

Clinical Profile, Immunosuppressive Management and Outcome in Adult Renal Allograft Recipients with COVID-19 Infection

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Introduction. Coronavirus disease 2019 (COVID-19) has placed considerable strain on healthcare systems worldwide with renal allograft recipients (RARs) representing a particularly vulnerable group. Lifelong immunosuppression increases susceptibility to severe infection and complicates clinical management. Data on COVID-19 in RARs from South Asia and other resource-limited settings are limited. This study describes the clinical features, immunosuppressive adjustments, and outcomes of renal allograft recipients with COVID-19.

Methods. We analyzed data from 26 adult RARs with moderate-to-severe COVID-19, confirmed by RT-PCR, who were admitted to the Department of Nephrology, Continental Hospital PLC (previous United Hospital Limited) Dhaka, Bangladesh, between December 2020 and June 2025.

Results. The mean age was 59.6 years (range: 34 to 75 years), and 73.1% of patients were male. DM (46.2%), glomerulonephritis (23.1%), and hypertensive nephrosclerosis (19.2%) were the most common primary renal diseases. The most frequent symptoms were cough (96.2%) and fever (88.5%). Immunosuppressive therapy was modified in all patients: antimetabolites were discontinued, corticosteroids were switched to dexamethasone or IV methylprednisolone, and CNIs were withheld in 34.6% or halved in 26.9%. Laboratory results showed elevated inflammatory markers (CRP 96.4 mg/L, ferritin 854.3 ng/mL, D-dimer 2016.3 ng/mL). Chest CT revealed ground-glass opacities in 76.9% (most commonly bilateral peripheral, 35%) and consolidation in 61.5%, with a mean total severity score of 9.6 ± 4.9 . Acute kidney injury occurred in 26.9%, among them 23.1% required dialysis and 7.7% remained dialysis-dependent. Mechanical ventilation was needed in 23.1%. Overall mortality was 26.9%, while 65.4% of patients recovered and were discharged.

Conclusion. COVID-19 is associated with significant morbidity and mortality in RARs. Despite most patients recovering, mortality remained significant, highlighting the need for careful immunosuppressive adjustment, aggressive infection control, and close monitoring in this high-risk population.

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INTRODUCTION

The novel severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), which causes coronavirus disease 2019 (COVID-19), has rapidly spread across the world, leading the World Health Organization (WHO) to declare a global pandemic in March 2020.¹ Kidney transplant recipients (KTRs) are among the most vulnerable to this pandemic, facing a dual threat: chronic immunosuppression necessary to prevent allograft rejection markedly increases susceptibility to severe infection, while the inflammatory cascade triggered by SARS-CoV-2 significantly affects both native and transplant kidney function.^{2,3}

Kidney transplant recipients experience substantially worse outcomes following COVID-19 infection than the general population. Systematic reviews and meta-analyses have consistently demonstrated that hospitalized kidney transplant recipients with COVID-19 face a mortality rate of 21 to 23%,^{4,5} which is markedly higher than the 1 to 5% typically reported in the general population.⁶ The risk of critical illness is equally concerning, with ICU admission reported in 24% and mechanical ventilation required in 18% of cases.⁴ Acute kidney injury occurs in up to 48 to 50% of infected transplant recipients, with 12 to 23% requiring renal replacement therapy.³⁻⁵ Several factors drive this vulnerability: older age, a heavy load of comorbidities such as diabetes and hypertension, and immunosuppressive medications that suppress the body's natural defenses against viruses.^{4,7}

The optimal management of immunosuppressive therapy in kidney transplant recipients with COVID-19 remains an area of considerable uncertainty and ongoing debate. While some evidence supports the reduction or discontinuation of antimetabolites (mycophenolate mofetil or azathioprine) to facilitate viral clearance, findings across different cohorts have been inconsistent.² Current expert recommendations suggest discontinuing mycophenolic acid or mTOR inhibitors in patients with moderate to severe disease, reducing calcineurin inhibitor (CNI) doses by approximately 30 to 50%, and maintaining or increasing corticosteroid therapy.² However, these strategies must be carefully balanced against the risk

of graft rejection, particularly in highly sensitized patients or those with recent transplantation.²

