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# Identifying a Kidney Transplant Recipient COVID Phenotype to Aid Test Utilization in the Setting of Limited Testing Availability—Does One Exist?

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## ABSTRACT

The high morbidity and mortality of COVID-19 in immunocompetent patients raises significant concern for immunosuppressed kidney transplant recipients (KTRs). This level of concern, both on the part of the KTRs and transplant professionals, is heightened by a lack of prior knowledge on how Severe Acute Respiratory Syndrome 2 virus (SARS-CoV-2) may manifest differently in immunosuppressed patients. Characterizing how KTRs may present differently than the general population would allow for more targeted and timely evaluation and treatment of KTRs with COVID-19 infection.

Methods. Without prior knowledge of how this virus would affect our transplant center's delivery of care to KTRs who are SARS-CoV-2 positive or patients under investigation, and in the setting of limited testing availability, we initiated a quality assurance and improvement project (QAPI) to track KTRs followed at our transplant center through the SARS-CoV-2 testing process.

Results. Of the 53 symptomatic patients, 20 (38%) tested positive for SARS-CoV-2 either on presentation to the emergency department or referral to a designated outpatient testing center. In addition, 16 (80%) of the 20 patients who tested positive required inpatient treatment. Intriguingly, patients with a history of polyoma BK viremia (BKV) had a higher incidence of testing positive for SARS-CoV-2 compared to patients without a history of BKV (80% and 28%, respectively; $P = .002$). The Positive Predictive Value and Likelihood ratio was 80% and 6.6 for this association, respectively. Among our KTRs tested, those receiving belatacept had a lower likelihood of testing positive for SARS-CoV-2. This finding approached, but did not achieve, statistical significance ($P = .06$).

THE landscape of the Severe Acute Respiratory Syndrome 2 virus (SARS-Cov-2) pandemic is constantly changing. Limited resources and swiftly evolving information are influencing real-time changes in our approach to triaging, evaluation, and management of patients suspected to be infected by this virus.

It is unclear if immunocompromised patients are at a higher risk for contracting this viral infection as compared to the general population. While it is true that other non-novel viruses tend to cause more severe disease in immunocompromised patients [1], no conclusive data are available to suggest an increased susceptibility or severity of SARS-Cov-2 infection in immunosuppressed kidney

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transplant recipients (KTRs). At the time of writing, isolated case reports from early days of the pandemic in the Wuhan district of China are yet to be comprehensively analyzed and validated.

Emerging claims of comorbid associations and effective therapeutics in the mainstream and social media lack consistent and conclusive evidence, raise uncertainty among health care professionals, and cause confusion among the general public. The initial knowledge of a syndromic presentation with fever and flu-like symptoms has now expanded to include other systemic symptoms such as vague malaise, body aches, and gastrointestinal symptoms, mimicking many common infectious diseases [2].

To use our transplant center's resources most efficiently during a pandemic with potentially scarce testing capability, we recognized that it might be necessary to limit the use of tests to those patients most likely to benefit from diagnostic testing. We started a quality assurance and improvement (QAPI) project to identify a patient phenotype that is associated with confirmed COVID-19 disease so that we may effectively prioritize patients for testing. Here we report our initial observations on the first 53 patients from our center tested for SARS-CoV-2.

## MATERIALS AND METHODS

This was a single center, retrospective chart review performed as a QAPI project to assess similarities in kidney transplant recipients who tested positive for SARS-CoV-2 as compared to those who tested negative and guide testing recommendations in the setting of limited testing availability during the early COVID-19 pandemic.

Data were collected for kidney or kidney-pancreas transplant recipients from March 10, 2020, through April 9, 2020. Patients were included if they were over the age of 18, were receiving transplant care at our center, and were tested for SARS-CoV-2 during this time period. Asymptomatic patients screened for SARS-CoV-2 as a prerequisite for placement in a skilled nursing facility were excluded.

All variables were collected from the electronic medical record. The $\chi^2$ test was used to compare categorical data, and a Student $t$ test was used to compare continuous data. A $P$ value of $< .05$ was considered to be statistically significant.

