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P.S. and R.P. are equal contributors and participated in research design, writing of the article, data analysis and final editing. N.K. participated in data collection and editing the article. A.P. participated in data collection and editing the article. S.Y. participated by data collection and data analysis. T.K. participated by data collection and data analysis. S.N. participated in research design, writing of the paper and data analysis and M.S. participated in research design and writing of the article.

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Abstract:
Renal involvement in severe or critical acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection is frequent. Acute kidney injury (AKI) in African American (AA) kidney transplant recipients (KTRs) with COVID-19 is not well described. We report our experience with a predominantly AA cohort (79%) of KTRs with COVID-19 infections in the Detroit Metropolitan area.

Methods:
In this retrospective, single-center study, we identified 39 KTRs who tested positive for SARS-CoV-2 between March 16 and April 25th, 2020. Data from electronic medical records were retrieved and compared between KTRs without AKI and KTRs with AKI.

Results:
One pt was excluded due to DGF. Final analysis of AKI in KTRs with proven COVID-19 was done on 38 patients of which 30 were AA (79%). AKI occurred in 71.1% of COVID-19 KTRs (n=27), of whom 6 (22.2%) patients required HD. The incidence of AKI in our cohort was 71% (27/38). AKI rate among AA was 76.7% vs. 50% in non-AA cohort (p=0.195). In a univariate logistic regression analysis, AA race was not significantly associated with AKI OR (3.4, CI [0.68-17.4], p=0.14). After risk adjustment by race, patients with diabetes showed a significantly higher risk of AKI (adjusted OR 19.85 CI [1.65-58.66], p=0.012). KTRs with AKI had more preexisting RAAS inhibitor use than KTRs without AKI (P=0.03).

Conclusions:
KTRs infected with SARS-CoV-2 have a high incidence of AKI, with associated increased morbidity and mortality. Though no racial differences in mortality were noted in our KTRs with AKI, we await data from registries to help elucidate this difference.
Introduction

Renal involvement in severe or critical acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection is relatively frequent.\(^{1-4}\) In the published literature of Coronavirus Disease 2019 (COVID-19), AKI is associated with increased mortality.\(^{1,2}\) KTX recipients (KTRs) appear to be at high risk for severe COVID-19 due to coexisting conditions and, presumably, chronic immunosuppression. The incidence of AKI in KTRs with COVID-19 is still being defined, and reported incidence has been variable (30-57%) depending on patient demographics and AKI definition.\(^{5-11}\) Also, it is not known if race increases the risk or worsens the prognosis of AKI in KTRs with Covid-19. We report hereby our experience in a cohort of predominantly African American (AA) KTRs with COVID-19 presenting with and without AKI.

Materials and Methods

In this retrospective, single-center study, we identified 50 consecutive KTRs who tested positive for SARS-CoV-2 between March 16 and April 25th, 2020. The study was approved by the Henry Ford Health System Investigation Review Board (IRB) with waiver for consent. Data from electronic medical records were retrieved for demographic, epidemiologic, clinical, laboratory, and radiologic characteristics; and hemodialysis (HD) treatment and renal outcomes. These were compared between KTRs without AKI and KTRs with AKI. Further comparison was done between KTRs with AKI with and without HD. Serial laboratory values were tracked during hospitalization; at admission and days 1, 3, 5, 7, 14 and 21.

Treatment guidelines for COVID treatment were followed per Henry Ford Hospital policy outlined by a COVID-19 team, which was led by infectious disease. The initial management included providing supportive care, which was escalated, under the guidance of the primary team, as clinically indicated. Immunosuppressants were managed by the transplant team. General approach
included reduction of the IS, with antimetabolite agent being the first to be reduced or completed discontinued followed by CNI as dictated by patient condition. COVID-19 positive KTRs were treated with hydroxychloroquine (HCQ) 400 mg twice a day for 24 hours followed by 200 mg twice a day for 4 days, if admitted and QTc less than 500 msec. A course of methyl prednisolone 1mg/kg twice a day was prescribed for 3-7 days, under the guidance of transplant ID. AKI treatment include fluid resuscitation, maintenance of hemodynamic stability with use of fluids or vasopressors as indicated. None of the patient received Remdesivir. Those with persistent fever, ARDS, elevated inflammatory markers, or high levels of IL-6 received TOCI.