The clinical presentation of COVID-19 in kidney transplant recipients closely resembles that of the general population, with fever, cough, and dyspnea representing the most common symptoms.⁶ However, the progression to severe disease appears to be accelerated in this immunocompromised group, with acute respiratory distress syndrome developing in over half of hospitalized patients in pooled analyses.⁶ Radiological findings frequently reveal bilateral ground glass opacities (GGO) and consolidation, with disease severity correlating strongly with adverse outcomes. Laboratory abnormalities, including elevated inflammatory markers (CRP, ferritin, D-dimer), lymphopenia, and worsening renal function, serve as important prognostic indicators.⁷

The morbidity and mortality burden of COVID-19 in RARs extends beyond acute infection: 27 to 30% of hospitalized patients require ICU admission, and approximately 22% die, mainly from sepsis, ARDS, and multiorgan failure.³ Advanced age, diabetes mellitus, dyspnea at presentation, pneumonia, and AKI have been identified as independent predictors of mortality.^{7,8} Furthermore, survivors often experience persistent graft dysfunction, with a subset progressing to permanent dialysis dependence.³ The substantial case fatality ratio, coupled with the high rates of critical illness and renal complications, highlights the urgent need for optimized management strategies in this high-risk population.

Despite the growing body of evidence from high-income countries, data on COVID-19 in kidney transplant recipients from South Asia and other resource-limited settings remain scarce. Differences in healthcare infrastructure, immunosuppressive protocols, and patient demographics could shape outcomes in ways that studies from wealthier settings simply do not capture. This study describes the clinical features, immunosuppressive adjustments, and outcomes of adult renal allograft recipients with RT-PCR-confirmed COVID-19 admitted with moderate to severe disease at a tertiary care center in Dhaka, Bangladesh. By characterizing this vulnerable population in a resource-limited South Asian setting, we aim to inform clinical

management strategies and contribute to the global understanding of COVID-19 in kidney transplant recipients.

MATERIALS AND METHODS

We conducted this retrospective study at the Department of Nephrology, Continental Hospital PLC, (Previous United Hospital Limited), Dhaka, Bangladesh, between December 2020 and June 2025, including 26 adult RARs with COVID-19 infection. Patients were selected based on the following criteria:

Inclusion Criteria

- Adults (≥ 18 years) with a functioning renal allograft
- RT-PCR confirmed COVID-19
- Hospitalized with moderate to severe disease
- Complete clinical, laboratory, radiological, and outcome data available in medical records

Exclusion Criteria

- Age < 18 years
- Mild / asymptomatic cases managed as outpatients
- COVID-19 diagnosed without RT-PCR confirmation
- History of dialysis after transplantation (graft failure)
- History of acute graft rejection
- Incomplete medical record

Ethical Issue

This study was conducted using routinely collected clinical data without any interventional procedures. Ethical approval was obtained from the department head and ethics committee prior to data analysis. This study posed no additional risk to patients and did not interfere with routine patient management. All patient data were anonymized before analysis in accordance with the Declaration of Helsinki. Informed consent for treatment and the use of anonymized clinical data for research purposes was obtained from the patients or their families.

Data Collection Procedure

This retrospective study included 26 adult renal

transplant recipients with RT-PCR confirmed COVID-19 at a single tertiary center in Bangladesh. Demographic, clinical, laboratory, radiological, treatment, and outcome data were collected. Clinical data included symptoms, vital signs, oxygen requirements, and the need for renal replacement therapy. Laboratory and radiological evaluations assessed organ function and inflammatory status, including chest CT findings and Total Severity Scores (TSS).

Data Analysis

All collected data were entered into Microsoft Excel and analyzed using SPSS version 25. Continuous variables were expressed as mean $\pm$ standard deviation (SD), and categorical variables were summarized using frequencies and percentages.

RESULTS

In this study of 26 Renal Allograft Recipients (RARs) with COVID-19, demographic characteristics, initial oxygen saturation, immunosuppressive modifications, and overall outcomes are summarized in Table 1.

The baseline characteristics of the 26 kidney transplant recipients are summarized in Table 2. The mean age was 59.6 years (range: 34 to 75), with a male predominance (73%). The median time since transplantation was 85.7 months. Diabetes mellitus (46%), glomerulonephritis (23%), and hypertensive nephrosclerosis (19%) were the most common primary renal diseases. Common presenting symptoms included cough (96%), fever (88%), and dyspnea (46%).

