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Outcomes of COVID-19 in solid organ transplant recipients: A matched cohort study

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Abstract

Whether solid organ transplant (SOT) recipients are at increased risk of poor outcomes due to COVID-19 in comparison to the general population remains uncertain. In this study, we compared outcomes of SOT recipients and non-SOT patients hospitalized with COVID-19 in a propensity score matched analysis based on age, race, ethnicity, BMI, diabetes, and hypertension. After propensity matching, 117 SOT recipients and 350 non-SOT patients were evaluated. The median age of SOT recipients was 61 years, with a median time from transplant of 5.68 years. The most common transplanted organs were kidney (48%), followed by lung (21%), heart (19%), and liver (10%). Overall, SOT recipients were more likely to receive COVID-19 specific therapies and to require ICU admission. However, mortality (23.08% in SOT recipients vs. 23.14% in controls, $P = .21$) and highest level of supplemental oxygen ($P = .32$) required during hospitalization did not significantly differ between groups. In this propensity matched cohort study, SOT recipients hospitalized with COVID-19 had similar overall outcomes as non-SOT recipients, suggesting that chronic immunosuppression may not be an independent risk factor for poor outcomes in COVID-19.

KEYWORDS

COVID-19, viral infections, transplant

Abbreviations: BIPAP, Bilevel Positive Airway Pressure; COVID-19, coronavirus disease; CKD, chronic kidney disease; CRP, C-reactive protein; DM, diabetes mellitus; HTN, hypertension; ICU, intensive care unit; PCR, polymerase chain reaction; SARS-CoV-2, severe acute respiratory syndrome coronavirus 2.

1 | INTRODUCTION

As the COVID-19 pandemic continues unabated, its impact on solid organ transplant (SOT) candidates and recipients has been profound, manifesting itself not only in morbidity and mortality but also adversely impacting organ procurement and transplant rates in many centers.[1-4] Given the high rates of infection-related complications in this immunocompromised population, there was an initial concern that SOT recipients infected with SARS-CoV-2 would be at higher risk for severe disease and death.[5] This concern was further compounded by the fact that many important risk factors for severe COVID-19 identified early in the pandemic, such as advanced age, diabetes, hypertension, and chronic kidney disease are disproportionally present among SOT recipients and candidates.[6] As a result, the Centers for Disease Prevention and Control (CDC) included SOT recipients among those at increased risk for severe illness from SARS-CoV-2 infection and a multitude of initial reports described high rates of mortality among SOT recipients.[7-17]

The emerging association between a hyperinflammatory state and severe COVID-19, however, has also raised the possibility that some chronically immunosuppressed patients could be protected from these deleterious effects. High-dose corticosteroids have thus far emerged as the only treatment shown to decrease mortality in randomized trials.[18] Subsequently, small series of hospitalized SOT recipients with variable control groups have suggested that outcomes may be similar to those in the general population.[19-23] Here, we report on a propensity matched cohort study among SOT recipients and non-SOT patients hospitalized with COVID-19 to assess the impact of transplant status on clinical outcomes.

2 | METHODS

2.1 | Patients

All adult (age >18 years) patients, including SOT recipients and non-transplant recipients, from Columbia University Irving Medical Center and hospitalized with COVID-19 between March 10, 2020, and May 30, 2020, were included in the analysis. Diagnosis of COVID-19 was based on a positive RT-PCR for SARS-CoV-2 from a nasopharyngeal swab. One hundred and five of these SOT recipients were included in two previous reports from our center, which did not include the propensity matched analysis in this manuscript.[9,24] All patients were followed up to 28 days or were censored at time of death or discharge. Data were extracted from ICD-10 codes and electronic medical record review. This study was approved by the Columbia University institutional review board.

2.2 | Therapeutic approach

The overall therapeutic approach for SOT recipients and non-SOT recipients in this period was the same. Investigational agents, including remdesivir, in either clinical trials or the expanded access program were considered in all patients with COVID-19 at our center. Early in the pandemic, off label hydroxychloroquine with or without azithromycin was also considered. Beginning April 10, 2020, intravenous methylprednisolone 1 mg/kg/d for at least 5 days was also given to those patients with severe COVID-19, defined as having an oxygen saturation below 94% on ambient air or requiring supplemental oxygenation. One or more doses of intravenous tocilizumab 4-8 mg/kg (maximum 800 mg) were also considered for patients with at least 7 days of symptoms, progressive respiratory distress, and rising levels of inflammatory markers such as C-reactive protein, ferritin, or interleukin-6 (IL-6). Among SOT recipients, baseline immunosuppression was moderately decreased upon diagnosis, with a particular emphasis on decreasing or discontinuing antimetabolite drugs such as mycophenolate or azathioprine.