**Definitions:**

AKI and staging of AKI was based on the Kidney Disease: Improving Global Outcomes (KDIGO) criteria. The recovery of AKI was defined as the return of kidney function to baseline. Hypoxia was defined as the need for supplemental oxygen to maintain SaO2 >94%. Shock was defined as the need for vasopressors. Cardiac injury was defined as an elevation in cardiac biomarker (ie, high sensitivity troponin) above the 99th percentile of upper reference limit, or new abnormalities shown in electrocardiography and/or echocardiography.

**Statistical Analyses:**

Categorical variables were described as frequency rates and percentages, and continuous variables were described using mean, median, and interquartile range (IQR) values. Rate comparisons were performed by the chi-squared test or Fisher’s exact test. Kruskal–Wallis test was used to compare median across groups according to the number of groups and distribution of the variable. Risk factors for AKI were analyzed using a logistic regression model. Variables that were significantly associated with AKI on univariate analysis were analyzed with patient race in a bivariate model.
Statistical significance was defined at p-value <0.05. All statistical analyses were performed using SPSS software version 26.

Results

One patient was excluded due to delayed graft function (DGF). The final analysis of AKI in KTRs with proven COVID-19 was done on 38 patients of which 30 were AA (79%). AKI occurred in 71.1% of COVID-19 KTRs (n=27), out of which 6 (22.2%) patients required HD. Clinical and demographic characteristics of kidney transplant patients with and without AKI are described in Table 1.

A total of 23 recipients (60.5%) were male, the median age was 61.5 years (range, 27-89), median posttransplant time at infection was 5.9 years (range, 0.6-34 years), 3 patients were under 1 year from transplantation, and 3 recipients (7.9%) were Hispanic. There was no statistically significant difference between the demographic characteristics of the groups. In our cohort, 25 recipients (65.8%) had a deceased donor KTX; 36 (94.7%) had hypertension, 21 (55.3%) had diabetes mellitus, and 7 (18.4%) had documented heart disease. Diabetes mellitus was noted to be more prevalent in KTRs with AKI as compared to those without AKI (p=0.005), (Table 1). After risk adjustment by race, patients with diabetes showed a significantly higher risk of AKI (adjusted odds ratio 9.85, CI 1.65-58.66, P=0.012) (Table 2). Concerning immunosuppression, 34 patients (89.5%) were receiving tacrolimus, 28 (73.7%) were receiving prednisone, and 26 (68.4%) were receiving mycophenolate-based immunosuppression at admission.

Out of the 27 KTRs, AKI was seen in 76.7% of AA (n=23) vs 50% non-AA (N=4); and in 55.3% of subjects (n=21), AKI was present at hospital admission. Prior to hospitalization, median estimated glomerular filtration rate (eGFR) was 52ml/min/1.73m². In 60.5% of subjects (n=23), baseline eGFR was < 60 ml/min per 1.73 m². KTRs with AKI had more preexisting RAAS (Renin
angiotensin aldosterone system) inhibitor use than KTRs without AKI (P=0.03). Concerning staging of AKI, 18 KTRs had stage 1, 2 had stage 2, and 8 had stage 3 at presentation. Predominance of AA race was shown among the KTRs with AKI requiring HD (Table 3). On univariate analysis, AA race was not associated with AKI (OR 3.4 [CI 0.68-17.4], P=0.14). Graft loss due to death occurred in 30.4% (7 of 23) AA KTRs with AKI. Overall, 43.5% (10 of 23) of AAs regained function, completely (n=8, 34.8%) or partially (n=2, 8.7%) during the follow-up period. In this predominantly AA cohort of KTRs with proven COVID-19 illness, the overall death rate was 23.6% (9 of 38), in AA 23.3% (7 of 30) and others 25% (2 of 8). Patients who developed AKI saw an increase in death rate to 33.3% (9 of 27); whereas in patients with AKI requiring HD, the death rate increased further to 66.7% (4 of 6).

