

BRIEF COMMUNICATION

COVID-19 in lung transplant recipients: A single-center experience

Sinan Turkkkan¹  | Muhammet Ali Beyoglu¹  | Mehmet Furkan Sahin¹  |
Alkin Yazicioglu¹  | Yasemin Tezer Tekce²  | Erdal Yekeler¹

¹ Department of Thoracic Surgery and Lung Transplantation, Ankara City Hospital, University of Health Sciences, Ankara, Turkey

² Department of Infectious Diseases, Ankara City Hospital, University of Health Sciences, Ankara, Turkey

Correspondence

Sinan Turkkkan, Department of Thoracic Surgery and Lung Transplantation Clinic, Ankara City Hospital, University of Health Sciences, Üniversiteler Mahallesi 1604. Cadde No: 9 Çankaya/Ankara.
Email: dr.turkkkan@hotmail.com

Abstract

Background: Coronavirus disease 2019 (COVID-19) is a global health problem. However, the course of this disease in immunosuppressed patients remains unknown. This study aimed to describe the course of COVID-19 infection and its effects on lung transplant recipients.

Methods: This was a single-center, retrospective, observational study. The recipients with suspicious symptoms and/or a contact history with infected individuals were diagnosed with COVID-19 by performing a reverse transcription-polymerase chain reaction (RT-PCR) test using samples obtained from the nasopharynx swabs or bronchial lavage. We classified the patients into mild, moderate, and high severity groups according to their clinical conditions. In patients with positive RT-PCR results, cell cycle inhibitor drugs were withdrawn, while steroids were maintained at the same level as in patients without clinical deterioration.

Results: Of the seven recipients diagnosed with COVID-19 infection, one experienced a re-infection. Each recipient had at least one comorbidity. Smell disorder (12.5%), cough/dyspnea (37%), and fever/chills/shivering (37%) were the most frequent symptoms. The mean follow-up time after infection was 108 days. No deaths were recorded due to COVID-19; however, the pulmonary function test values of two recipients were decreased during subsequent follow-ups.

Conclusion: In our small group of transplant recipients with COVID-19, there were two cases of pulmonary function deterioration and a case of re-infection, and no recipient died. It is suggested that steroid therapy should be initiated in the early period in patients with pulmonary opacities.

KEYWORDS

COVID-19, lung transplant, rejection

Abbreviations: CNI, calcineurin inhibitor; COVID-19, coronavirus disease 2019; CRP, C-reactive protein; FEV1, forced expiratory volume in one second; HFO, high-flow oxygen; HQN, hydroxychloroquine; IL-6, interleukin-6; MMF, mycophenolate mofetil; mTOR, mammalian target of rapamycin; PFT, pulmonary function tests; RT-PCR, reverse transcription-polymerase chain reaction; SARS-CoV-2, severe acute respiratory syndrome coronavirus 2; SOT, solid organ transplant

1 | INTRODUCTION

The coronavirus disease 2019 (COVID-19) pandemic caused by the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) has spread worldwide within a short time.¹ The course of COVID-19 in immunosuppressed patients remains unknown. Previous studies have reported that COVID-19 has high morbidity and mortality rates in solid organ transplant (SOT) recipients.² Available data on COVID-19 regarding lung transplant recipients are limited compared to liver and kidney transplant recipients. Therefore, we sought to describe the course and short- to long-term COVID-19 outcomes in lung transplant recipients.

2 | MATERIALS AND METHODS

2.1 | Patient selection and examination

This was a single-center, retrospective, observational study to evaluate the effects of COVID-19 in lung transplant recipients. A positive SARS-CoV-2 reverse transcription-polymerase chain reaction (RT-PCR) test was required for diagnosis. The test was performed using nasopharyngeal swab or bronchial lavage samples obtained from recipients presenting with suspicious symptoms and/or a contact history with individuals infected with SARS-CoV-2.