## RESULTS

All of the 53 patients included in this cohort were tested for SARS-CoV-2. Individuals who remained patients under investigation were excluded from the analysis. Overall, the average age was 59.5 years for SARS-CoV-2 positive patients and 56.7 years for patients who tested negative, slightly older than the average (55.4 years old) KTR followed at our center (Table 1). We did not observe any significant association between patient sex, level of education, or history of diabetes on the SARS-CoV-2 test result.

A cough was the most common symptom, followed by fever and shortness of breath (Table 2). None of these symptoms, individually, had a statistically significant association with a positive test result. Patients presenting with only 1 symptom (53% of our total cohort) were more likely to test negative for SARS-CoV-2. However, presenting with more than 1 symptom (fever, cough, and shortness of breath) did associate with a positive test result ($X^2$ [1, N = 53] = 1.63, $P$ = .047). The majority (75%) of the SARS-CoV-2 positive patients required hospital admission.

There are emerging data from China that patients in the ABO-A blood group may be more susceptible to SARS-CoV-2 infection. We observe a similar trend (Table 1). Compared with the total KTR population followed at our center, a larger percentage of SARS-CoV-2 positive patients are ABO-A (40%, compared with 34.11%). However, this trend failed to achieve statistical significance ($P$ = .22). Similarly, among patients who presented for SARS-CoV-2 testing, patients with ABO-A were slightly more likely to test positive compared to the non-ABO-A patients tested (42% and 38%, respectively). However, this trend also failed to achieve statistical significance ($P$ = .74).

Our cohort of KTRs showed no significant difference in absolute lymphocyte counts (ALC) between patients who tested positive and negative for SARS-CoV-2 (Table 3). In this cohort, both the average and median ALC were less than 1 in all patients who were tested for SARS-CoV-2 infection, both those with and without COVID-19. However, hemoglobin and hematocrit were both significantly higher in patients with COVID-19 when compared to patients who tested negative for SARS-CoV-2, possibly reflecting hemoconcentration in the former. No significant associations were identified among the other laboratory data points reviewed (Table 3).

We also tested the hypothesis that patients with a history of a transplant-related virus might be more prone to COVID-19 (Table 4). Interestingly, we found a high association between a SARS-CoV-2 positive test result and a known history of polyoma BKV (specifically, serum quantitative PCR greater than Log 3 viremia, $P$ = .002). Although our cohort is relatively small and larger cohorts are necessary to confirm our findings, a history of BK infection had a positive predictive value of 80% at predicting a SARS-CoV-2 positive test result and a specificity of 94%. Furthermore, a history of BK infection had a positive likelihood ratio of 6.6. We considered the possibility that continuous variables associated with a BK infection history might also predict SARS-CoV-2 infection. In particular, receiver operator curves were constructed using BK log titer at peak level of viremia and duration of viremia exceeding log 3. However, this data exploration was limited by having only 2 patients with a negative SARS-CoV-2 test and a history of BK viremia (Fig 1). We did not find an association with a history of cytomegalovirus infection.

Our transplant center has a large cohort of patients receiving belatacept as their primary immunosuppressant agent (Table 4). Intriguingly, we observed a trend toward a lower rate of SARS-CoV-2 positive testing among the patients on belatacept maintenance therapy in this cohort. In the patients who resulted SARS-CoV-2 positive, only 2 (10%) were on belatacept-based immunosuppression. This was in contrast to calcineurin inhibitors, where 85% of SARS-CoV-