Regarding COVID-19 markers, 29 KTRs (76.3%) had lymphopenia at admission; and testing for inflammatory markers showed a proinflammatory milieu. Ferritin level ≥900 ng/mL was documented in 47.4% of KTRs (n=18), C-reactive protein level ≥5 mg/dL in 39.5% (n=15), 14 subjects (36.8%) had procalcitonin level ≥0.2 ng/mL, and 25 (65.8%) had D-dimer level ≥0.5 μg/mL. For comparison of laboratory parameters, patients were divided into 3 groups, non-AKI, AKI with HD and AKI without HD. It showed statistically significant difference in the 3 groups in neutrophil to lymphocyte ratio (p=0.02) at 14 days; absolute lymphocyte count (p=0.04) at day 14; CRP (p=0.03) at day 3 and (p=0.02) at day 14; hemoglobin (p=0.008) at day 1, (p=0.01) at day 14, (p=0.05) at day 21. (Figure 1). KTRs with AKI, more frequently had bilateral pneumonic infiltrates (p<0.001), developed ARDS (p<0.001), and needed mechanical ventilation (p<0.001), (Table 4). A higher incidence of complications such as cardiac injury (p=0.05), anemia (p=0.016), shock (p<0.001), and need for ICU admission (p<0.001) was also noted in the 3 groups. (Table 4,
supplementary material). Mortality rate was 0% in patient without AKI, 28.3% in pts with AKI and increased to 66.7% in pts with dialysis requiring AKI (p=0.008).

The mainstay of therapy was the discontinuation of the antimetabolite. Patients requiring inpatient non-ICU care were treated with HCQ. Patients requiring supplemental oxygen or those requiring ICU for ventilator-dependent respiratory failure received SM along with HCQ. Of these, a small proportion received HCQ with AZI, with careful monitoring of the QTc interval. It was discontinued in 2 patients due to prolonged QTc. 3 patients with persistent fever, ARDS, elevated inflammatory markers, or high levels of IL-6 received TOCI.

**Discussion**

The incidence and significance of AKI in KTRs with COVID-19 illness either from direct kidney injury or multiorgan failure (MOF) is not well understood. In this report, we present our experience in the diagnosis and management of AKI in AA KTRs with COVID-19. The State of Michigan had the 3rd largest incidence of COVID-19 and death of all states of the Union at the time our study was started. As the majority of COVID-19 cases congregated in Wayne County and the City of Detroit, our study has a predominance of AA KTRs. This high proportion of AA in our cohort makes our study unique compared to the published literature to date. The statistics of the state of Michigan document that, in the general population, AA have a higher incidence of COVID-19-related admissions and death than other racial groups. In the United States and the United Kingdom, COVID-19 is more virulent and lethal in black subjects. Our study did not identify race to be associated with risk of AKI, (OR 3.4 [CI 0.68-17.4], P=0.14). The small sample size and lack of diversity in our study population may be contributing factors to the lack of observed effect. In data reported so far, only a small percentage of studies report the racial background of
affected patients.\textsuperscript{15} To get a clearer picture of racial disparities, we need detailed data reported by race and ethnic group nationally.

We have shown that the incidence of AKI in KTRs is higher than that reported in the general population with COVID-19 (71\% Vs 2.5-29\%).\textsuperscript{1,2,3} Compared to reports of AKI in Caucasian KTRs, the incidence of AKI in AA KTRs is also higher (30\% Vs 76.6\%).\textsuperscript{5} Although AA race was not an independent predictor of AKI in our cohort, we speculate that the higher prevalence of diabetes in AA may be the reason for the high incidence of AKI in these patients. Recent data suggests that DM is associated with more severe disease and increased mortality in COVID 19 illness.\textsuperscript{16,17} Poor outcomes in patients with DM may be attributed to the chronic systemic inflammation prompted by the elevated circulating level of IL-6 that accompanies diabetes and the metabolic syndrome, and its complex trans-signaling pathway.\textsuperscript{18} Majority of KTRs with AKI exhibited a high burden of disease and proinflammatory milieu, and more frequently required advanced life-sustaining treatment than KTRs without AKI. AA KTRs predominated in the group requiring ICU care and life-support.

