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CASE REPORT

Liver transplantation for acute liver failure in a SARS-CoV-2 PCR-positive patient

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Current guidelines recommend deferring liver transplantation (LT) in patients with severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection until clinical improvement occurs and two PCR tests collected at least 24 hours apart are negative. We report a case of an 18-year-old, previously healthy African-American woman diagnosed with COVID-19, who presents with acute liver failure (ALF) requiring urgent LT in the context of SARS-CoV-2 polymerase chain reaction (PCR) positivity. The patient was thought to have acute Wilsonian crisis on the basis of hemolytic anemia, alkaline phosphatase:bilirubin ratio <4, AST:ALT ratio >2.2, elevated serum copper, and low uric acid, although an unusual presentation of COVID-19 causing ALF could not be excluded. After meeting criteria for status 1a listing, the patient underwent successful LT, despite ongoing SARS-CoV-2 PCR positivity. Remdesivir was given immediately posttransplant, and mycophenolate mofetil was withheld initially and the SARS-CoV-2 PCR test eventually became negative. Three months following transplantation, the patient has made a near-complete recovery. This case highlights that COVID-19 with SARS-CoV-2 PCR positivity may not be an absolute contraindication for transplantation in ALF. Criteria for patient selection and timing of LT amid the COVID-19 pandemic need to be validated in future studies.

KEYWORDS
clinical research / practice, immunosuppressive regimens - induction, infection and infectious agents - viral, liver disease: metabolic, liver transplantation / hepatology

Abbreviations: ACE2, angiotensin-converting enzyme 2; ALF, acute liver failure; Ct, cycle threshold; ICU, intensive care unit; IV, intravenous; LT, liver transplantation; MARS, molecular adsorbent recirculating system; NP, nasopharyngeal; PCR, polymerase chain reaction; SARS-CoV-2, severe acute respiratory syndrome coronavirus 2; WD, Wilson's disease.

Lavanya Yohanathan and Cristina Corsini Campioli are co-first authors, and contributed equally to the development of the manuscript.

1 | BACKGROUND

Most patients with COVID-19 have a mild or asymptomatic disease course; however, about 19% require admission to an intensive care unit (ICU) because of multiorgan failure.[1] These severe infections can result in up to 60% mortality.[2,3] Furthermore, signs of hepatobiliary damage have been observed in 14%–53% of patients with severe presentations of COVID-19.[4] Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) may directly damage the biliary ducts by binding to angiotensin-converting enzyme 2 (ACE2) on the surface of cholangiocytes; the particularly low abundance of ACE2 on hepatocyte surfaces suggests that SARS-CoV-2 may not directly infect the liver parenchyma.[4] Other potential mechanisms of hepatotoxicity include hyperinflammation and hypoxia-associated metabolic derangements.[5] Histopathologic changes in the liver include hepatic steatosis, portal fibrosis, ductal proliferation with lymphocytic infiltrates, lobular cholestasis, and acute liver-cell necrosis with central-vein thrombosis.[6,7] Patients with pre-existing cirrhosis appear to be at higher risk of mortality from COVID-19.[8] Acute liver failure (ALF) attributable to COVID-19 is exceptionally rare.[9,10] Liver transplantation (LT) remains the best treatment option for decompensated cirrhosis and ALF. However, Transplantation Society Guidelines recommend that LT candidates have resolved COVID-19 symptoms with two negative PCR tests done at least 24 hours apart before proceeding to LT.[4] Herein, we report a case of ALF resulting in LT in a patient with COVID-19 and ongoing SARS-CoV-2 polymerase chain reaction (PCR)-positivity.