Common presenting symptoms included cough (96%), fever (88%), and dyspnea (46%).

Immunosuppression was modified in all patients: antimetabolites were discontinued, steroids were switched to low-dose dexamethasone (73%) or IV methylprednisolone (27%), and calcineurin inhibitors were stopped (35%), reduced by half (27%), or continued at the same dose (38%). All patients received LMWH, 88% received remdesivir and additional therapies included antibiotics, favipiravir (12%), rivaroxaban (42%), and tocilizumab (8%).

Biochemical parameters, and radiological features

Table 1. All Patients Baseline Data (n = 26)

No	Age/Sex (year / Male or Female)	Duration Of Transplant (months)	Primary Disease	Comorbidities	O ₂ Saturation During Admission (%)	Immunosuppression Modification	Management Of COVID-19 Pneumonia	Overall Outcome
1	52 / F	76	ADPKD	HTN, DM	97	Complete Cessation of MMF, Steroid switched to low-dose dexamethasone, Continued CNI	LMWH, Antibiotics Remdesivir, Rivaroxaban	Discharged
2	34 / M	118	IgA Nephropathy	NODAT, CAN HTN	96	Complete Cessation of MMF, Steroid switched to Low-Dose dexamethasone, 50% Dose Reduction of CNI	LMWH, Antibiotics Remdesivir Rivaroxaban	Discharged
3	54 / M	48	DM	HTN Post PCI State CAN	89	Complete Cessation of MMF, Steroid switched to Low-Dose Dexamethasone, Complete Cessation of CNI (Tacrolimus)	LMWH, Antibiotics Remdesivir	Death due to Sepsis, MOF & NSTEMI
4	55 / M	76	DM	HTN IHD	84	Complete Cessation of MMF, Steroid switched to IV Methylprednisolone 50% Dose Reduction of CNI	LMWH, Antibiotics Remdesivir Rivaroxaban	Discharged
5	62 / F	62	Chronic Pyelonephritis	HTN, DM, CAN	90	Complete Cessation of MMF, Steroid switched to Low-Dose Dexamethasone Complete Cessation CNI (Tac)	LMWH Antibiotics Remdesivir	Death due to Sepsis & MOF
6	70 / M	82	Hypertensive nephrosclerosis	DM, IHD COPD	85	Complete Cessation of MMF, Steroid switched to Steroid switched to IV Methylprednisolone , Complete Cessation of CNI (Tacrolimus)	LMWH Antibiotics Remdesivir	Death due to Sepsis, MOF & ARDS
7	43 / F	122	Presumed GN	HTN, NODAT Bronchial Asthma CAN	95	Complete Cessation of MMF, Steroid switched to low-dose dexamethasone, Complete Cessation of CNI (Tacrolimus)	LMWH Antibiotics Remdesivir Rivaroxaban	Discharged
8	55 / M	75	DM	HTN	85	Complete Cessation of MMF, Steroid switched to low-dose dexamethasone, Continued CNI (Tac)	LMWH Remdesivir Tocilizumab	Discharged
9	75 / M	129	DM	HTN ICM, COPD	89	Complete Cessation of MMF, Steroid switched to Low-Dose Dexamethasone, 50% Dose Reduction of CNI	LMWH Antibiotics Remdesivir Apixaban	Discharged
10	55 / M	38	Presumed GN	HTN, DM	96	Complete Cessation of MMF, Steroid switched to low-dose dexamethasone, Continued CNI (Tac)	LMWH Antibiotics Favipiravir	Discharged