Inpatient treatment was recommended for all recipients with positive RT-PCR. Individuals presenting with symptoms of cough, fever, dyspnea, generalized muscle pain, and/or radiological findings were hospitalized while waiting for the RT-PCR results. Outpatients with mild symptoms or negative RT-PCR results were advised to visit the hospital in case of symptoms such as fever, cough, dyspnea, and diffuse pain. Coagulation parameters, including D-dimer, fibrinogen, troponin, interleukin-6 (IL-6), ferritin, as well as routine hemogram, biochemistry, C-reactive protein (CRP), and procalcitonin tests were performed. Posteroanterior (PA) chest radiography and thoracic computed tomography (CT) were also performed. Respiratory virus PCR panel and influenza rapid test were performed using oropharyngeal swabs obtained from each hospitalized patient. COVID-19-specific imaging findings (infiltration, consolidation, and ground-glass opacities) were examined using PA radiography and CT.

2.2 | Patient classification

We classified the patients into mild, moderate, and high severity groups according to their clinical conditions. Patients who were followed up as outpatients or those who did not need supplemental oxygen were classified in the mild severity group. Patients who were admitted in the ward requiring supplemental nasal oxygen at any time during their follow-up were included in the moderate severity group, whereas those requiring noninvasive ventilation, high-flow oxygen (HFO) treatment, or invasive mechanical ventilation were included in the high severity group.

2.3 | Immunosuppressive therapy

In patients with positive RT-PCR results, cell cycle inhibitor drugs were withdrawn. The target blood levels of calcineurin inhibitor (CNI) and mammalian target of rapamycin (mTOR) inhibitor did not change. Steroids were maintained at the same level as in patients without clinical deterioration.

2.4 | COVID-19 treatment

Recipients with suspected COVID-19 symptoms and/or positive RT-PCR results were treated in accordance with the recommendations of the Ministry of Health Scientific Committee. In the early stages of the pandemic, hydroxychloroquine (HQN) (loading dose, 2×600 mg; maintenance dose, 2×200 mg/day for 4 days) and favipiravir (loading dose, 2×1600 mg; maintenance dose, 2×600 mg/day for 4 days) were preferred. Subsequently, HQN was removed from the treatment with the newly released information reported in the literature,^{3,4} and remdesivir is preferred in eligible patients as reported by a clinical study. Steroid augmentation (1 mg/kg methylprednisolone administered as 2 doses per day; after 5 days, 1×1 mg/kg; and then by reducing 20 mg/day every 5 days, it was decreased to 10 mg/day) and pulse steroid therapy were administered (1000 mg/day for 3 days; subsequently 1×1 mg/kg for 5 days; and by decreasing 20 mg/day every 5 days, it was reduced to 10 mg/day) in case of clinical and/or radiological deterioration. Venous thromboembolism prophylaxis with low-molecular-weight heparin and, if needed, empirical antibiotic treatment were initiated in inpatient lung transplant recipients. The pulmonary function test (PFT) and 6-minute walking test were evaluated after the first month of infection.

3 | RESULTS

RT-PCR tests for SARS-CoV-2 infection were performed in 11 lung transplant recipients. Of the 11 recipients, 6 (54.5%) had suspicious symptoms/findings, while 5 (45.5%) were in contact with infected individuals. All the samples of symptomatic patients were positive for SARS-CoV-2 RT-PCR ($n = 6$), while only two samples of those who were screened with a contact history (2/5 [40%]) revealed positive results (Figure 1). In total, eight COVID-19 cases, of which, one was a reinfection that occurred approximately 8 months after the first infection, were detected in seven patients (two females and five males). Of these seven recipients, one (12.5%) had an impaired sense of smell, three (37%) presented with cough and dyspnea, and three (37%) with fever, chills, and shivering.

The mean age was 48 years (range, 38–56 years). Three patients underwent lung transplantation for bronchiectasis, two for chronic obstructive pulmonary disease, one for idiopathic pulmonary fibrosis, and one for histiocytosis X. The average time after transplantation was 38.5 months (range, 11–82 months; median, 28 months), and the average follow-up period of patients after COVID-19 infection was 108

