        | 0 (0)                 |
| Numbers of comorbidities, no. (%)                    |                                 |                              |                       |
| None                                                  | 13 (59)                         | 3 (19)                       | 1 (25)                |
| One                                                   | 3 (14)                          | 4 (25)                       | 1 (25)                |
| Two                                                   | 2 (9)                           | 5 (31)                       | 2 (50)                |
| 3 or more                                             | 4 (18)                          | 4 (25)                       | 0 (0)                 |
| Liver disease severity, no. (%)                      |                                 | 13 (81)                      |                       |
| Child–Pugh A                                         |                                 |                              |                       |
| Child–Pugh B                                         | 2 (13)                          |                              |                       |
| Child–Pugh C                                         | 1 (6)                           |                              |                       |
| Immunosuppressors regimen, no. (%)                   |                                 |                              |                       |
| Monotherapy                                          |                                 | 1 (25)                       |                       |
| Tacrolimus                                            |                                 | 1 (25)                       |                       |
| Tritherapy                                            |                                 | 3 (75)                       |                       |
| Cyclosporin A + prednisone + MMF                     |                                 | 1 (25)                       |                       |
| Tacrolimus + prednisone + azathioprine               |                                 | 1 (25)                       |                       |
| Tacrolimus + prednisone + MMF                        |                                 | 1 (25)                       |                       |

*Other aetiologies: Primary sclerosing cholangitis (n = 1); Cystic fibrosis (n = 2); Autoimmune hepatitis (n = 1)
*SARS-CoV-2 = Severe acute respiratory syndrome coronavirus 2; CLD = Chronic liver disease; LT = Liver transplant; HBV = Hepatitis B virus; NAFLD = Non-alcoholic fatty liver disease; HCV = Hepatitis C virus; ALD = Alcoholic liver disease; MMF = Mycophenolate mofetil
those with CLD and cirrhosis, and LT recipients, respectively. The mean age at the time of COVID-19 diagnosis was 50 years, 60 years, and 55 years, respectively. The three main causes of CLD were hepatitis B virus (HBV) in 36 (19.0%); non-alcoholic fatty liver disease (NAFLD) in 32 (19.25%); and hepatitis C virus (HCV) in 18 (31.25%), in the groups with CLD without cirrhosis, CLD with cirrhosis, and LT recipients, respectively.

Concerning the time between the LT and the COVID-19, 3 of the patients were transplanted 3, 12, and 13 years before the infection. One patient was recently transplanted 3 months before the infection, and he was hospitalized. Regarding the immunosuppression of the 4 LT recipients, 1/4 (25%) were on monotherapy with tacrolimus. The remaining 3/4 (75%) were on triple therapy, one with cyclosporin A plus prednisone and mycophenolate mofetil (MMF), one with tacrolimus plus prednisone and azathioprine, and one with tacrolimus plus prednisone and MMF.

**Presenting symptoms and biological evolution of SARS-CoV-2 infection**

Clinical symptoms and biological data at time of COVID-19 infection are shown in Table 2. Respiratory symptoms at diagnosis (fever, dry cough, tiredness, shortness of breath, ageusia/anosmia) were present in 55%, 44%, and 100% of patients with CLD without cirrhosis, CLD with cirrhosis, and LT recipients, respectively. Gastrointestinal symptoms (abdominal pain, diarrhea, nausea, or

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**Table 2: Presenting symptoms and biological evolution of SARS-CoV-2 infection**