Table 1. Continued

No	Age/Sex (year / Male or Female)	Duration Of Transplant (months)	Primary Disease	Comorbidities	O ₂ Saturation During Admission (%)	Immunosuppression Modification	Management Of COVID-19 Pneumonia	Overall Outcome
11	48 / F	119	FSGS	HTN, NODAT CAN	90	Complete Cessation of MMF, Steroid switched to low-dose dexamethasone, Complete Cessation of CNI (Tacrolimus)	LMWH Antibiotics Remdesivir	Death due to Sepsis & MOF
12	75 / M	87	Presumed Gn	HTN, Morbid Obesity, Post PCTA State, Post- Transplant FSGS	83	Complete Cessation of MMF, Steroid switched to low-dose dexamethasone, Continued CNI (Tac)	Antibiotics LMWH Remdesivir Tocilizumab Rivaroxaban	Discharged
13	63 / M	52	DM	HTN, Post PCTA State	90	Complete Cessation of MMF, Steroid switched to low-dose dexamethasone, Continued CNI (Tac)	LMWH Antibiotics Remdesivir Rivaroxaban	Discharged
14	60 / M	134	DM	HTN,	95	Complete Cessation of MMF, Steroid switched to low-dose dexamethasone, Continued CNI (Tac)	LMWH Antibiotics Remdesivir Rivaroxaban	Discharged
15	66 / F	114	Hypertensive nephrosclerosis	NODAT Bronchial Asthma	98	Complete Cessation of MMF, Steroid switched to low-dose dexamethasone, Continued CNI (Tac)	LMWH Antibiotics Favipiravir	Discharged
16	55 / M	79	DM	HTN, CABG	88	Complete Cessation of MMF, Steroid switched to Steroid switched to IV Methylprednisolone, Complete Cessation of CNI	LMWH Antibiotics Remdesivir	Death due to Sepsis, ARDS, Secondary Bacterial Infection
17	51 / M	23	Hypertensive Nephrosclerosis	DM Bronchial Asthma	98	Complete Cessation of Azathioprine, Steroid switched to low-dose dexamethasone, Continued CNI (Tac)	LMWH Antibiotics Favipiravir	Discharged
18	71 / M	115	Obstructive Nephropathy	HTN,DM	97	Complete Cessation of MMF, Steroid switched to low-dose dexamethasone, 50% Dose Reduction of CNI	LMWH Antibiotics Remdesivir	Death due to Cardiogenic Shock
19	70 / M	95	DM	HTN, Permanent Pacemaker	98	Complete Cessation of MMF, Steroid switched to low-dose dexamethasone, Complete Cessation of CNI (Tacrolimus)	LMWH Antibiotics Remdesivir Rivaroxaban	Discharged
20	61 / M	118	DM	HTN, CABG, COPD	90	Complete Cessation of MMF, Steroid switched to low-dose dexamethasone, Continued CNI (Tac)	LMWH Antibiotics Remdesivir	Discharged

Table 1. Continued

No	Age/Sex (year / Male or Female)	Duration Of Transplant (months)	Primary Disease	Comorbidities	O ₂ Saturation During Admission (%)	Immunosuppression Modification	Management Of COVID-19 Pneumonia	Overall Outcome
21	69 / M	84	Hypertensive Nephrosclerosis	NODAT	88	Complete Cessation of MMF, Steroid switched to Steroid switched to IV Methylprednisolone low-dose dexamethasone, Complete Cessation of CNI (Tacrolimus)	LMWH Antibiotics Remdesivir Rivaroxaban	Discharged
22	57 / F	29	DM	DM	92	Complete Cessation of MMF, Steroid switched to low-dose dexamethasone, 50% Dose Reduction of CNI	LMWH Remdesivir Rivaroxaban	Discharged
23	74 / M	104	DM	HTN, COPD	85	Complete Cessation of MMF, Steroid switched to Steroid switched to IV Methylprednisolone low-dose dexamethasone 50% Dose Reduction of CNI	LMWH Antibiotics Remdesivir	Death due to Neutropenic Sepsis, Type 2 Respiratory failure, ARDS
24	53 / M	106	Hypertensive Nephrosclerosis	NODAT, Bronchial Asthma	89	Complete Cessation of MMF, Steroid switched to Steroid switched to IV Methylprednisolone, 50% Dose Reduction of CNI	LMWH Antibiotics Remdesivir	Discharged
25	71 / M	89	DM	HTN,	78	Complete Cessation Of MMF, Steroid switched to Steroid switched to IV Methylprednisolone , Complete Cessation of CNI (Tacrolimus)	LMWH Antibiotics Remdesivir	Discharged
26	51 / F	27	Lupus Nephritis	HTN, NODAT	91	Complete Cessation of MMF, Steroid switched to low-dose dexamethasone, Continued CNI (Tac)	LMWH Antibiotics Remdesivir Rivaroxaban	Discharged

shown in Table 3. Inflammatory markers were elevated (CRP = 96.4 mg/L, procalcitonin = 3.1 ng/mL, ferritin = 854 ng/mL, D-dimer = 2016 ng/mL). Chest CT most commonly showed ground-glass opacities (77%), with bilateral peripheral distribution (35%) and consolidation (62%), often with pleural effusion (38%). Six patients (23%) had normal CT findings. The mean total severity score was 9.6 ± 4.9 .