**Table 1. Patient Demographics**

| Variable n (%) | All Patients | COVID Positive n = 20 (36) | COVID Negative n = 33 (62) | *P* Value |
|---|---|---|---|---|
| Mean age (years) | 57.8 | 59.5 | 56.69 | .46 |
| Sex | - | - | - | .21 |
| Male | 26 (49) | 12 (60) | 14 (42) | - |
| Female | 27 (51) | 8 (40) | 19 (58) | - |
| Education | - | - | - | .34 |
| Beyond high school | 14 (32) | 4 (24) | 10 (37) | - |
| High school and below | 30 (68) | 13 (76) | 17 (63) | - |
| Race | - | - | - | .35 |
| White/Caucasian | 22 (42) | 6 (30) | 16 (48) | - |
| African American | 22 (42) | 10 (50) | 12 (36) | - |
| Hispanic | 8 (15) | 3 (15) | 5 (15) | - |
| Asian | 1 (2) | 1 (5) | 0 | - |
| Insurance type | - | - | - | .02 |
| Medicare/Medicaid | 38 (72) | 18 (90) | 20 (61) | - |
| Others (Private Insurance) | 15 (28) | 2 (10) | 13 (39) | - |
| Mean household income of patient's zip code (USD/year) | - | 60,820.33 | 67,199.37 | .36 |
| Blood group | - | - | - | .74 |
| ABO-A blood group | 19 (37) | 8 (40) | 11 (35) | - |
| Non-ABO-A blood group | 32 (63) | 12 (60) | 20 (65) | - |
| Mean time since transplant (years) | 8.20 | 6.36 | 9.31 | .14 |
| Donor type | - | - | - | .39 |
| Living | 17 (32) | 5 (25) | 12 (36) | - |
| Deceased | 36 (68) | 15 (75) | 21 (64) | - |
| Diabetes | - | - | - | .18 |
| Yes | 23 (43) | 11 (55) | 12 (36) | - |
| No | 30 (57) | 9 (45) | 21 (64) | - |
| Chronic obstructive pulmonary disease | - | - | - | .28 |
| Yes | 3 (6) | 2 (10) | 1 (3) | - |
| No | 50 (94) | 18 (90) | 32 (97) | - |
| Cardiomyopathy | - | - | - | .55 |
| Yes | 16 (30) | 7 (35) | 9 (27) | - |
| No | 37 (70) | 13 (65) | 24 (73) | - |
| Valvular disease | - | - | - | .16 |
| Yes | 7 (13) | 1 (5) | 6 (18) | - |
| No | 46 (87) | 19 (95) | 27 (82) | - |
| Asthma | - | - | - | .58 |
| Yes (n = 4) | 4 | 1 (5) | 3 (9) | - |
| No (n = 49) | 49 | 19 (95) | 30 (91) | - |
| Nonasthma lung disease | - | - | - | .74 |
| Yes | 12 (23) | 5 (25) | 7 (21) | - |
| No | 41 (77) | 15 (75) | 26 (79) | - |
| Smoking status | - | - | - | - |
| Never smoker | 26 (49) | 9 (45) | 17 (52) | - |
| Former smoker | 25 (47) | 11 (55) | 14 (42) | - |
| Light tobacco smoker (n = 1) | 1 (2) | 0 (0) | 1 (3) | - |
| Current smoker (n = 1) | 1 (2) | 0 (0) | 1 (3) | - |

2 positive patients were on tacrolimus. Belatacept-based immunosuppression regimens were used for 13 patients within our total cohort tested. We observed a lower rate of SARS-CoV-2 positive testing in the belatacept patients compared to the patients receiving other regimens (15% and 45%, respectively). This lower rate nearly achieved significance ($P = .056$) in this small cohort. We did not find an association between either a biopsy proven rejection history or active therapy with an angiotensin converting enzyme inhibitor or angiotensin receptor blocker in our cohort (Table 4).

Medical record archiving prevented ascertainment of transplant induction medication and ABO blood group for 2 patients transplanted prior to 1998. These 2 patients were not included in the analysis of these 2 categories.

## DISCUSSION

The COVID-19 pandemic represents the most significant health crisis to face our society in over a century. Many changes to how we assess, test, and monitor KTRs during

**Table 2. Presenting Symptoms**

| Variable n (%) | All Patients n = 53 | COVID Positive n = 20 (38) | COVID Negative n = 33 (62) | *P* Value |
|---|---|---|---|---|
| Presenting symptoms | | | | |
| Fever | 30 (57) | 11 (67) | 19 (58) | .85 |
| Cough | 28 (53) | 14 (70) | 14 (42) | .06 |
| Shortness of breath | 25 (47) | 12 (60) | 13 (39) | .14 |
| CNS symptoms/encephalopathy | 7 (13) | 2 (10) | 5 (15) | .59 |
| GI symptoms/diarrhea | 16 (30) | 8 (40) | 8 (24) | .22 |
| Anosmia | 1 (2) | 0 (0) | 1 (3) | - |
| Dysgeusia | 2 (4) | 1 (5) | 1 (3) | .71 |
| Symptoms prompting hospital admission | 38 (72) | 15 (75) | 23 (70) | - |

this unique time in medical history are necessary. The initially limited availability of viral testing, coupled with the recommendation to use telehealth as a way to reduce patient exposure to the medical center, prompted a QAPI to search for a KTR phenotype that indicated a higher probability of having COVID-19 disease.