KTRs with AKI had more preexisting RAAS inhibitor use than KTRs without AKI (P=0.03). Other studies\textsuperscript{4-8} did not substantiate this risk but inability to autoregulate blood flow in the kidney may explain this higher risk. ACE2 receptor is involved in viral entry into the cell, and some have postulated RAAS inhibitors may potentiate this effect.\textsuperscript{19} Though, Menon et al show that ACE2 levels were not altered by exposures to renin angiotensin aldosterone system (RAAS) inhibitors.\textsuperscript{20} Identifying the pathophysiology of AKI in KTRs with COVID-19 is beyond the scope of this study. Nevertheless, several potential mechanisms have been published. Diabetics with chronic kidney disease who develop COVID-19 may be at a higher risk of AKI through an upregulation of ACE2mRNA expression, which may aggravate the proinflammatory and profibrotic
environment in the kidneys.\textsuperscript{20} The presence of viral particles morphologically identical to SARS-CoV-2 in renal tissue provides evidence of the virus capability for direct renal infection and tubular injury.\textsuperscript{21} Whether AA and, thereby, AA KTRs are predisposed genetically to a more severe COVID-19 phenotype\textsuperscript{22,23} or the socioeconomic and cultural determinants are accountable for the excess risk observed in our cohort merits further investigation. Given the small size of our cohort and the lack of racial diversity, proper identification of race-related risk factors was not possible.

Conclusions

Our study shows that AA KTRs infected with SARS-CoV-2 and present with high incidence of AKI, with associated increased morbidity and mortality. The high incidence of AKI in our cohort might reflect the overall illness and comorbidities of the population we serve.
References


Figure Legends

**Figure 1:** The serial laboratory trends in the patients with KTRs without AKI [AKI (0)], with AKI without dialysis need [AKI (+) HD (-)], and with AKI needing hemodialysis [AKI (+) HD (+)]. Kruskal-Wallis 1 way analysis of variance showed statistically significant difference in groups in Neutrophil to lymphocyte ratio (p=0.02) at 14 days; Absolute Lymphocyte count (p=0.04) at day 14; CRP (p=0.03) at day 3 and (p=0.02) at day 14; Hemoglobin (p=0.008) at day 1, (p=0.01) at day 14, (p=0.05) at day 21. Abbreviations: ALC, Absolute lymphocyte count; AKI, Acute kidney injury; CRP, C reactive protein; GFR, Glomerular filtration rate; Hb, hemoglobin; HD, hemodialysis; KTRs, Kidney transplant recipient; NLR, Neutrophil to Lymphocyte ratio.
Table 1. Characteristics of kidney transplant patients with and without AKI