2 | CASE

An 18-year-old, previously healthy African-American woman presented with 3 days of fever, nausea, vomiting, diarrhea, epigastric pain, shortness of breath (day 0), and known recent SARS-CoV-2 exposure. At presentation to the hospital (day 3), she tested positive for SARS-CoV-2 by PCR from a nasopharyngeal (NP) sample. Vital signs demonstrated blood pressure 133/66 mmHg, pulse 103, respiratory rate 28 breaths per minute, temperature 37.5°C, and oxygen saturation as low as 80% on ambient air. Physical examination revealed scleral icterus and epigastric tenderness. Notable laboratory results (Table 1) included severe coagulopathy, hyperbilirubinemia (total12.7 mg/dl, direct 9.1 mg/dl), aminotransferase elevations (AST 596 U/L, ALT 37 U/L), profound hemolytic anemia (hemoglobin =4.5 g/dl, MCV = 133.6, reticulocyte 13.4%, peripheral smear -mild spherocytosis), elevated creatinine (7.5 mg/dl). Arterial blood gas demonstrated a pH = 7.17, pO2 = 39 mmHg, pC02 = 24, and bicarbonate =9 mmol/L. Her Quick COVID-19 Severity Index (qCSI) Risk[11] was high-intermediate for risk of critical illness at 24 hours, defined by oxygen requirement (>10 L/min by low-flow device, high-flow device, noninvasive, or invasive ventilation) or death.

Evaluation for acute hepatitis including viral, autoimmune serologies, and acetaminophen levels were negative. Serum inflammatory markers were markedly elevated including ferritin (99,230 mcg/L) and C-reactive protein (245 mg/L). A normal ceruloplasmin (21.1 mg/dl) was noted along with a low alkaline phosphatase (21 U/L) and uric acid (2.4 mg/dl) and an elevated serum copper (4.67 mcg/ml). Urine copper quantification was precluded by anuria. A slit lamp examination did not identify Kayser–Fleischer rings. Chest radiography did not show any consolidations or pleural effusions. Computed tomography of the abdomen and pelvis demonstrated nonspecific mesenteric stranding, a normal appearing liver, and splenomegaly (14.8 cm). Liver ultrasonography showed a slightly hypoechoic hepatic echotexture. Bone marrow biopsy revealed mild hemophagocytosis.

The patient's timeline is shown in Figure 1. She was admitted to a dedicated COVID-19 ICU. Intermittent hemodialysis (IHD) was initiated (day 4) and continuous renal replacement therapy (CRRT) was started on day 6. Treatment for SARS-CoV-2 included 2 units of convalescent plasma (day 5–6). Hepatic and renal failure precluded the use of remdesivir. Due to a direct antiglobulin test (gel-based assay) that was positive (regular direct antiglobulin test, tube-based assay, was negative) and ongoing hemolysis, intravenous (IV) methylprednisolone 125 mg daily was administered.

TABLE 1 Index laboratory tests at presentation and the day of liver transplantation

Lab	Index	Transplant	Lab	Index	Transplant	Lab	Index	Transplant
WBC	37.3	18.1	Na^+	125	146	CoV-PCR	Positive	Positive
HGB	4.5	7.5	K^+	5.5	4.4	CK	6,512	2,529
MCV	133.6	98.7	HCO_3	7	23	Ferritin	99,230	9,455
PLT	171	39	Creatinine	7.50	2.57	CRP	245	19.9
Haptoglobin	<14	—	BUN	38	36	Ammonia	64	125
INR	6.9	2.8	Ca^{2+}	6.8	8.5	Lactate	19	9.5
Fibrinogen	91	190	Glucose	98	136	ESR	17	—
D-Dimer	14,372	—	AST	596	545	Uric acid	2.4	—
LDH	>2,250	1,563	ALT	37	419	Copper	4.74	—
T. Bilirubin	12.7	21.9	ALP	21	50	Ceruloplasmin	21.1	—
D. Bilirubin	9.1	17.2	ALB	2.1	2.7			