2.3 | Analytic approach

All SOT recipients hospitalized and RT-PCR positive for SARS-CoV-2 were included as cases. We used the nearest-neighbor propensity-score matching method in this analysis. The propensity-score model includes age categories (below 55, between 55 and 70, and above 70 years of age), gender (M/F), BMI (above or below 30 kg/m^2), race (white, black, and other), ethnicity (Hispanic and non-Hispanic), hypertension, and diabetes. For each case, three control patients were matched except for one case, who matched only two controls samples. The covariates in propensity score models were presented before and after propensity score matching in Table 1.

Mean and standard deviation, number, and percent were used to summarize clinical characteristics and outcomes including death and hospital discharge. Chi-square and two-sample *t* test were used to compare categorical or continuous variables between these groups. Inpatient mortality was analyzed by Kaplan-Meier survival analysis. Finally, outcomes on intubation and hospital discharge were also summarized. All analyses were conducted by using SAS statistical software (version 9.4, SAS Institute, Cary, NC).

3 | RESULTS

3.1 | Patient characteristics

During the study period, 2,714 patients were hospitalized with SARS-CoV-2 infection, including 117 SOT recipients. Patient demographics for each group before and after propensity matching are displayed in Table 1. Prior to matching, SOT recipients were younger (median age 61 years vs. 66 years, P <.01), more likely to be male (65% vs. 54%, P = .02), and white (39.3% vs. 23.5%, P < .01), but less likely to have Hispanic ethnicity (36.4% vs. 51.2%, P < .01). SOT recipients were also more likely to have hypertension (89.7% vs. 60.7%, P < .01) and diabetes (70.1% vs. 39.4%, P < .001), but less

likely to have admission BMI > 30 kg/m^2 (21.4% vs. 36.7%, $P < .001$) compared to the non-transplant population.

Due to these differences in major COVID-19 risk factors, patients in the control group were subsequently selected for propensity score matching based on age categories (<55, 55-70, and >70 years of age), gender, race, ethnicity, BMI (above or below 30 kg/m^2), diabetes, and hypertension. A total of 350 patients were selected for the matched control group, for a 3:1 ratio comparison with the SOT recipients. After matching, there were no significant differences in any of these demographic and medical morbidities between the SOT recipient and non-transplant groups (Table 1).

3.2 | Transplant-related characteristics

The transplant-related characteristics of the 117 SOT recipients included in this cohort are displayed in Table 2. The overall median age in this cohort was 61 years (IQR 50-69). There were 56 kidney, 22 heart, 12 liver, 25 lung, and 2 pancreas transplant recipients. The median time from transplant of SARS-CoV-2 diagnosis was 5.68 years (IQR 1.93-10.02). Baseline immunosuppression data are displayed in Table 2.

3.3 | COVID-19 clinical course, disease severity, and mortality

Laboratory values, antiviral treatment, and disease severity are displayed in Table 3. There were no significant differences in peak white blood cell count, liver enzymes, total bilirubin, or nadir albumin levels between groups. Both initial and peak serum creatinine levels were significantly higher among SOT recipients compared to controls ($P < .01$). While some inflammatory markers including initial and peak CRP, procalcitonin, D-Dimer, and IL-6 were not significantly different, both initial and peak ferritin were significantly higher among SOT recipients compared to controls ($P < .01$ and 0.09, respectively).

SOT recipients were significantly more likely to receive COVID-19 treatments including hydroxychloroquine, remdesivir, tocilizumab, and high-dose corticosteroids (all $P < .01$, Table 3). In terms of disease severity, SOT recipients were more likely to have been admitted to the intensive care unit (32.5% vs. 27.7%, $P = .04$) although highest level of oxygen support was not different between the groups. There were no differences in median length of stay between the control and SOT groups either before (5.84 days, [IQR 2.88, 11.87] vs. 8.87 [IQR 4.811-16.8], respectively) or after propensity matching (6.67 days, [IQR 3.12, 13.13] vs. 8.87 [IQR 4.811-16.8], respectively).

Overall, there was no difference in 28-day survival probability between the SOT recipients (77.9%, 95%CI 69.4-86.4%) and propensity matched controls (71%, 95%CI 64.4-76.7%), $P = .168$ (Figure 1). In addition, there was no significant difference in 28-day mortality between different solid organ transplant types ($P = .69$).