Respiratory profile shown in Table 4. The mean

oxygen saturation was 92%, with respiratory support ranging from room air (27%) to low-flow nasal cannula (23%), high-flow nasal cannula (19%), Bi-PAP (8%), and mechanical ventilation (23%). Preexisting renal disease included chronic allograft nephropathy (CAN) (19%) and recurrent UTIs (12%), with a mean serum creatinine of 1.64 mg/dL.

AKI occurred in 27% of patients, 23% of patients required renal replacement therapy, and 8% remained dialysis-dependent. Overall mortality

Table 2. Baseline Characteristics of Renal Allograft Recipients

Baseline characteristics	(n = 26)	(%)
Mean age, y	59.62 ± 10.48 (34 to 75)	
Gender		
Male	19	73.08
Female	7	26.92
Mean time interval between kidney transplantation (mo)	85.69 ± 40.12 (23 to 134)	
Primary Disease	-	-
DM	12	46.15
Glomerulonephritis	6	23.07
IgA nephropathy	1	3.85
FSGS	1	3.85
Lupus nephritis	1	3.85
Presumed Glomerulonephritis (GN)	3	11.54
Hypertensive Nephrosclerosis	5	19.23
ADPKD	1	3.85
Obstructive Nephropathy	1	3.85
Chronic pyelonephritis	1	3.85
Renal abnormality	-	-
Chronic allograft Nephropathy (CAN)	5	19.23
Post Transplant FSGS	1	3.85
Clinical Features	-	-
Cough	25	96.15
Fever	23	88.46
Myalgia	15	57.69
Dyspnea	12	46.15
Sore throat	15	57.69
Diarrhea	3	11.54
Immunosuppression modification	-	-
Complete cessation of MMF	25	96.15
Complete cessation of Azathioprine	1	3.85
Steroid switched to low-dose Dexamethasone	19	73.07
Steroid switched to IV Methylprednisolone	7	26.9
Complete cessation of CNI (Tacrolimus)	9	34.62
Continued CNI (Tacrolimus)	10	38.49
50% dose reduction of CNI (Tac)	7	26.9
Management of COVID-19 pneumonia	-	-
LMWH	26	100.00
Antibiotics	21	80.77
Remdesivir	23	88.46
Rivaroxaban	11	42.31
Apixaban	1	3.85
Tocilizumab	2	7.69
Favipiravir	3	11.54

Abbreviations: CAN, chronic allograft nephropathy, NODAT; new-onset diabetes after transplantation; COPD, chronic obstructive pulmonary disease, CABG; coronary artery bypass grafting, LMWH; low molecular weight heparin, MMF; mycophenolate mofetil, CNI; calcineurin inhibitors

Table 3. Laboratory, X-ray, and CT Findings of Study Patients at Baseline (n = 26)

Laboratory Findings	Mean ± SD	
Hemoglobin, g/dL	11.92 ± 1.56	
Total count of WBCs, /μL	14307.69 ± 7195.37	
Neutrophil (%)	77.58 ± 18.00	
CRP, mg/L	96.38 ± 86.66	
Procalcitonin, ng/mL	3.10 ± 2.47	
Serum Ferritin, ng/mL	854.31 ± 357.07	
Serum Creatinine, mg/dL	1.66 ± 0.68	
Serum D-Dimer, ng/mL	2016.25 ± 1286.86	
X-ray findings		
Normal	7	26.92
Consolidation (unilateral/bilateral)	11	42.31
Consolidation and pleural effusion	4	15.38
Bilateral patchy opacity	4	15.38
CT findings		
Ground Glass Opacity (GGO)	20	76.92
Bilateral peripheral	7	35.0
Bilateral peripheral + subpleural GGO	3	15.0
Diffuse bilateral GGO	5	25.0
Unilateral GGO (left / right)	3	15.0
Central GGO	2	10.0
Reverse halo sign	2	10.0
Intralobular septal thickening	3	11.54
Consolidation	16	61.54
Bilateral consolidation	3	18.8
Unilateral consolidation (left/right)	3	18.8
Bilateral basal consolidation	3	18.8
Consolidation with pleural effusion	6	37.5
Subpleural bands	3	18.8
Normal CT scan	6	23.08
Mean Total severity score	9.56 ± 4.88	

was 27%, primarily due to sepsis with ARDS, multi-organ failure, cardiogenic shock, or type 2 respiratory failure.