<table>
<thead>
<tr>
<th></th>
<th>KTRs without AKI (N=11)</th>
<th>KTRs with AKI (N=27)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Demographics</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Median Age (IQR) – years</td>
<td>54 [44.5-72.5]</td>
<td>62 [54.5-69]</td>
<td>0.412</td>
</tr>
<tr>
<td>Male (%)</td>
<td>7 (77.8)</td>
<td>12 (57.1)</td>
<td>0.419</td>
</tr>
<tr>
<td>African American (%)</td>
<td>7 (63.6)</td>
<td>23 (85.2)</td>
<td>0.284</td>
</tr>
<tr>
<td>Deceased donor kidney transplant (%)</td>
<td>6 (60.0)</td>
<td>17 (65.4)</td>
<td>0.889</td>
</tr>
<tr>
<td><strong>Coexisting conditions</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HTN (%)</td>
<td>9 (81.8)</td>
<td>27 (100.0)</td>
<td>0.078</td>
</tr>
<tr>
<td>DM (%)</td>
<td>2 (18.2)</td>
<td>19 (70.4)</td>
<td>0.005</td>
</tr>
<tr>
<td>CAD (%)</td>
<td>2 (18.2)</td>
<td>5 (19.2)</td>
<td>1</td>
</tr>
<tr>
<td>RAAS inhibitor use (%)</td>
<td>1 (9.1)</td>
<td>13 (48.1)</td>
<td>0.03</td>
</tr>
<tr>
<td><strong>Presenting symptoms and signs</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cough (%)</td>
<td>6 (54.5)</td>
<td>23 (85.2)</td>
<td>0.088</td>
</tr>
<tr>
<td>Fever (%)</td>
<td>6 (54.5)</td>
<td>17 (63.0)</td>
<td>0.722</td>
</tr>
<tr>
<td>Diarrhea (%)</td>
<td>4 (36.4)</td>
<td>13 (48.1)</td>
<td>0.721</td>
</tr>
<tr>
<td>Arrythmia (%)</td>
<td>0 (0.0)</td>
<td>6 (22.2)</td>
<td>0.154</td>
</tr>
<tr>
<td>AMS (%)</td>
<td>0 (0.0)</td>
<td>4 (14.8)</td>
<td>0.303</td>
</tr>
<tr>
<td>Headache (%)</td>
<td>2 (18.2)</td>
<td>2 (7.4)</td>
<td>0.564</td>
</tr>
<tr>
<td>Hypoxia (%)</td>
<td>1 (9.1)</td>
<td>20 (74.1)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td><strong>Laboratory &amp; Imaging</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lymphopenia (%)</td>
<td>4 (36.4)</td>
<td>25 (92.6)</td>
<td>0.001</td>
</tr>
<tr>
<td>Anemia (%)</td>
<td>2 (18.2)</td>
<td>18 (66.7)</td>
<td>0.014</td>
</tr>
<tr>
<td>Bilateral infiltrates on CXR (%)</td>
<td>1 (9.1)</td>
<td>17 (63.0)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td><strong>Complications</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cardiac injury (%)</td>
<td>1 (9.1)</td>
<td>14 (51.9)</td>
<td>0.026</td>
</tr>
<tr>
<td>Shock (%)</td>
<td>0 (0.0)</td>
<td>8 (29.6)</td>
<td>0.077</td>
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<tr>
<td>ARDS (%)</td>
<td>0 (0.0)</td>
<td>12 (44.4)</td>
<td>0.008</td>
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<tr>
<td>Intubation (%)</td>
<td>0 (0.0)</td>
<td>9 (33.3)</td>
<td>0.038</td>
</tr>
<tr>
<td>ICU admission (%)</td>
<td>0 (0.0)</td>
<td>13 (48.1)</td>
<td>0.006</td>
</tr>
<tr>
<td>Death (%)</td>
<td>0 (0.0)</td>
<td>9 (33.3)</td>
<td>0.038</td>
</tr>
</tbody>
</table>

Table 1. The clinical characteristics of patients with and without acute kidney injury are summarized. Abbreviations: AKI, acute kidney injury; AMS, altered mental status; ARDS, acute respiratory distress syndrome; CXR, chest X ray; CAD coronary artery disease; DM, Diabetes; HTN, hypertension; ICU, intensive care unit; KTRs, kidney transplant recipient; RAAS, renin angiotensin aldosterone system.
Table 2. Bivariable analysis with recipient race for factors associated with AKI

<table>
<thead>
<tr>
<th>Race as AA (ref. non-AA)</th>
<th>OR (95% CI)</th>
<th>P value</th>
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<td></td>
<td>OR (95% CI)</td>
<td>P value</td>
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<tr>
<td>DM</td>
<td>9.85 (1.65-58.66)</td>
<td>0.012</td>
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<tr>
<td>RAAS inhibitor</td>
<td>8.31 (0.91-76.12)</td>
<td>0.061</td>
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</table>