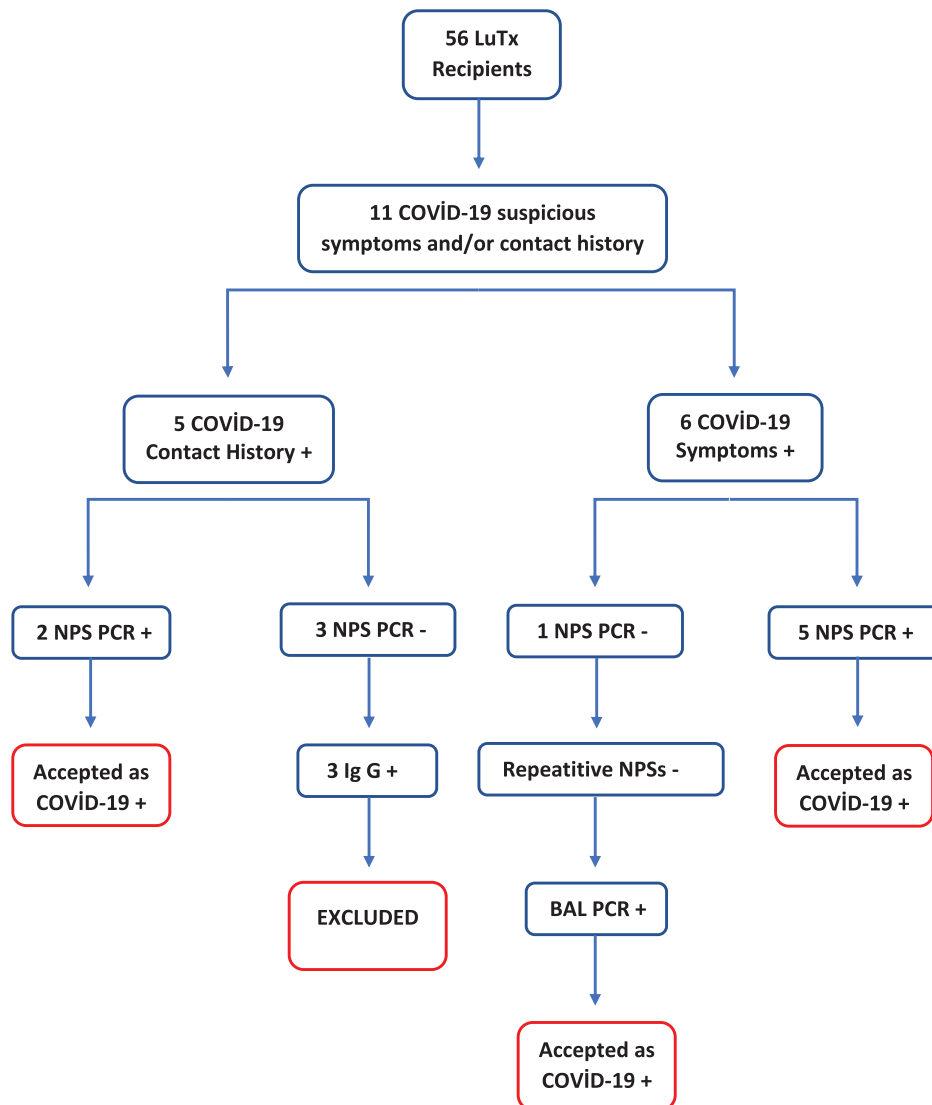


FIGURE 1 Diagnosis algorithm of the cases. BAL, bronchoalveolar lavage; COVID-19, coronavirus disease-2019; LuTx, lung transplant; NPS, nasopharyngeal swab; PCR, polymerase chain reaction

days (range, 20–240 days; median, 96 days). Each patient had at least one comorbidity (nephropathy, hypertension, diabetes mellitus in 1, 5, and 5 cases, respectively) (Table 2). Four patients received triple immunosuppressive therapy consisting of tacrolimus, mycophenolate mofetil (MMF), and corticosteroid. Three patients' immunosuppressive regimens consisted of an mTOR inhibitor (everolimus) along with a reduced level of tacrolimus and titrated doses of MMF and corticosteroid (Table 1).

Only one patient, who had a re-infection, was administered pulse steroid therapy in the last 6 months because of acute cellular rejection. No other cases were administered induction or rejection therapy in the last 6 months.

At admission, bilateral ground-glass opacities and consolidation in both lungs were observed on the radiographs/CT scan of three patients (37%). On days 7 and 8 of admission, the imaging of two more cases revealed characteristic bilateral lung findings, and cytokine storm find-

ings were shown in blood tests (total, five cases [62.5%] with pulmonary disease).

The mean, range, and median laboratory values of patients in each class are shown in Table 2. Unlike lymphopenia, which further decreased with disease severity, D-dimer, ferritin, and IL-6 values were found to increase with the severity of the disease.