DISCUSSION

The COVID-19 pandemic has imposed a heavy burden on RARs, who face a higher risk of severe infection, AKI, and death due to their impaired immune systems and common comorbidities like DM, HTN, and ischemic heart disease.^{4,5,7} This study evaluated the clinical features, management, renal outcomes, and mortality of RARs hospitalized with COVID-19 pneumonia.

Demographics

Our study showed a relatively older age with a male predominance, a demographic pattern consistent with international reports of COVID-19

Table 4. Respiratory Parameters and Renal Outcome Variables in Renal Allograft Recipients (n = 26)

Respiratory Parameters	n	P (%)
Mean Oxygen saturation level (%)	92.12 ± 5.32	
Respiratory support required		
Room air	7	26.92
Low-flow nasal cannula	6	23.08
High flow nasal cannula	5	19.23
BiPAP (Bilevel Positive Airway Pressure)	2	7.69
Mechanical ventilation	6	23.08
Renal outcome parameters		
Urine routine examination		
NAD	15	57.69
Protein trace	4	15.39
Protein +	3	11.54
Protein ++	3	11.54
Leucocyte ++	1	3.85
UTI	4	15.39
Preexisting renal abnormality		
Nothing significant	19	73.08
CAN	5	19.23
Recurrent UTI	3	11.54
Serum creatinine at admission, mg/dL	1.66 ± 0.68	
Serum creatinine after 7 days, mg/dL	3.35 ± 2.64	
Renal outcome		
Acute Kidney Injury (AKI)	7	26.92
AKI requiring renal replacement therapy	6	23.08
Dialysis dependent	2	7.69
Remain same	17	65.38
Urinary Tract Infection (UTI)	3	11.54
Overall outcome		
Discharged (recovered)	17	65.38
Discharged on maintenance hemodialysis (MHD) 3/week	2	7.69
Death	7	26.92
Death due to sepsis with multiorgan failure (MOF)	1	14.29
Death due to septic shock	1	14.29
Death due to sepsis with ARDS	3	42.86
Death due to cardiogenic shock	1	14.29
Death due to type 2 respiratory failure with ARDS and pneumonia	1	14.29

in KTRs. Crespo *et al.* described an older Spanish cohort with a mean age of 73.6 years and 75% male predominance, while Akalin *et al.* and Alberici *et al.* reported younger cohorts with male predominance ranging from 65 to 80%.⁹⁻¹¹ Importantly, Nowak *et al.* demonstrated that advanced age and male remain independent predictors of severe disease and mortality among KTRs with COVID-19, even in the post-vaccination era, supporting our findings.¹² A large multinational study by the Asian Society of Transplantation Registry (ASTREGO) reported

outcomes of 87 kidney transplant recipients (KTRs) with COVID-19 across seven Asian countries. The relatively young cohort (median age = 43 years) mostly had mild-to-moderate disease, yet overall mortality was 23%, with higher deaths among those needing intensive care. Disease severity at presentation independently predicted mortality, highlighting persistently high COVID-19 mortality in Asian KTRs, especially when healthcare resources are limited.¹³

The interval between transplantation and COVID-19 diagnosis in our cohort was longer than that reported by Crespo *et al.* (49 months) and Santeusano *et al.* (69.6 months), but comparable to the Asian cohorts reported by Kee *et al.* and the ASTREGO registry, suggesting that COVID-19 risk persists well beyond the early post-transplant period.^{9,13,14} Geethanjali *et al.*, similarly reported that severe COVID-19 can occur at any stage posttransplant, highlighting the ongoing vulnerability of long term KTRs in the region.¹⁵

Primary Renal Disease and Comorbidities

DM, glomerulonephritis, and hypertensive nephrosclerosis were the most common primary renal diseases, while hypertension, DM, and IHD were the most common comorbidities. These findings are consistent with prior reports indicating that metabolic and cardiovascular comorbidities substantially increase the risk of severe COVID-19 and mortality in KTRs.^{7,13,15} Specifically, DM and HTN have been consistently associated with higher rates of hospitalization, ICU admission, and death among KTRs with COVID-19. Recent multicenter analyses further confirmed that diabetes and cardiovascular disease are independent risk factors for adverse outcomes in this population.^{4,7}