Table 2. shows results of bivariate analysis after risk adjustment by race, for diabetes and RAAS inhibitor use.
Table 3. Describes the racial composition of patients with AKI and in different stages of AKI

<table>
<thead>
<tr>
<th>AKI staging</th>
<th>All N=38</th>
<th>African Americans N=30</th>
<th>Others N=8</th>
<th>p-Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>AKI group</td>
<td>27</td>
<td>23 (85.1%)</td>
<td>4 (14.8%)</td>
<td>0.195</td>
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<tr>
<td>Non-AKI Group</td>
<td>11</td>
<td>7 (63.6%)</td>
<td>4 (36.4%)</td>
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</table>

<table>
<thead>
<tr>
<th>Stages of AKI</th>
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<tbody>
<tr>
<td>Stage 1</td>
<td>18</td>
<td>16 (88.9%)</td>
<td>2 (11.1%)</td>
<td>0.136</td>
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<tr>
<td>Stage 2</td>
<td>2</td>
<td>2 (50%)</td>
<td>2 (50%)</td>
<td></td>
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<tr>
<td>Stage 3</td>
<td>8</td>
<td>6 (75%)</td>
<td>2 (25%)</td>
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<table>
<thead>
<tr>
<th>RRT</th>
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<tbody>
<tr>
<td>No RRT</td>
<td>21</td>
<td>19 (90.5%)</td>
<td>2 (9.5%)</td>
<td>0.204</td>
</tr>
<tr>
<td>RRT requiring</td>
<td>6</td>
<td>4 (66.7%)</td>
<td>2 (33.3%)</td>
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</tbody>
</table>