SARS-CoV-2 specific treatment was administered in the seven recipients with positive RT-PCR tests, including the case of re-infection (Table 1). Remdesivir was only administered to the patient (12.5%) who had a re-infection. The other seven cases (87.5%) during the initial infection received favipiravir. Steroid augmentation protocol was applied to four patients (50%): two patients (25%) with dyspnea and radiographic findings at the time of hospitalization and two patients (25%) with new-onset dyspnea and infiltration or ground-glass opacities during the cytokine storm period that were not previously present at admission. Pulse steroid was administered to three patients because

TABLE 1 Demographic data of the cases

Cases	Age	Indication	Immunosuppressive therapy	Time after tx (months)	Co-morbidity	Initial Symptoms	Severity	Time after COVID-19 (days)	Medication for COVID-19	Outcomes
1	56	COPD	Tac+ Eve+ MMF+ CS	53	Low GFR, HT	Cough, dyspnea	High	240	Fav+Hqn+ (PS, after detecting ACR)	ACR, CLAD, oxygen dependency for 24 h/day
2	49	IPF	Tac+ MMF+ CS	25	DM, HT	Cough, dyspnea, fever, Chills, shivering	High	150	Fav+SA+PS	Recovered
3	48	Bronchiectasis	Tac+ MMF+ CS	29	DM, HT	No	Moderate	145	Fav+SA+PS	Recovered
4	52	H-X	Tac+ Eve+ MPA+ CS	82	DM	Smell dysfunction	Mild	142	Fav	Recovered
5	53	COPD	Tac+ Eve+ CS	26	DM, HT	Cough, dyspnea	High	60	Fav+SA+PS	Loss of FEV ₁ value by %30, decrease in 6-MWT
6	38	Bronchiectasis	Tac+ MMF+ CS	22	HT	Fever, Chills, shivering	Moderate	55	Fav+SA	Recovered
7 (re-infection)	56	COPD	Tac+ Eve+ MMF+ CS	60	HT, Low GFR	No	Mild	52	Rem	Recovered
8	41	Bronchiectasis	Tac+ MMF+ CS	11	DM	Fever, Chills, shivering	Mild	20	Fav	Recovered

Abbreviations: ACR, Acute cellular rejection; CLAD, Chronic lung allograft dysfunction; COPD, Chronic obstructive pulmonary; CS, Corticosteroid; DM, diabetes mellitus; Eve, everolimus; Fav, favipiravir; FEV₁, forced expiratory volume in first second; GFR, glomerular filtration rate; HT, hypertension; H-X, histiocytosis-x; mTOR, mammalian target of rapamycin inhibitor; MMF, mycophenolate mofetil; MPA, mycophenolic acid; PS, pulse steroid; Rem, remdesivir; SA, steroid augmentation; Tx, transplantation; 6-MWT, six minute walking test.

TABLE 2 Laboratory data of the cases according to disease severity

Parameters	Mild (n = 3)	Moderate (n = 2)	High severity (n = 3)	Total (mean, range, median)
Leucocytes (x10 ³ /ml)	4.7 (4.1–5.7)	3.0 (2.3–3.7)	6.0 (1.7–9.4)	4.8 (1.7–9.4; 4.3)
Lymphocytes (x10 ³ /ml)	1.2 (1.0–1.4)	1.4 (1.4–1.4)	1.3 (0.2–2.9)	1.3 (0.2–2.9; 1.3)
% Leucocytes	68.8 (62.7–74.3)	60.5 (55.6–65.5)	80.3 (72.5–86)	71.0 (55.6–86.0; 71.5)
%Lymphocytes	18.7 (12.6–22)	29.8 (25.3–34.4)	13.5 (8.6–22.1)	19.5 (8.6–34.4; 21.7)
Lowest % lymphocytes	16.4 (10.2–22)	8.35 (6.1–10.6)	4.8 (3.2–6.6)	9.1 (3.2–22; 8.3)
Fibrinogen (g/L)	3.82 (2.22–5.16)	2.58 (2.03–3.13)	4.73–4.14 (4.03–6.03)	3.8 (2.0–6.0; 4.1)
D-dimer (mg/L)	0.6–0.6 (0.5–0.7)	0.6 (0.3–0.9)	0.9 (0.6–1.4)	0.7 (0.3–1.4; 0.7)
LDH (U/L)	339 (234–525)	383 (234–532)	374 (300–485)	363 (231–532; 318)
Ferritin (μg/L)	15 (12–18)	21 (10–32)	101.6 (23–146)	53 (10–146; 23)
IL-6 (at admission) (pg/L)	6.26 (5.9–6.9)	9.75 (8.9–10.6)	40.2 (19.9–64.4)	21.9 (6.9–64.4; 15.2)
IL-6 (maximum) (pg/L)	11 (6.9–14.3)	46.6 (38.0–55.3)	112 (36.4–235)	64.3 (6.9–235.0; 38.0)
CRP (g/L)	36.6 (6–91)	20.5 (12–29)	75.3–100 (18–108)	47.1 (6–108; 18)
Procalcitonin (μg/L)	0.033 (0.02–0.05)	0.03 (0.03–0.03)	0.17 (0.03–0.47)	0.08 (0.02–0.47; 0.04)
Initial chest X-ray	no	no	Bilateral infiltration (3)	