Clinical Features

Fever and cough were the most frequent presenting symptoms among our patients, followed by dyspnea, while gastrointestinal symptoms were relatively uncommon. This clinical profile closely consistent with the findings reported by Akalin *et al.*, Jasuja *et al.*, and Jha *et al.*, indicating that the initial presentation of COVID-19 in KTRs is similar to that of the general population, although progression to severe disease is more frequent.^{10,16,17}

Radiological Features and Respiratory Severity

The Chest radiography and CT findings in our cohort were dominated by consolidation and ground- glass opacities (GGO), predominantly with bilateral peripheral distribution, indicating moderate pulmonary involvement. A meaningful proportion of patients required mechanical ventilation, while others required high-flow nasal cannula or noninvasive ventilation. Similar associations between extensive radiological involvement, ventilatory support requirements, and mortality have been reported by Demir *et al.*, and Nowak *et al.*, highlighting the prognostic significance of respiratory severity in KTRs.^{12,18.}

Immunosuppressive Modification and COVID-19 Treatment

All patients underwent modifications of immunosuppressive therapy, including discontinuation of antimetabolites. Maintenance corticosteroids were adjusted to alternative regimens according to the disease severity. Patients with mild-to-moderate oxygen requirements received low-dose dexamethasone, consistent with the RECOVERY trial demonstrating a mortality benefit in hospitalized patients requiring oxygen support.¹⁹ In patients with progressive COVID-19 pneumonia or worsening inflammatory markers, IV methylprednisolone was administered to suppress the hyperinflammatory response. This approach is supported by transplant cohort experiences, suggesting that short-course IV methylprednisolone may improve respiratory outcomes without significantly increasing the risk of acute rejection when accompanied by appropriate immunosuppressive adjustments.^{9,10}

Calcineurin inhibitors (CNIs) were continued, dose reduced, or temporarily withheld depending on illness severity and drug–drug interaction concerns. This management strategy aligns with international experience and expert recommendations. Studies by Akalin *et al.*, Crespo *et al.*, and Demir *et al.* emphasized early withdrawal of antimetabolites and individualized CNI modification as central to COVID-19 management in kidney transplant recipients.^{9,10,18} Importantly, Nowak *et al.* reported that temporary reduction of immunosuppression during acute infection was not associated with an increased risk of acute rejection.¹²

Supportive therapy included universal anticoagulation with low molecular weight heparin, and the most patients received remdesivir. Remdesivir was well tolerated, with no nephrotoxicity observed. Consistent with multicenter data, its use in KTRs has not been associated with an increased risk of AKI and may reduce ICU mortality when administered early.²⁰ Although experience with oral antivirals such as nirmatrelvir / ritonavir in transplant recipients remains limited, emerging evidence supports their feasibility with careful management of drug–drug interactions, particularly involving CNIs.²¹

A small number of patients with progressive hypoxemia and worsening inflammatory markers despite corticosteroids received tocilizumab, an IL-6 receptor antagonist, to attenuate the COVID-19 associated cytokine storm.²² This is supported by the RECOVERY trial, which showed that tocilizumab improved 28-day survival in hospitalized COVID-19 patients with hypoxia and systemic inflammation when added to standard care.²³ Data specific to KTRs are limited but reassuring on safety: a multicenter cohort of 80 KTRs treated with tocilizumab reported no drug-related safety issues, though mortality remained high given the severity of illness in this subgroup.²² Given this mixed evidence, tocilizumab was reserved for select patients with severe, progressive disease unresponsive to corticosteroids alone.

Incidence of Acute Kidney Injury and Need for Dialysis

A notable proportion of our cohort developed AKI, of whom a majority required renal replacement therapy, and a smaller subset remained dialysis-dependent at last follow-up, indicating significant graft injury within a portion of the recipients. Chronic allograft dysfunction was present in a subset of patients, several of whom subsequently developed AKI during the course of the illness. This finding suggests that pre-existing graft dysfunction may predispose RARs to worsening graft function during acute viral illness.