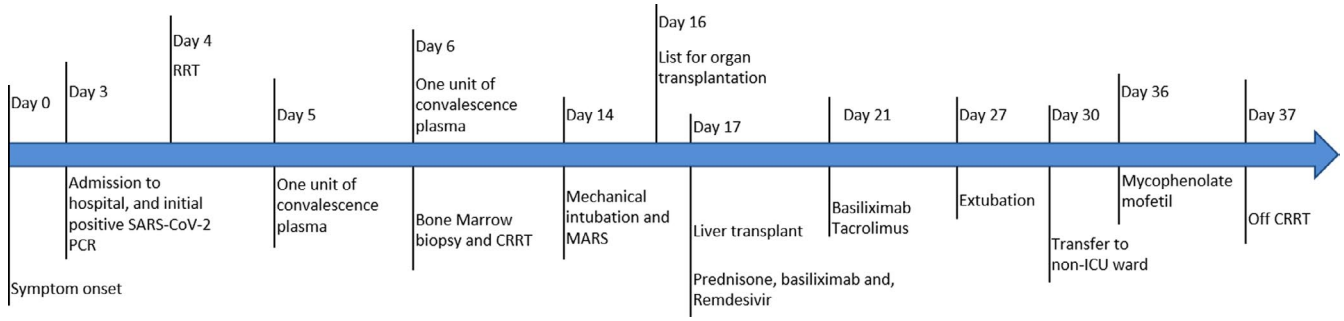

FIGURE 1 Patient event timeline [Color figure can be viewed at wileyonlinelibrary.com]

TABLE 2 Severe acute respiratory syndrome coronavirus-2 polymerase chain reaction timeline

Days since initial symptoms	3	7	11	13	14	15	16	17[a]	21	27
SARS-CoV–2 PCR nasopharyngeal swab	+	+	+	+	+	+		+	+	–
SARS-CoV–2 PCR bronchoalveolar lavage							+			
Ct value			22.9[b]		14-20[c]	14-20[c]	23.3[b]		30.8[b]	

Abbreviations: Ct, cycle threshold; PCR, polymerase chain reaction; SARS-CoV-2, severe acute respiratory syndrome coronavirus 2.

[a]Day of liver transplant.

[b]Real-time reverse transcription polymerase chain reaction, SARS-CoV-2, molecular detection.

[c]Real-time reverse transcription polymerase chain reaction, influenza A and B, SARS-CoV-2, PCR, rapid.

Transjugular liver biopsy was recommended by the liver transplant team (day 4) given concern for Wilson's disease (WD) but was not performed due to rapidly rising INR, eventually exceeding 20. Genetic testing for WD was obtained. Despite supportive care, the patient progressively deteriorated and met criteria for ALF with hepatic encephalopathy (HE), coagulopathy, and absence of known prior liver disease. Although the patient had an abnormal hepatic echotexture on ultrasound, and the spleen was mildly enlarged, there were no specific findings of portal hypertension or clear evidence of cirrhosis on CT imaging. On day 14, patient had progressive HE (plasma ammonia 111 mcmol/L) despite lactulose and rifaximin. She continued to require higher respiratory support receiving 65% Fio2 by Optiflow requiring endotracheal intubation and mechanical ventilation. The chest radiograph prior to intubation demonstrated bilateral airspace opacities and retrocardiac consolidation. Albumin dialysis using the Molecular Adsorbent Recirculating System (MARS) was initiated as a potential bridge to transplant (four consecutive daily sessions). An intracranial pressure monitor could not be placed due to coagulopathy. Head CT was performed on days 11, 12, 14, and 16 that did not demonstrate overt cerebral edema but did show subtle narrowing of the lateral and third ventricle suggestive of subtle edema. Prolonged video electroencephalogram demonstrated diffuse, severe disturbances in cerebral function but no evidence of seizures or epileptiform activity. Extensor posturing and myoclonic jerks were noted on exam despite MARS, but this improved with institution of IV hypertonic saline. Neurologists following the patient daily, noted intact brainstem reflexes, and did not see any signs of irreversible brain injury.