Abbreviations: CRP, C-reactive protein; IL-6, interleukin-6; LDH, lactate dehydrogenase.

they did not show any improvement in symptoms, inflammatory markers, or radiological findings despite steroid augmentation. Since clinical improvement was achieved with pulse steroid therapy, other management strategies with convalescent plasma, IL-6, and IL-1 receptor antagonists were not used. Although no bacterial infection was detected in any of the cases, antibiotic treatment was administered upon admission or during the follow-up period based on previous culture and antibiogram results (1 colistimethate, 2 meropenem, 5 tazobactam, and sultamicillin).

The target levels of CNI and mTOR did not change; however, MMF administration was stopped after detecting positive RT-PCR results.

Seven (87.5%) cases were followed up as inpatients, and one (12.5%) was followed up as an outpatient. Two (28.5%) of these hospitalized cases were admitted to the intensive care unit, whereas five (71.5%) were admitted to the ward. The average intensive care unit stay was 13 days (range: 8–18 days), and the mean hospitalization period for these two patients was 33.5 days (range: 25–42 days), while the average hospital stay for the others was 18.6 days (median 17 days, range 15–42 days). Two patients required HFO, noninvasive mechanical ventilation, and supportive oxygen treatments with reservoir masks. Supplemental oxygen with a nasal cannula was applied to two patients hospitalized in the ward. Two (28.5%) cases did not require oxygen during their hospital stay.

3.1 | Treatment results

The patient with smell dysfunction treated as an outpatient improved within five days of treatment. Only one patient required long-term oxygen therapy. Decreased PFT values were detected in two cases during subsequent follow-ups, while the PFT values of the five remaining

cases were stable. No patient had any co-infection or signs of COVID-19 complications involving organs other than respiratory. No patient died due to COVID-19.

4 | DISCUSSION

Data regarding COVID-19 infection in lung transplant recipients are gradually increasing. However, to date, most studies have focused on management and short-term results.⁵ In this study, we aimed to investigate the effects of COVID-19 on demographic, clinical, and laboratory data of patients and its short- to long-term clinical outcomes in lung transplant recipients.

From the beginning of the pandemic, several clinicians and researchers were curious to know the impact of COVID-19 on SOT recipients and the recipients with the greatest disease severity. We detected eight cases of COVID-19 (14.3%) among 56 recipients under follow-up, of which, one was a re-infection. Although our sample of lung transplant recipients is very small, the 14% incidence of the disease among recipients suggests that recipients should be vigilant regarding COVID-19 prevention.

It is a well-known fact that infections in immunosuppressed patients may present with atypical symptoms and signs, which may lead to delays in diagnosis and treatment.⁶ It is also known that COVID-19 intensifies by causing cytokine storms in the following days.⁷ Since we had sufficient numbers of negative pressure, isolation, intensive care, and ward room allocated to our clinic in the hospital, we preferred treating our patients by hospitalizing them regardless of their symptoms. Therefore, we detected new-onset fever, dyspnea, and chest radiography findings in two cases (25%), which were related to cytokine storms on the 7th to 8th days of hospitalization.