In our cohort, patients with a history of BKV were at increased risk to test positive for COVID-19 compared to patients without a history of BKV. The reason for this association is unclear. One hypothesis is that despite a reduction in immunosuppression to manage BKV, these KTRs remain too immunosuppressed to control and clear the SARS-CoV-2 infection. Early reports indicate that the severe acute respiratory syndrome associated with COVID-19 can be attributed to excessive proinflammatory host immune responses [3]. Therefore, an alternative hypothesis is that patients with a history of BKV may be at risk for more clinically apparent disease compared to KTRs without a history of BKV because of their reduced immunosuppression. The lack of a reliable laboratory test to quantitatively measure the responsiveness of the immune system in immunosuppressed patients complicates the search for a mechanistic link between BKV and COVID-19. Although statistically significant in our small patient cohort, larger studies of KTRs with COVID-19 disease and a history of BKV will be required to confirm and better understand this association. As a finding from this QAPI, we recommend that until additional data become available, KTRs with a history of BKV should be prioritized for SARS-CoV-2 testing.

**Table 3. Mean Serum Laboratory Values at Time of COVID Testing**

| Serum Laboratory Variables (mean) | COVID Positive (n = 20) | COVID Negative (n = 33) | *P* Value |
|---|---|---|---|
| Hemoglobin prior to diagnosis (g/dL) | 11.11 | 10.48 | .350 |
| Hb (g/dL) | 12.33 | 9.98 | .002 |
| Hematocrit (%) | 37.76 | 31.94 | .037 |
| White × 1000/μL | 7.10 | 9.12 | .208 |
| Absolute lymphocyte count, prior to diagnosis × 1000/μL | 1.05 | 1.17 | .562 |
| Absolute lymphocyte count × 1000/μL | 0.88 | 0.85 | .914 |
| Platelets × 1000/μL | 201.24 | 208.26 | .837 |
| Sodium (mmol/L) | 136.88 | 138.37 | .157 |
| Potassium (mmol/L) | 4.55 | 4.32 | .319 |
| Chloride (mmol/L) | 100.12 | 102.59 | .082 |
| Bicarbonate (mmol/L) | 19.35 | 20.78 | .448 |
| Blood urea nitrogen (mg/dL) | 40.71 | 40.85 | .986 |
| Creatinine (mg/dL) | 2.60 | 3.03 | .596 |
| Glucose (mg/dL) | 258.65 | 130.07 | .057 |
| Aspartate aminotransferase (U/L) | 71.38 | 27.45 | .269 |
| Alanine aminotransferase (U/L) | 51.00 | 24.40 | .200 |
| C-reactive protein, highly sensitive (mg/L) | 95.89 | 118.48 | .530 |
| Ferritin (ng/mL) | 2042.07 | 2481.18 | .737 |

An interesting finding in our cohort was the signal toward a potential protective effect of belatacept immunosuppression in patients who were tested but were not SARS-CoV-2 positive. This difference was significantly different from the SARS-CoV-2 positive cohort, who were more likely to be on tacrolimus-based immunosuppression. Over 250 kidney transplant recipients at our center are maintained on belatacept as part of their immunosuppressive regimen, and the majority are late conversions (> 3 months post-transplant) from calcineurin inhibitor therapy. Recently, concerns about belatacept and an increased risk of opportunistic infection have been emphasized [4]. Bertrand et al found that belatacept was associated with an incidence of 9.8 opportunistic infections/100 person years, including Pneumocystis jirovecii pneumonia and cytomegalovirus disease [4]. This was more commonly seen in the group who converted to belatacept early, <6 months post-transplant. This observation is in contrast to our QAPI data, which showed a lower rate of SARS-CoV-2 positive testing in belatacept-treated patients. Notably, Bertrand et al did not propose an explanation for the increase in opportunistic infections seen with belatacept. Based on what is known about belatacept's impact on the immune system, it is unclear why belatacept was associated with a protective effect in our cohort.