AKI is more common and severe in RARs than in the general population. While AKI occurs in 10 to 15% of hospitalized COVID-19 patients in the general population, its incidence in RARs

is markedly higher, ranging from 30 to 57% in individual studies, with pooled analyses around 40 to 44%.^{5,24,25}

Our observed AKI rate was lower than that reported by Geethanjali *et al.* who documented a markedly higher incidence of AKI among KTRs with COVID-19. Their study emphasized the influence of disease severity and modifications in immunosuppressive therapy on graft outcomes. The discrepancy between their findings and ours may be attributed to differences in patient characteristics, severity of infection, sample size, and management protocols.¹⁵

Overall, our results reinforce the existing evidence that COVID-19 carries a significant threat to graft function, particularly in recipients with underlying CAN. Close monitoring of renal and inflammatory parameters and individualized adjustment of immunosuppressive therapy are essential to minimize the risk of irreversible graft dysfunction.

Mortality

The overall mortality rate in our study, with most deaths attributable to sepsis complicated by ARDS, multi-organ failure, and cardiogenic shock, was consistent with previously reported mortality rates of 20 to 30% among hospitalized KTRs.^{10,18,20} Moreover, recent systematic reviews and meta-analyses have confirmed that transplant recipients experience significantly higher mortality rates than the general hospitalized population.^{4,5} The increased vulnerability reflects the combined effects of chronic immunosuppression, a high burden of comorbidities such as diabetes and cardiovascular disease, and impaired antiviral immune responses.

Data from the ERACODA (European Renal Association COVID-19 Database) collaboration demonstrated higher rates of ICU admission, mechanical ventilation, and secondary infections among transplant recipients with COVID-19.²⁰ In a large multicenter cohort study of 250 kidney transplant recipients from India, Kute and colleagues reported substantial mortality and identified AKI as an important predictor of poor outcome.²⁶ Similarly, Bajpai *et al.* observed high mortality rates in patients with severe disease,

particularly among those requiring intensive care support.²⁷ Nowak *et al.* and Geethanjali *et al.* identified advanced age, DM, cardiovascular disease, severe lung involvement, and AKI as independent predictors of mortality, with AKI being particularly significant due to its association with systemic inflammation, hemodynamic instability and poor outcomes.^{12,15}

Taken together, these findings reinforce that KTRs remain a high-risk population despite advances in supportive care and antiviral therapy, underscoring the importance of early risk stratification and individualized management strategies to improve survival.

Clinical Implications

This study highlights the persistent vulnerability of RARs to severe COVID-19. Early modification of immunosuppression, prompt anti-viral therapy, close monitoring of clinical, biochemical, respiratory, and renal parameters, and aggressive supportive care are essential to improving outcomes. High-risk patients particularly the elderly and those with DM, cardiovascular disease, or preexisting CAN require early hospitalization and a multidisciplinary approach involving nephrology, infectious disease, and critical care teams. Individualized treatment strategies that balance infection control with graft preservation remain fundamental to optimizing outcomes in this vulnerable population.

Strengths and Limitations

A Strength of this study is the detailed clinical and laboratory characterization of RARs during the pandemic in a tertiary center. However, the relatively small sample size and single-center design limit generalizability. The retrospective nature may also introduce bias in treatment uniformity and data completeness.

CONCLUSIONS

Renal transplant recipients are highly vulnerable to adverse outcomes following COVID-19 infection, with clinical severity ranging from mild symptoms to severe pneumonia and multi-organ failure. Prompt adjustment of immunosuppressive therapy, along with supportive care and antiviral therapy, is critical for preserving graft function

and improving outcomes. Despite a high rate of recovery, mortality remains significant, highlighting the need for intensive monitoring, individualized management. Further multicentric prospective studies are warranted to establish standardized treatment protocols for this high-risk population.

DECLARATION

Author Contributions

Sonia Mahjabin. Conceptualization, study design, data collection, supervision, and overall management of the study. Corresponding author responsible for manuscript preparation and submission.

Nurul Islam. Clinical supervision, data interpretation and critical review of the manuscript.

Rezwanur Rahman. Clinical supervision, data interpretation, presentation of results, and critical review of the manuscript.

Elena Haque. Data collection, clinical management of patients and review of the manuscript.

Muntasir Ebne Mobin. Results review, formal analysis and presentation of results.

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Conflict of Interest

The authors declare no conflicts of interest.

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