4 | DISCUSSION

Understanding patient characteristics associated with poor outcomes related to COVID-19 remains a high priority, particularly in distinct groups such as those on chronic immunosuppression. Among SOT recipients, there is ongoing debate as to whether

TABLE 1 Baseline characteristics and 28-day mortality of SOT recipients and control group before and after propensity score matching

	Before propensity score matching			After propensity score matching		
	Control (N = 2597)	Case (N = 117)	*P* value	Control (N = 350)	Case (N = 117)	*P* value
Age (years)						
<55	742 (28.57%)	46 (39.32%)	<.01	131 (37.43%)	46 (39.32%)	.93
55 to 70	840 (32.35%)	49 (41.88%)		151 (43.14%)	49 (41.88%)	
>70	1,015 (39.08%)	22 (18.80%)		68 (19.43%)	22 (18.80%)	
Male	1,406 (54.14%)	76 (64.96%)	.02	237 (67.71%)	76 (64.96%)	.58
Race						
White	609 (23.45%)	46 (39.32%)	<.01	120 (34.29%)	46 (39.32%)	.58
Black	541 (20.83%)	27 (23.08%)		93 (26.57%)	27 (23.08%)	
Other	1,447 (55.72%)	44 (37.61%)		137 (39.14%)	44 (37.61%)	
Hispanic Ethnicity	1,329 (51.17%)	43 (36.44%)	<.01	130 (36.83%)	43 (36.44%)	.86
BMI						
<30	1,645 (63.34%)	92 (78.63%)	.08	271 (77.43%)	92 (78.63%)	.79
>30	952 (36.66%)	25 (21.37%)		79 (22.57%)	25 (21.37%)	
HTN	1,576 (60.69%)	105 (89.74%)	<.01	318 (90.86%)	105 (89.74%)	.72
Diabetes	1,022 (39.35%)	82 (70.09%)	<.01	247 (70.57%)	82 (70.09%)	.92
28-day mortality	628 (24.18%)	27 (23.08%)	.05	81 (23.14%)	27 (23.08%)	.41

TABLE 2 Transplant-related characteristics

	SOT recipients (n = 117)
Age, median (IQR)	61 (50-69)
Organ, n (%)	
Kidney	56 (48)
Pancreas ± kidney	2 (2)
Liver ± kidney	12 (10)
Heart ± kidney	22 (19)
Lung	25 (21)
Years from transplant to diagnosis, median (IQR)	5.68 (1.93-10.02)
Days of symptoms prior to Dx, median (IQR)	4 (2-7)
Chronic immunosuppression, n (%)	
CNI	105 (90)
Mycophenolate	94 (80)
Steroids	71 (61)
Belatacept	8 (7)
mTOR inhibitor	5 (4)

chronic immunosuppression represents an additional risk factor for poor outcomes in addition to advanced age, obesity, diabetes mellitus, and hypertension. In the present study, mortality among hospitalized SOT recipients was similar to that of a propensity matched cohort suggesting that the observed chronic immunosuppression did not portend a worse prognosis with COVID-19.

Although this finding is contradictory to the initial reports, which pointed to relatively increased morbidity and mortality in the SOT population, it is consistent with described outcomes in more recent analyses.[19,21-23,25] For example, a small non-matched case-control study of 35 SOT recipients compared to 100 non-transplant patients consecutively hospitalized patients showed similar mortality (23% vs. 25%, $P = .8$).[21] A large multicenter prospective registry of SOT recipients with COVID-19 also reached similar conclusions when an analysis of surrogates for immunosuppression intensity (recent vs. remote transplant, thoracic vs. non-thoracic transplant, recent augmented immunosuppression, and number of immunosuppressive agents) showed no association with mortality. As the authors acknowledged, however, cautious interpretation of these results was warranted due to various limitations including reliance on clinician reporting, confounding variables, and limited sample size.[19] Finally, a study using a large database of patients with severe COVID-19 admitted to intensive care units in several US hospital compared outcomes between 98 SOT recipients and 288 non-SOT patients via propensity score matched cohort and found no differences in 28-day mortality (40% vs. 43%, RR 0.92, 95% CI 0.7-1.22).[22] However, this analysis was limited to a very specific sub-population of critically ill patients and cannot therefore

TABLE 3 COVID-19 disease course including laboratory values, treatments, and disease severity