Abbreviations: AKI, acute kidney injury; RRT, renal replacement therapy.
<table>
<thead>
<tr>
<th>Demographics</th>
<th>KTRs without AKI (N=11)</th>
<th>KTRs with AKI not needing Dialysis (N=21)</th>
<th>KTRS with AKI needing HD (N=6)</th>
<th>p-value</th>
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</thead>
<tbody>
<tr>
<td>Median Age (IQR) - years</td>
<td>54 [44.5-72.5]</td>
<td>62 [55-69]</td>
<td>63.5 [49-74.5]</td>
<td>0.7</td>
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<tr>
<td>Male (%)</td>
<td>7 (77.8)</td>
<td>8 (53.3)</td>
<td>4 (66.7)</td>
<td>0.476</td>
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<tr>
<td>African American (%)</td>
<td>7 (63.6)</td>
<td>19 (90.5)</td>
<td>4 (66.7)</td>
<td>0.288</td>
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<tr>
<td>Deceased donor kidney transplant (%)</td>
<td>6 (60.0)</td>
<td>16 (76.1)</td>
<td>3 (33.3)</td>
<td>0.252</td>
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<tr>
<td>Coexisting conditions</td>
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<td></td>
</tr>
<tr>
<td>HTN (%)</td>
<td>9 (81.8)</td>
<td>21 (100.0)</td>
<td>6 (100.0)</td>
<td>0.075</td>
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<tr>
<td>DM (%)</td>
<td>2 (18.2)</td>
<td>15 (71.4)</td>
<td>4 (66.7)</td>
<td>0.013</td>
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<tr>
<td>CAD (%)</td>
<td>2 (18.2)</td>
<td>4 (20.0)</td>
<td>1 (16.7)</td>
<td>0.981</td>
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<tr>
<td>RAAS inhibitor use (%)</td>
<td>1 (9.1)</td>
<td>9 (42.9)</td>
<td>4 (66.7)</td>
<td>0.044</td>
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<td>Presenting symptoms and signs</td>
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<tr>
<td>Fever (%)</td>
<td>6 (54.5)</td>
<td>12 (57.1)</td>
<td>5 (83.3)</td>
<td>0.456</td>
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<tr>
<td>Cough (%)</td>
<td>6 (54.5)</td>
<td>17 (81.0)</td>
<td>6 (100.0)</td>
<td>0.082</td>
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<tr>
<td>Diarrhea (%)</td>
<td>4 (36.4)</td>
<td>11 (52.4)</td>
<td>2 (33.3)</td>
<td>0.721</td>
</tr>
<tr>
<td>Headache (%)</td>
<td>2 (18.2)</td>
<td>1 (4.8)</td>
<td>1 (16.7)</td>
<td>0.687</td>
</tr>
<tr>
<td>Arrhythmia (%)</td>
<td>0 (0)</td>
<td>5 (23.8)</td>
<td>1 (16.7)</td>
<td>0.214</td>
</tr>
<tr>
<td>AMS (%)</td>
<td>0 (0)</td>
<td>2 (9.5)</td>
<td>2 (33.3)</td>
<td>0.099</td>
</tr>
<tr>
<td>Hypoxia (%)</td>
<td>1 (9.1)</td>
<td>15 (71.4)</td>
<td>6 (100.0)</td>
<td>0.002</td>
</tr>
<tr>
<td>Laboratory &amp; Imaging</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lymphopenia (%)</td>
<td>4 (36.4)</td>
<td>19 (90.5)</td>
<td>6 (100.0)</td>
<td>0.001</td>
</tr>
<tr>
<td>Anemia (%)</td>
<td>2 (18.2)</td>
<td>12 (57.1)</td>
<td>6 (100.0)</td>
<td>0.016</td>
</tr>
<tr>
<td>Any Radiographic changes (%)</td>
<td>1 (9.1)</td>
<td>20 (95.2)</td>
<td>6 (100.0)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Bilateral infiltrates on CXR (%)</td>
<td>1 (9.1)</td>
<td>13 (61.9)</td>
<td>4 (66.7)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Complications</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cardiac injury (%)</td>
<td>1 (9.1)</td>
<td>11 (52.4)</td>
<td>3 (50.0)</td>
<td>0.05</td>
</tr>
<tr>
<td>Shock (%)</td>
<td>0 (0)</td>
<td>4 (19.0)</td>
<td>4 (66.7)</td>
<td>0.005</td>
</tr>
<tr>
<td>ARDS (%)</td>
<td>0 (0)</td>
<td>6 (28.6)</td>
<td>6 (100.0)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Intubation (%)</td>
<td>0 (0)</td>
<td>3 (14.3)</td>
<td>6 (100.0)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>ICU admission (%)</td>
<td>0 (0)</td>
<td>7 (33.3)</td>
<td>6 (100.0)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Death (%)</td>
<td>0 (0)</td>
<td>5 (23.8)</td>
<td>4 (66.7)</td>
<td>0.008</td>
</tr>
</tbody>
</table>

Table 4. The clinical characteristics of each group (without AKI, with AKI which was further divided into HD requiring group and non-HD requiring group) are summarized. Abbreviations: AKI, acute kidney injury; AMS, altered mental status; ARDS, acute respiratory distress syndrome; CXR, chest X ray; CAD coronary artery disease; DM, Diabetes; HTN, hypertension; ICU, intensive care unit; KTRs, kidney transplant recipient; RAAS, renin angiotensin aldosterone system
Figure 1: The serial laboratory trends in the patients with KTRs without AKI [AKI (0)], with AKI without dialysis need [AKI (+) HD (-)], and with AKI needing hemodialysis [AKI (+) HD (+)]. Kruskal-Wallis one way analysis of variance showed statistically significant difference in groups in Neutrophil to lymphocyte ratio (p=0.02) at 14 days; Absolute Lymphocyte count (p=0.04) at day 14; CRP (p=0.03) at day 3 and (p=0.02) at day 14; Hemoglobin (p=0.008) at day 1, (p=0.01) at day 14, (p=0.05) at day 21. Abbreviations: ALC, Absolute lymphocyte count; AKI, Acute kidney injury; CRP, C reactive protein; GFR, Glomerular filtration rate; Hb, hemoglobin; HD, hemodialysis; KTRs, Kidney transplant recipient; NLR, Neutrophil to Lymphocyte ratio.