Not surprisingly, we identified an association between the number of classic symptoms (fever, cough, and shortness of

**Table 4. Transplant Relevant Details**

| Variable n (%) | All Patients n = 53 | COVID Positive n = 20 (38) | COVID Negative n = 33 (62) | *P* Value |
|---|---|---|---|---|
| Induction immunosuppression | | | | |
| Thymoglobulin (n = 18) | 18 (34) | 9 (45) | 9 (27) | - |
| Alemtuzumab (n = 13) | 13 (25) | 6 (30) | 7 (21) | - |
| Atgam (n = 2) | 2 (4) | 0 (0) | 1 (3) | - |
| Basiliximab (n = 11) | 11 (21) | 5 (13) | 6 (18) | - |
| Unknown (n = 9) | 9 (17) | 0 (0) | 9 (27) | - |
| Maintenance immunosuppression | | | | |
| Tacrolimus | 34 (64) | 17 (85) | 17 (52) | .01 |
| Belatacept | 13 (25) | 2 (10) | 11 (33) | .056 |
| Cyclosporine | 4 (8) | 1 (5) | 3 (9) | - |
| Sirolimus | 1 (2) | 0 (0) | 1 (3) | - |
| Everolimus | 1 (2) | 0 (0) | 1 (3) | - |
| Mycophenolate | 34 (64) | 13 (65) | 21 (64) | - |
| Azathioprine | 4 (8) | 0 (0) | 4 (12) | - |
| Prednisone | 50 (94) | 18 (90) | 32 (97) | - |
| Triple maintenance immunosuppression (n = 35) | 35 (66) | 11 (55) | 24 (73) | - |
| History of allograft rejection | - | - | - | .14 |
| Yes | 14 (27) | 8 (40) | 7 (21) | - |
| No | 38 (73) | 12 (60) | 26 (79) | - |
| H/O BK virus infection (n = 10) | - | - | - | .002 |
| Positive | 10 (19) | 8 (40) | 2(6) | - |
| Negative | 43 (81) | 12 (60) | 31(94) | - |
| RAAS blockade use prior to testing | - | - | - | .55 |
| Yes | 11 (21) | 5 (25) | 6 | - |
| No | 42 (79) | 15 (75) | 27 | - |

breath) reported and the likelihood of testing positive for SARS-CoV-2. Nonetheless, 24% of screened patients presenting with only 1 symptom were confirmed as SARS-CoV-2 positive on testing. Therefore, similar to the general population, a high level of suspicion for COVID-19 is warranted in KTRs with limited symptomatology. Patients presenting with 2 or 3 symptoms were more likely to have a positive test, but this finding may be skewed by the relatively high false negative rate of the SARS-CoV-2 test, which is reported to be as high as 40% in the general population.

Given concerns regarding testing availability and the need to limit the KTR's contact with medical facilities during this pandemic, we sought to identify symptoms more frequently associated with COVID-19 positivity. We reviewed multiple symptom types, including fever, dyspnea, cough, gastrointestinal symptoms, neurologic symptoms, anosmia, and dysgeusia. As a cohort, KTRs with COVID-19 demonstrated all of these symptoms in varying combinations, with the exception of anosmia (none of our SARS-CoV-2 positive KTRs had documented anosmia). The most common presenting symptoms were cough (70%), dyspnea (60%), and fever (55%). While not as prevalent, these symptoms were also frequently present among those who tested negative for SARS-CoV-2 (42% of such patients had cough, 39% had dyspnea, and 58% had fever). When only 1 of these symptoms was present, there was no statistically significant difference between patients with or without COVID-19. However, the presence of more than 1 of these common presenting symptoms did correlate with SARS-CoV-2 positivity. If patients had 2 or 3 of these symptoms (cough, dyspnea, and/or fever), they were more likely to test positive for SARS-CoV-2, and this difference was statistically significant. Given this, we advocate for expediting testing of our KTRs who present with at least 2 of these 3 symptoms during the COVID-19 pandemic. As knowledge and experience with COVID-19 increased, anosmia and dysgeusia became increasingly recognized as symptoms of this syndrome [5,6].