| Variables | CLD without cirrhosis (n = 22) | CLD with cirrhosis (n = 16) | LT recipients (n = 4) |
|-----------|-------------------------------|----------------------------|---------------------|
| COVID-19 symptoms, no. (%) | | | |
| Respiratory | 12 (55) | 7 (44) | 4 (100) |
| Gastrointestinal | 1 (5) | 1 (6) | 0 (0) |
| Both | 3 (14) | 1 (6) | 0 (0) |
| No symptoms | 6 (27) | 7 (44) | 0 (0) |
| Biochemical characteristics, mean (IQR) | | | |
| Serum sodium baseline, mmol/L | 140 (135–143) | 139 (135–144) | 140 (135–142) |
| Serum sodium during COVID-19, mmol/L | 137 (130–142) | 137 (117–142) | 144 (144) |
| Serum creatinine baseline, µmol/L | 80 (52–618) | 74 (54–158) | 77 (54–143) |
| Serum creatinine during COVID-19, µmol/L | 87 (46–740) | 114 (61–179) | 91 (91*) |
| Prothrombin time baseline | 12 (10–25) | 12 (11–23) | 11 (10–12) |
| INR baseline | 1.0 (0.9–1.3) | 1.0 (0.8–1.5) | 1.0 (0.8–1.0) |
| Serum albumin baseline, g/dL | 42 (25–46) | 39 (29–51) | 38 (27–48) |
| Serum albumin during COVID-19, g/dL | 36 (17–44) | 33 (22–47) | 31 (31*) |
| Total bilirubin baseline, µmol/L | 10 (4–382) | 11 (6–79) | 14 (8–21) |
| Total bilirubin during COVID-19, µmol/L | 10 (5–420) | 13 (7–189) | 6 (6*) |
| Alanine transaminase baseline, U/L | 27 (5–287) | 22 (9–106) | 19 (13–52) |
| Alanine transaminase during COVID-19, U/L | 47 (6–482) | 41 (9–116) | 23 (23*) |
| Alkaline phosphatase baseline, U/L | 69 (30–212) | 72 (48–258) | 76 (62–123) |
| Alkaline phosphatase during COVID-19, U/L | 86 (50–630) | 133 (40–500) | 100 (100*) |

* Data available for only one patient

SARS-CoV-2 = Severe acute respiratory syndrome coronavirus 2; CLD = Chronic liver disease; LT = Liver transplant; COVID-19 = Coronavirus disease 2019; ND = No data; INR = International normalized ratio
In patients with CLD without cirrhosis, 8 of 22 (36.4%) were hospitalized. Among them, 1 of 22 (4.5%) was transferred to the ICU, and 2 of 22 (9.1%) died from COVID-19-related complications. In patients with CLD with cirrhosis, 6 of 16 (37.5%) were hospitalized. Among them, 3 of 16 (18.8%) were transferred to the ICU and 2 of 16 (12.5%) died of COVID-19-related complications. In LT recipients, 1 of 4 (25.0%) was hospitalized. He was not transferred to the ICU and completely recovered.

**COVID-19 related deaths**
Overall, four patients died from COVID-19-related complications. Table 3 summarizes the main clinical characteristics, which are as follows:

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### Table 3: Clinical Characteristics of COVID-19-related Deaths

| CLD without cirrhosis | CLD with cirrhosis | LT recipients |
|-----------------------|--------------------|---------------|
| **Hospitalized** | **ICU** | **Died** |
| 8/22 (37.5%) | 3/16 (18.8%) | 8/22 (36.4%) |
| 14/22 (63.6%) | 1/16 (6.3%) | 14/22 (63.6%) |
| **Non-invasive ventilation** | **Non-invasive ventilation** | **Non-invasive ventilation** |
| 10/16 (62.5%) | 2/16 (12.5%) | 3/16 (18.8%) |
| 1/16 (6.3%) | 1/16 (6.3%) | 0/4 (0.0%) |

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**Figure 2: Clinical course and outcome of SARS-CoV-2 infection**

Overall, 15/42 (35.7%) of the COVID-19-infected patients were hospitalized. Seven out of 42 (35.7%) needed respiratory support. Four of 42 (9.5%) were transferred to the ICU, and 4/42 (9.5%) died of COVID-19-related complications.

CHUM = Centre hospitalier de l’Université de Montréal; SARS-CoV-2 = Severe acute respiratory syndrome coronavirus 2; ICU = Intensive care unit; CLD = Chronic liver disease; CP = Child–Pugh; LT = Liver transplant

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**vomiting**) were only present in two patients, one in CLD without cirrhosis and one with cirrhosis. Patients were asymptomatic in 27%, 44%, and 0% of each group, respectively.