On day 16, and after extensive multidisciplinary review, she was listed for liver transplant as status 1A (calculated MELDNa = 53), despite a persistently positive NP SARS-CoV-2 PCR, and absence of SARS-CoV-2 antibodies. PCR cycle threshold (Ct) values were consistently between 14 and 16 (Table 2). The presumptive diagnosis was ALF secondary to WD, but atypical COVID-19-related liver disease could not be completely excluded. The patient was hemodynamically stable on CRRT, not requiring vasopressor support at the time of listing. She was receiving FiO2 of 0.30 by mechanical ventilation with new left lower lobe consolidation and mild patchy infiltrate in the right lung base. Empiric IV ceftriaxone, vancomycin, and anidulafungin were initiated. Serial blood cultures remained negative. Ferritin and C-reactive protein had improved to 7491 mcg/L and 8.6 mg/L, respectively (Table 1).

The patient underwent a deceased donor LT on day 17. The intraoperative course was unremarkable. The explant demonstrated cirrhosis, confluent hepatocyte necrosis, and steatohepatitis (Figure S2). Quantitative copper was 1200 mcg/dry weight liver, confirming WD. The genetic analysis later demonstrated two pathogenic variants of the ATP7B gene (amino acid change: p.H1069Q [His1069Gln]; amino acid change: p.D730Gfs*25 [Asp730Glyfs*25]).

Postoperatively, her liver ultrasound demonstrated patency of all vascular anastomoses with normal waveforms. The immunosuppression regimen consisted of standard steroid taper, with basiliximab (20 mg IV on day 17 and day 21), which is our standard regimen for patients with renal failure. Mycophenolate mofetil was held due to COVID-19.[12] Tacrolimus was initiated on day 21. Remdesivir was

started immediately posttransplant (200 mg Per NG tube day 1, 100 mg days 2–10). On day 21 (4 days post transplant), the PCR Ct value was 30.8 (Table 2), and the PCR resulted negative on day 27 (10 days post transplant). No antibodies to SARS-CoV-2 were detected on days 4 and 15. The SARS-CoV-2 PCR on the explant was positive.

The patient was extubated on day 27. Shewas maintained on CRRT until day 37, transitioned to IHD and ultimately regained renal function off IHD with a current creatinine of 0.91 mg/dl. Currently, she has a stable, moderately elevated transaminase levels (AST = 109, ALT = 173 U/L, total bilirubin of 1.3 mg/dl). The alkaline phosphatase increased to >1000 U/L and a liver biopsy on day 47 demonstrated mild zone 3 injury without evidence of T cell–mediated rejection. Ultrasound with Doppler of the allograft at that time was normal.

She also had severe weakness in a lower motor neuron pattern and diffuse muscle pain. Creatinine kinase was elevated to a peak of 6512 U/L and urine myoglobulin was positive indicating rhabdomyolysis. There was concern for COVID-19-related myopathy; prednisone was initiated at 60 mg per os daily with significant improvement in myalgias. An EMG demonstrated a diffuse myopathic process. A deltoid muscle biopsy showed severe, active myopathy, myosinolysis, and denervation atrophy favoring critical illness myopathy, but a superimposed process could not be ruled out given prior empiric prednisone treatment. Creatinine kinase normalized on day 39. She was dismissed home after a physical rehabilitation stay and is without cognitive or neurologic deficits.

3 | DISCUSSION

We present a case of LT in the setting of a persistently positive SARS-CoV-2 PCR. Although there have been reports on COVID-19 in transplant recipients in the postoperative period, to our knowledge, this is the first intentional LT performed in a patient with active SARS-CoV-2 PCR positive infection.[13] One other case has been reported; however, the patient was PCR negative prior to LT.[14] Liver injury is widely reported in the setting of COVID-19, although ALF due to COVID-19 seems to be exceedingly rare and reported cases have potential competing diagnoses for ALF.[9,10] Infections have been reported to trigger acute Wilsonian crisis, and more recently, one case of SARS-CoV-2 infection "unmasked" WD was published.[15]