	Propensity matched controls (N = 350)	SOT-recipient cases (N = 117)	*P* value
Labs, median (IQR)			
WBC, ×1,000/μL			
Initial	7.59 (5.38-10.97)	5.58 (3.30-7.58)	<.01
Peak	11.48 (7.47-18.17)	10.14 (6.60-15.94)	.08
Creatinine, mg/dL			
Initial	1.25 (0.89-2.08)	2.02 (1.29-4.78)	<.01
Peak	1.59 (1.07-3.24)	3.19 (1.58-6.30)	<.01
AST, U/L			
Initial	40 (25-61)	31 (21-47)	.03
Peak	64 (35-118)	50.5 (32-93)	.04
ALT, U/L			
Initial	24 (17-43)	20 (13-30)	<.01
Peak	42 (22-83)	37 (25-85.5)	.85
Total bilirubin, mg/dL			
Initial	0.4 (0.3-0.6)	0.4 (0.3-0.6)	.06
Peak	0.6 (0.4-1.1)	0.6 (0.4-1)	.71
Albumin, g/dL			
Initial	3.70 (3.2-4)	3.7 (3.3-4)	.79
Nadir	2.9 (2.4-3.4)	3 (2.35-3.3)	.38
CRP, mg/L			
Initial	110.32 (47.78-198.46)	88.68 (41.93-140.92)	.04
Peak	176.17 (74.39-288.73)	148.78 (70.46-223.47)	.1
Ferritin, ng/mL			
Initial	715.10 (357.6-1411)	1001 (495.45-2066.5)	.01
Peak	989.9 (455.6-2395)	1568 (729.05-2741.5)	.01
Procalcitonin, ng/mL			
Initial	0.28 (0.12-0.90)	0.30 (0.15-0.82)	.4
Peak	0.64 (0.16-3.38)	0.63 (0.21-2.55)	.74
D-Dimer, ug/mL			
Initial	1.66 (0.93-3.47)	1.53 (0.79-3.06)	.17
Peak	3 (1.27-9.34)	2.62 (1.30-10.7)	.68
IL-6, pg/mL			
Initial	21 (8-50.8)	17.73 (8.3-48.75)	.65
Peak	36.25 (11.65-110)	49.88 (15.35-132)	.28

(Continues)

TABLE 3 (Continued)

	Propensity matched controls (N = 350)	SOT-recipient cases (N = 117)	*P* value
Therapy			
Hydroxychloroquine	176 (50.29%)	80 (68.38%)	<.01
Remdesivir	8 (2.29%)	9 (7.69%)	<.01
Tocilizumab	13 (3.71%)	26 (22.22%)	<.01
High dose steroids	74 (21.14%)	35 (29.91%)	<.01
Disease severity			
ICU admission	97 (27.71%)	38 (32.48%)	.01
Initial level of supplemental O_2[a]			
Ambient Air	208 (62%)	62 (66.67%)	.29
Nasal cannula	72 (21.49%)	24 (25.81%)	
Non-rebreather mask	43 (12.84%)	6 (6.45%)	
High flow	1 (0.3%)	0 (0%)	
Mechanical ventilation	11 (3.28%)	1 (1.08%)	
Highest level of supplemental O_2[a]			
Ambient Air	51 (15.22%)	18 (19.35%)	.31
Nasal cannula	108 (32.24%)	35 (37.63%)	
Non-rebreather mask	80 (23.88%)	14 (15.05%)	
High flow	12 (3.58%)	5 (5.38%)	
Mechanical ventilation	84 (25.07%)	21 (22.58%)	
Outcomes by day 28			
Death	81 (23.14%)	27 (23.08%)	.41
Hospital discharge	257 (73.43%)	82 (70.09%)	
Still intubated	6 (1.71%)	3 (2.56%)	
Still hospitalized (not intubated)	6 (1.71%)	5 (4.27%)	

[a]335 control and 93 SOT patients.

address whether SOT recipients are more likely to have severe disease as defined by requirement of intensive care unit admission or mechanical ventilation. As such, this current study offers a broader picture of the overall comparative risks associated with COVID-19 in SOT recipients in a larger population and adds to the current literature. Nevertheless, it should be noted that nearly all of these studies, including our cohort, are mostly limited to hospitalized individuals. While our analysis demonstrates comparable outcomes for hospitalized SOT recipients with other hospitalized patients, it is unable to determine if chronically immunosuppressed patients with COVID-19 are more likely to require hospitalization or are hospitalized earlier in their clinical course—factors that might confound interpretation of the overall risk for immunosuppressed patients.[14]

As extensively described by now, SARS-CoV-2 infection seems to trigger a dysregulated hyperinflammatory cascade that is associated with high mortality in a small but significant proportion of individuals. Termed COVID-19 associated hyperinflammation (COVID-HI), the exact mechanism remains undefined but appears to overlap with several known inflammatory syndromes such as cytokine release syndrome (CRS), macrophage activation syndrome, hemophagocytic lymphohistiocytosis (HLH), and involving multiple pathways including T cell and macrophage activation as well as complement fixation.[26,27] As shown by the reduction in mortality with dexamethasone, dampening that cascade seems to prevent the more severe complications of COVID-19. Similarly, the chronic immunosuppression that SOT recipients experience may facilitate a similar effect, possibly negating the consequences of potential increased susceptibility that results from the very same immunosuppression. As further analyses clarify more specific inflammatory mechanisms in COVID-19, a more targeted approach in the management of immunosuppression would be feasible, potentially leading to improved outcomes. However, the impact of specific immunosuppressive mechanisms and the degree of overall immunosuppression remain unknown.