**Patient treatment and outcome**
Treatment protocol evolved during the study period according to the international guidelines. Hospitalized patients received oxygen with adapted ventilatory support to treat hypoxemia. Antithrombotic therapy was done with enoxaparin (or heparin if renal dysfunction) administered daily. Other treatments, such as azithromycin 250 mg once daily plus ceftriaxone 2 grams daily, were administered on a case-by-case basis. Outpatients did not receive any specific treatment.

Outcome for each group is shown in Figure 2. Overall, 15 of 42 infected patients (35.7%) were hospitalized. Among them, 4 of 42 (9.5%) were transferred to the intensive care unit (ICU) and 4 of 42 (9.5%) died of COVID-19-related complications. In patients with CLD without cirrhosis, 8 of 22 (36.4%) were hospitalized. Among them, 1 of 22 (4.5%) was transferred to the ICU, and 2 of 22 (9.1%) died from COVID-19-related complications. In patients with CLD with cirrhosis, 6 of 16 (37.5%) were hospitalized. Among them, 3 of 16 (18.8%) were transferred to the ICU and 2 of 16 (12.5%) died of COVID-19-related complications. In LT recipients, 1 of 4 (25.0%) was hospitalized. He was not transferred to the ICU and completely recovered.
Table 3: Clinical characteristics of patients who died of SARS-CoV-2 infection

| Patient | Sex | Age (y) | Etiology of CLD | Cirrhosis (Child–Pugh) | Comorbidities | Residency at moment of infection | COVID-19 symptoms | COVID-19 treatment | Transfer to ICU | Ventilatory support | Cause of death |
|---------|-----|---------|-----------------|------------------------|---------------|----------------------------------|-------------------|-------------------|----------------|-------------------|----------------|
| Patient 1 | Male | 82 | Hepatitis B | No | None | Seniors’ residence | Respiratory No | No | Yes; non-invasive | Cardio-respiratory arrest | COVID-19 related lung injury |
| Patient 2 | Male | 50 | Hepatitis B | No | Obesity; arterial hypertension | Personal home | Respiratory No | Yes | Yes; invasive | Multiorgan failure | COVID-19 related lung injury |
| Patient 3 | Male | 52 | Hepatitis B | Yes; CP A | Trisomy 21 | Seniors’ residence | Respiratory No | No | Yes; non-invasive | Cardio-respiratory arrest | COVID-19 related lung injury |
| Patient 4 | Male | 69 | Hepatitis C | Yes; CP A | Quadriparesthesia; COPD; dyslipidemia; overweight | Seniors’ residence | Asymptomatic | No | Yes; non-invasive | Cardio-respiratory arrest | COVID-19 related lung injury |

SARS-CoV-2 = Severe acute respiratory syndrome coronavirus 2; CLD = Chronic liver disease; COVID-19 = Coronavirus disease 2019; ICU = Intensive care unit; COPD = Chronic obstructive pulmonary disease

- Patient 1: 82-year-old man who lived in a seniors’ residence home. He had chronic hepatitis B (e-antigen positive and e-antibody positive) without cirrhosis. His medical history included past esophagectomy for ruptured aortic aneurysm. He tested COVID-19 positive following development of respiratory symptoms that required hospitalization. Most recent laboratory tests available before COVID-19 infection showed HBV DNA levels of 2,236 IU/mL and normal blood counts and liver tests. Chest X-ray showed bilateral infiltrates, and non-invasive respiratory support was administered. Unfortunately, COVID-19-related lung injury progressed rapidly, causing death.

- Patient 2: 50-year-old man native from Republic of Congo with a medical history of chronic hepatitis B without cirrhosis, arterial hypertension, and obesity (BMI = 30). He was admitted from the emergency room with fever, dysgeusia, dyspnea, and desaturation and tested positive for COVID-19. Most recent laboratory tests available before COVID-19 infection showed HBV DNA levels of 2,236 IU/mL and normal blood counts and liver tests. Chest X-ray showed bilateral infiltrates, and non-invasive respiratory support was administered. Unfortunately, COVID-19-related lung injury progressed rapidly, causing death.