Some of these patients remain minimally symptomatic (and so can easily spread the infection), while others were reported to progress and require hospitalization. The exact prevalence of these symptoms is not entirely clear, although 1 Italian study that surveyed hospitalized patients with COVID-19 found that 33.9% of patients had alteration in either taste or smell, the vast majority of whom developed these symptoms prior to requiring hospitalization [7]. In our cohort of patients, we did not identify anosmia or dysgeusia as common presenting symptoms in KTRs. Even if one speculated that there could have been false negative test results, the overall prevalence of these symptoms was quite low in the entire cohort of patients, much lower than reported in other studies [7].

Many of the reports of anosmia and dysgeusia in COVID-19 were not published until late March and early April 2020, and so our KTRs were not routinely queried about them in our early experience with this infection. Given this, our low numbers may reflect under-reporting of taste and smell alterations. Based on our currently available data, we cannot

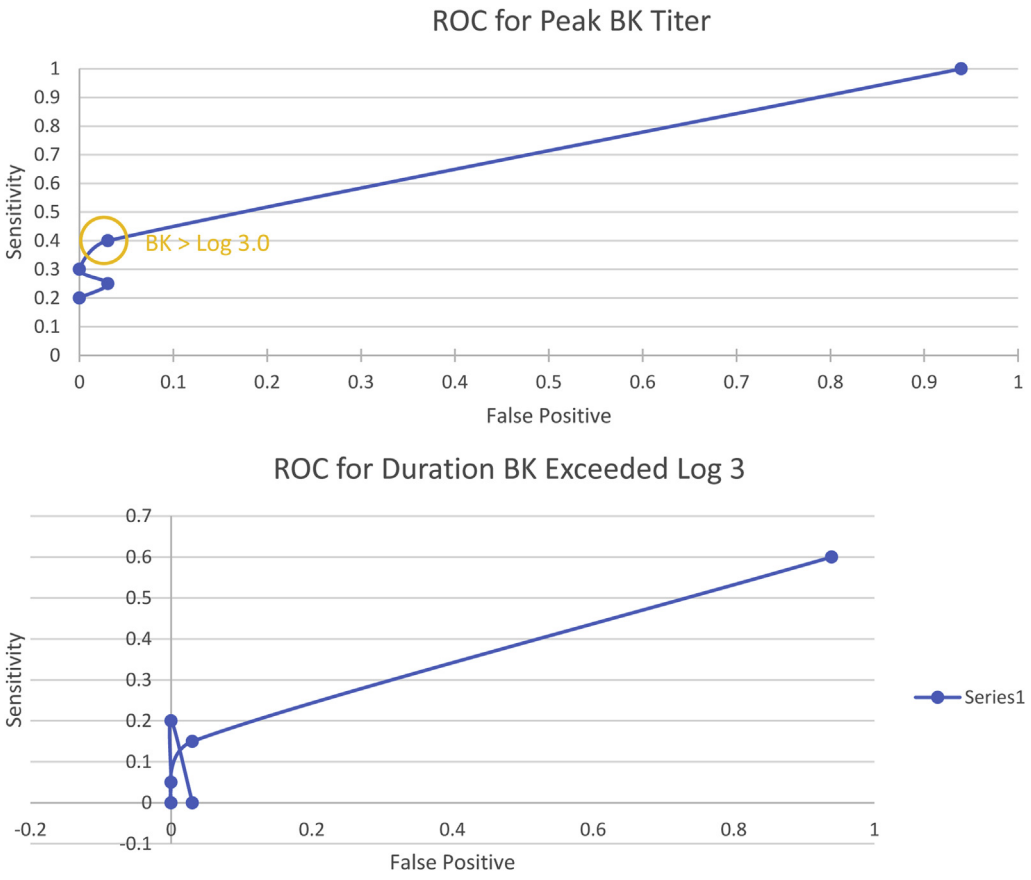


**Fig 1.** Receiver operator curves exploring the hypothesis that peak BK viremia titer and duration of Bk viremia exceeding Log 3 might predict SARS-CoV-2 test positivity in kidney transplant recipients.

state that anosmia or dysgeusia are common presenting symptoms in KTRs. However, given the potential for underreporting and reports in the literature that these symptoms present early in the disease process, it seems prudent to screen KTRs for anosmia and dysgeusia when evaluating for possible COVID-19 [8].