There are also insufficient data on SARS-CoV-2 re-infection. Only approximately 30 cases were reported by the end of 2020, and we did not find a re-infection case of a lung transplant recipient in the English literature. The most important aspect of this issue is the distinction between re-infection and reactivation.⁸ One of the cases was evaluated as a re-infection case, although sequence analysis was not performed. He received remdesivir as part of the study. His first attack led him to use long-term oxygen therapy, but as expected,^{8,9} he survived his second attack with mild symptoms.

Studies are ongoing to access demographic and laboratory data of patients with progressive disease.¹⁰ To date, some predictive markers have been suggested.¹¹ Although statistical analyses were not performed due to the small number of cases, there are some prominent findings. Our data, similar to the results of some other studies,¹² show that the elevation in D-dimer, CRP, and IL-6 observed at the time of application, the highest IL-6, ferritin, and procalcitonin levels were proportional to the severity of COVID-19. We observed that the low percentage of lymphocytes during patient follow-up was also proportional to the severity of the disease (the percentage of lymphocytes was lower in more severe disease), indicating that lymphocyte depletion is a part of the pathogenesis of COVID-19.¹³ In addition, the lymphocyte percentages of the two patients who required intensive care follow-up were the lowest in the study, and IL-6 levels were the two highest values.

Our practice in immunosuppressive drug management has been stopping antimetabolite drugs and going on the same target levels of CNI and mTOR inhibitor drugs. Several articles have pointed out the positive and negative aspects of everolimus and tacrolimus during the course of COVID-19.¹⁴⁻¹⁷ Moreover, many parameters determine the development and progression of the disease,¹⁸ but we believe that one of the important findings in our study was the presence of everolimus in the immunosuppressive drug regimens of three patients. This finding requires clinical and statistical verification in more patients.

At the beginning of the pandemic, there was insufficient data on the effects of high-dose steroids on COVID-19 in SOT recipients. Increasing data on this issue has encouraged clinicians during treatment applications.¹⁹ In cases with pulmonary involvement and findings of cytokine storm, high-dose steroid therapies (steroid augmentation/pulse application) were applied to four of our cases, three of whom were administered pulse steroids. Symptoms and inflammatory markers improved in all three patients after pulse steroids; thus, we did not need treatments such as tocilizumab and anakinra or other treatments.

The average follow-up period of COVID-19 cases was 108 days (median: 101 days), which could provide insight into the effects of the disease on recipients in the medium to long term. Serious forced expiratory volume in one-second (FEV1) declines was observed in two patients after infection. One was in our follow-up for approximately 8 months after infection and was accepted as CLAD stage 4 because of the 60% decrease in FEV1 values compared to the basal best value.²⁰ However, the other patient was not followed up for a sufficient time for CLAD assessment; hence CLAD classification was not made for him; however, a 30% reduction compared to the best FEV1 value was found in PFT performed in the second month of COVID-19. These two cases

may indicate the importance of COVID-19-induced morbidity in lung transplant recipients in the mid-long term.

The main limitation of our study is that it was single-centered and covered few cases. Due to the long follow-up period after COVID-19, it may lighten the medium- to long-term effects of the disease.

In conclusion, the course and long-term effects of COVID-19 on SOT recipients remain unclear. Our small group of transplant recipients with COVID-19 consisted of two cases with loss of pulmonary function and one case with a re-infection, suggesting that steroid therapy should be initiated in the early period in patients with pulmonary opacities. Although no patient died among our patients, moderate-to-severe COVID-19 with pulmonary signs may cause loss of graft function in lung transplant recipients.

ACKNOWLEDGMENTS

We acknowledge all the health-care professionals in our lung transplant clinic.

CONFLICT OF INTEREST

The authors declare that they have no competing interests.

DATA SHARING AND DATA ACCESSIBILITY

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

AUTHOR CONTRIBUTIONS

All the authors contributed equally to the review of the existing literature, drafting the article, and approval of the final document.