There are limitations in this study, including those inherent to retrospective cohort analyses. While this study includes a larger number of SOT recipient than those reported to date, including granular data and controls that are well matched on a large number of clinically important features, the number of SOT recipients included may not have led to enough sensitivity to detect relatively small differences in survival rates. The majority of the patients in this cohort developed COVID-19 early in the pandemic course, a period of limited interventions and testing, and thus a comparison with more recent outcomes and different treatment strategies (including the use of monoclonal antibodies) may not be fully achievable or relevant. Although the incidence of rejection after COVID-19 is a complication of particular interest, we were not able to capture that data for the purposes of this study. Other studies, however, have reported a low incidence of rejection so far, although some subclinical rejection episodes may have been missed.[19] Finally, it was not possible for this analysis to control for the strong likelihood that the SOT recipients in this cohort benefited from greater access to medical care, including potentially lower thresholds for hospitalization and receiving more aggressive therapies than the general population. However, many of the COVID-19 specific therapies received by the SOT patients during the early phases of the pandemic were subsequently found to have limited or any positive impact on outcomes,[24] and the similar rates of initial and highest level of oxygen support suggest that both groups had comparable COVID-19 related disease severity even during admission.

In summary, when matched for several well-established risk factors, SOT recipients hospitalized with COVID-19 had comparable mortality to the non-SOT patients. Further studies are needed

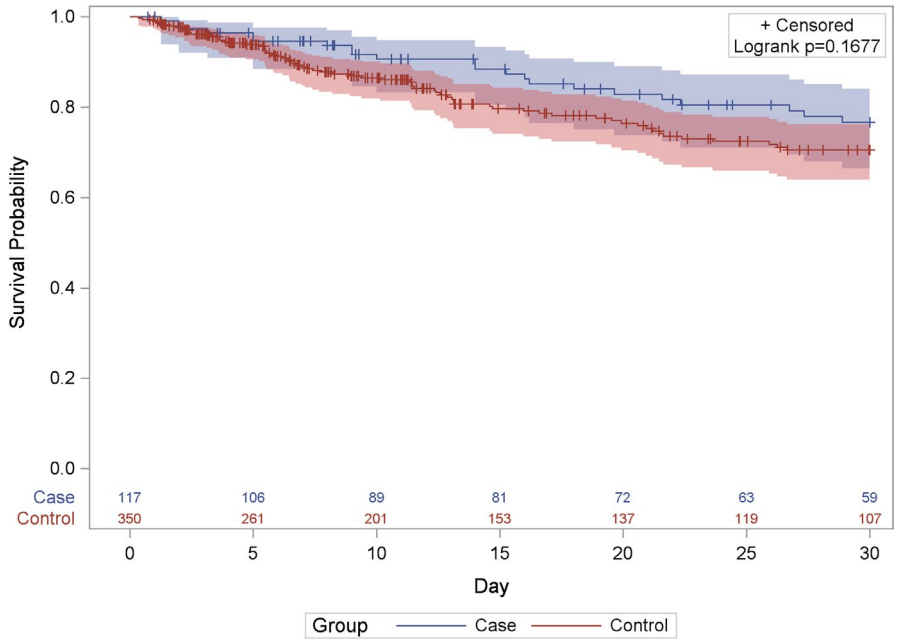

FIGURE 1 Twenty-eight-day survival probability in solid organ transplant recipients and matched controls

to assess specific transplant related modulators of COVID-19 disease.

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CONFLICT OF INTEREST

The authors declare no conflict of interest.

AUTHOR CONTRIBUTIONS

MRP, JZ, ECV: Contributed in conceptualization, data collection, data analysis and manuscript writing. QS: Contributed in data analysis and manuscript editing. DST, LP: Contributed in data collection and manuscript editing. ACU, SM: Contributed in conceptualization, data analysis and manuscript editing. SA, MAF, JCE: Contributed in conceptualization and manuscript editing. All authors approved the latest version of the manuscript.

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