- Patient 3: 52-year-old man who lived in a seniors’ residence home. He was known with chronic hepatitis B (e-antigen positive and e-antibody positive) with compensated cirrhosis under tenofovir treatment. His medical history included trisomy 21. The COVID-19 test was positive in March 2020. Baseline laboratory tests before COVID-19 infection showed HBV DNA levels of 95 IU/mL and normal cell counts, creatinine levels, and liver tests. The patient developed COVID-19-related lung injury which evolved rapidly, causing death.

- Patient 4: 69-year-old man living in a seniors’ residence home. He had compensated cirrhosis secondary to chronic HCV and had been successfully treated with sofosbuvir and ledipasvir in the past. He also suffered from post-traumatic quadriparesthesia, lymphedema of the inferior right leg, polyneuritis secondary to a septic arthritis, depressive disorder, psoriasis, COPD caused by U/L. The patient rapidly developed respiratory failure requiring transfer to the ICU. He needed massive oxygen support with intubation, curarization, prone ventilation, and finally, extracorporeal membrane oxygenation, together with renal replacement therapy. After 3 weeks of intensive care, he developed intracranial parenchymal hemorrhage with intracranial hypertension and died.
cigarette smoking, and dyslipidemia. He also had a history of alcohol and marijuana consumption. The patient was asymptomatic, but he was tested after close contact with a positive patient in his seniors’ residence. Baseline initial laboratory before COVID-19 infection were unremarkable. The patient developed COVID-19-related lung injury which rapidly evolved, causing death.

**DISCUSSION**

The originality of this monocentric cohort study is that it is a real-world experience that encompassed 5,637 patients, almost the entire population with CLD with or without cirrhosis, and in LT recipients who were seen in our institution between April 1 and July 31, 2020. The rates of hospitalization and death are lower when compared with international registries with rates of hospitalization of 81% and death of 20% (4–5) and when compared with European case series with rates of hospitalization of 81% and death of 16% (8,9).

Interestingly, in this cohort, HBV was the main etiology of liver disease, followed by NAFLD. This probably represent a bias of our cohort of patients, since we have a large long-term HBV cohort of patients being regularly followed every 6 months. NAFLD, on the other hand, is the most prevalent CLD being seen for the first time and investigated in our clinic. In addition, liver injury in patients with SARS-CoV-2 and chronic HBV coinfection was associated with severity and poor prognosis of disease (10–12). In our cohort, 3 patients with chronic hepatitis B infection died, but they had many comorbidities.

In the current study, the predominant cause of death was COVID-19-related lung injury, as observed in 4 patients. The age and multiple comorbidities probably played an important role in the severe outcomes (13). However, of the other 38 patients with CLD, 4 with and 4 without cirrhosis had 3 or more comorbidities and the outcome was favourable. The most striking observation is that all but one death occurred in patients who were living in seniors’ resident homes: this is in line with the majority of individuals who died of COVID-19 during the first wave of spring 2020. It will be of interest to reevaluate if this holds true in later waves of COVID-19, where fewer patients living in these facilities suffered death.

Management of immunosuppressors in LT recipients is a topic of current controversy (14,15). In the four cases, no changes in immunosuppressors were done. Only one patient needed hospitalization, without further respiratory complication. At last follow-up, the four patients are alive without any complaints. More data is needed to better understand the management of this special population.

The strength of the current study includes the exhaustive review of all the cases from this one tertiary centre, which gives a very accurate picture of the prevalence of COVID-19 infection and outcome. However, our findings must be interpreted in the context of the study’s potential limitations. First, it is a monocentric study. Second, even though patients were contacted by phone which diminishes the loss of follow-up, the number of presential consultations were reduced, a limitation inherent to the pandemic.

In conclusion, this study demonstrates an unexpectedly low prevalence and low mortality in the context of COVID-19 infection in a tertiary reference centre including all patients with CLD with or without cirrhosis and LT recipients. This study provides valuable information that will help in understanding the SARS-CoV-2 pandemic in real-world terms.

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