ALF secondary to WD was thought to be probable, but ALF as an unusual manifestation of COVID-19 could not be entirely excluded. The other diagnostic consideration was hemophagocytic syndrome as the patient met 5 criteria for this condition (elevated ferritin, cytopenia, splenomegaly, hypofibrinogenemia, and hemophagocytes on bone marrow biopsy). The clinical judgement was that the hemophagocytosis and elevated ferritin were secondary to severe SARS-CoV-2 infection, and that the splenomegaly, hypofibrinogenemia, and cytopenias were secondary to WD. The multidisciplinary decision to offer LT despite ongoing SARS-CoV-2 PCR positivity was based on a very high likelihood of death without transplantation, a reasonable likelihood that ALF was due to WD, and taking into account the hemodynamic stability, lack of significant lung injury, dramatic improvement in inflammatory markers (i.e., COVID-19 hyperinflammatory state), the 15-day window from the time of COVID-19 diagnosis to LT, young age, and prior excellent health. These positive factors were balanced against the potential possibility that ALF was due to COVID-19 (with an unknown post-LT course), the known increased risk of death in patients with COVID-19 infection who undergo any surgical procedure, possible increased risk of death in solid organ transplant recipients, as well as the unknown impact of starting high-dose immunosuppression in the mid of a COVID-19 infection.[13,16,17]

We used our standard renal-sparing immunosuppression protocol (basiliximab, corticosteroids, mycophenolate mofetil), except we held mycophenolate mofetil given her active SARS-CoV-2 infection and data suggesting increased mortality in LT recipients on mycophenolate mofetil.[12] We started remdesivir immediately after LT, despite the known risk of elevated liver tests with this medication as well as the patient's renal failure, as we thought the risk of COVID-19 outweighed the risk of remdesivir side effects.[18,19] It is unknown whether remdesivir contributed to the patient's eventual recovery from COVID-19 or if it contributed to the elevated aminotransferases after LT. The patient also had renal failure and myopathy, presumably from WD and critical illness myopathy, respectively. However, rhabdomyolysis can also occur with acute Wilsonian crisis.[20] Additional immunohistochemical stains on the muscle biopsy did not demonstrate presence of an interferonopathy-related myopathy that can be seen with SARS-CoV-2 infection.[21] The role of SARS-CoV-2 as a trigger for the development of ALF due to WD is unknown although there are prior case reports of other viral infections potentially triggering ALF due to WD.[22,23] The positive explant SARS-CoV-2 PCR adds further to the intrigue about the pathophysiologic role.

Regarding the SARS-CoV-2 infection and timing of LT, the PCR Ct value was still relatively low at 21. Although we could not verify if this were live/replicating virus due to inaccessibility of viral culture, this low value suggested ongoing high-grade shedding of viral nucleic acid. Recent data suggest that Ct values as low as 20 are associated with 76.7% recovery of viral growth in cell culture, whereas the rate drops to 2.9% above a Ct value of 30.[24] Not until the patient received remdesivir posttransplant did the Ct increase, and eventually, the PCR test became negative (day 27).

Given the potential for excellent long-term outcome for LT performed for ALF due to WD, and the near-certainty of mortality without transplantation, we opted to proceed to LT despite active SARS-CoV-2. At present, the patient has excellent graft function and has returned home. Given the high prevalence of SARS-CoV-2 infection, and the low incidence of reported ALF, it is imperative to look for another cause of liver disease when patients with ALF test positive for SARS-CoV-2. Although it is difficult to provide a general conclusion for proceeding to LT in the context of ongoing SARS-CoV-2 infection, we demonstrated that it is possible to do so successfully

when the underlying reason for ALF is not driven by SARS-CoV-2, there is limited pulmonary involvement and significant improvement in the "cytokine storm" as determined by inflammatory markers. The criteria applied herein for patient selection and LT timing needs to be validated in future studies.

DISCLOSURE

The authors of this manuscript have no conflicts of interest to disclose as described by the *American Journal of Transplantation*.

DATA AVAILABILITY STATEMENT

Deidentified data are available on reasonable request.

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SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section.

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