ORCID

Sinan Turkkán  <https://orcid.org/0000-0003-4029-419X>

Muhammet Ali Beyoglu  <https://orcid.org/0000-0003-4038-630X>

Mehmet Furkan Sahin  <https://orcid.org/0000-0002-7652-624X>

Alkın Yazicioglu  <https://orcid.org/0000-0001-5651-0569>

Yasemin Tezer Tekce  <https://orcid.org/0000-0002-7054-6186>

REFERENCES

1. WHO. <https://covid19.who.int/>. Accessed March 21, 2021.
2. Alfishawy M, Elbendary A, Mohamed M, et al. COVID-19 mortality in transplant recipients. *Int J Organ Transplant Med*. 2020;11(4):145-162.
3. Geleris J, Sun Y, Platt J, et al. Observational study of hydroxychloroquine in hospitalized patients with covid-19. *N Engl J Med*. 2020;382(25):2411-2418.
4. Gautret P, Lagier JC, Parola P, et al. Clinical and microbiological effect of a combination of hydroxychloroquine and azithromycin in 80 COVID-19 patients with at least a six-day follow-up: a pilot observational study. *Travel Med Infect Dis*. 2020;34:101663.
5. Morlacchi LC, Rossetti V, Gigli L, et al. COVID-19 in lung transplant recipients: a case series from Milan. *Italy Transpl Infect Dis*. 2020;22:e13356.
6. Fishman JA. Infection in organ transplantation. *Am J Transplant*. 2017;17(4):856-879.
7. Mehta P, McAuley DF, Brown M, et al. COVID-19: consider cytokine storm syndromes and immunosuppression. *Lancet*. 2020;395(10229):1033-1034.

8. Walker CS. What we know about covid-19 reinfection so far. *BMJ*. 2021;372:n99.
9. West J, Everden S, Nikitas N. A case of COVID-19 reinfection in the UK. *Clin Med (Lond)*. 2021;21(1):e52-e53.
10. Zhou F-, Yu T, Du R, et al. Clinical course and risk factors for mortality of adult inpatients with COVID19 in Wuhan, China: a retrospective cohort study. *Lancet*. 2020;395(10229):1054-1062.
11. Pereira MR, Mohan S, Cohen DJ, et al. COVID-19 in solid organ transplant recipients: initial report from the US epicenter. *Am J Transplant*. 2020;20(7):1800-1808.
12. Aversa M, Benvenuto L, Anderson M, et al. COVID-19 in lung transplant recipients: a single center case series from New York City. *Am J Transplant*. 2020;20:3072-3080.
13. Romanelli A, Mascolo S. Immunosuppression drug-related and clinical manifestation of Coronavirus disease 2019: a therapeutical analysis. *Am J Transpl*. 2020;20:1947-1948.
14. Guillen E, Pineiro GJ, Revuelta I, et al. Case report of COVID-19 in a kidney transplant recipient: does immunosuppression alter the clinical presentation? *Am J Transplant*. 2020;20:1875-1878.
15. Zhu L-, Xu X, Ma KE, et al. Successful recovery of COVID-19 pneumonia in a renal transplant recipient with long-term immunosuppression. *Am J Transplant*. 2020;20:1859-1863.
16. Aslam S, Mehra MR. COVID-19: yet another coronavirus challenge in transplantation. *J Heart Lung Transplant*. 2020;39(5):408-409.
17. Heron VC, Bach C-AT, Holmes NE, et al. Complete recovery from COVID-19 of a kidney-pancreas transplant recipient: potential benefit from everolimus? *BMJ Case Rep*. 2021;14:e238413.
18. Arentz M, Yim E, Klaff L, et al. Characteristics and outcomes of 21 critically ill patients with COVID-19 in Washington State. *JAMA*. 2020;323(16):1612-1614.
19. Fernandez-Cruz A, Ruiz-Antoran B, Munoz-Gomez A, et al. A retrospective controlled cohort study of the impact of glucocorticoid treatment in SARS-CoV-2 Infection mortality. *Antimicrob Agents Chemother*. 2020;64(9):e01168-20.
20. Verleden GM, Glanville AR, Lease ED, et al. Chronic lung allograft dysfunction: definition, diagnostic criteria, and approaches to treatment—a consensus report from the Pulmonary Council of the ISHLT. *J Heart Lung Transplant*. 2019;38(5):493-503.

How to cite this article: Turkkkan S, Beyoglu MA, Sahin MF, Yazicioglu A, Tezer Tekce Y, Yekeler E. COVID-19 In Lung transplant recipients: A single-center experience. *Transpl Infect Dis*. 2021;23:e13700. <https://doi.org/10.1111/tid.13700>