Recent media reports have highlighted the ways in which race and socioeconomic status are playing a role in the COVID-19 pandemic [9]. Little peer reviewed data have been published on this topic, particularly regarding how education level may affect incidence and outcomes of SARS-CoV-2 infection in different populations. One group reported that those with higher education level (i.e., doctoral degree) reported more adherence to social distancing and other preventative measures; this could have implications on infection risk, but this data have yet to be published in a peer reviewed journal [10]. Among our KTRs who were tested for COVID-19, 44 patients had data available regarding educational history. Of these 44 patients, 14 patients (31.8%) had obtained education beyond the high school level. Although a minority of our KTRs had been educated beyond high school, there was no significant difference between those who had COVID-19 and those who did not. It is unknown whether this is because educational history does not correlate with infection rates or if this reflects the fact that the majority of our cohort have not completed education beyond high school.

There was no statistically significant difference in sex between SARS-CoV-2 positive and negative patients in our KTR cohort. Some published data have indicated there may be a slight male predominance in this infection; one such paper reported 54.3% of patients hospitalized with COVID-19 were men [11]. Other data from China have also raised the question of a sex difference in infection, though it is unclear if this data can be extrapolated to the United States, as there is a larger proportion of men in China as compared to the US population [12]. Although some authors have reported a slight male predominance among patients, other cohorts show similar rates of infection among men and women [13]. Our data also show similar rates of COVID-19 between the sexes.

Some data suggest that even if men and women are infected at similar rates, mortality may be higher in men, although this has yet to be definitively proven. One analysis from China showed a statistically significant case fatality rate between the sexes (case fatality of 2.8% among men

and 1.7% among women) [13]. Since the focus of this work was on presenting symptoms and not final outcomes, we cannot comment on any potential sex difference in disease severity or mortality risk in our KTRs. This may be worth exploring in future work, but sex should not be a factor in deciding which KTRs to test for SARS-CoV-2 infection.

It is common knowledge that individuals with diabetes are at higher risk of infectious diseases, including respiratory pathogens. Moreover, diabetics are known to have a more severe disease when infected with respiratory viruses such as influenza, respiratory syncytial virus, etc. Thus far, there have been conflicting reports identifying diabetes as an isolated risk factor for the SARS-CoV-2 infection. Several small studies of patients hospitalized with COVID-19 [14,15] showed that a comorbid diagnosis of diabetes or overt hyperglycemia was not an identifiable risk factor for SARS-CoV-2 infection. Contrasting these findings, a report by the Chinese Center for Disease Control and Prevention reviewing more than 72,000 cases showed a 3-fold higher mortality risk of patients with diabetes as compared to the general population (7.3% vs 2.3%) [16]. Among the 20 patients that tested positive in our cohort, more than half (55%) were known to have diabetes with a mean HbA1c of 7.9. A lower proportion (36%) of patients that tested negative for SARS-CoV-2 were known to have diabetes. We speculate that KTRs with diabetes are also more likely to have other cardiopulmonary comorbidities, placing them at a higher risk of contracting COVID-19 and perhaps having worse outcomes in comparison to the general population.

Many viral illnesses can cause bone marrow suppression, and it is not unusual for a viral infection to present with cytopenias. Early experience with COVID-19 indicates lymphopenia to be a common feature, with 1 study showing significant lymphopenia in as many as 72% of patients with COVID-19 [11]. While lymphopenia may be a diagnostic clue to COVID-19 on initial evaluation of a patient, the literature also suggests that severity of lymphopenia may correlate with the overall severity of disease. Multiple retrospective studies have found that patients with more severe disease had lower absolute lymphocyte counts than patients with more mild disease, and 1 group noted that lymphopenia was more common in patients who ultimately died of their infection [14,15]. While this data may be significant in risk stratification and prognostication of patients with COVID-19 in general, we believe it will be less helpful in the evaluation of KTRs with suspected or confirmed COVID-19. Our cohort of KTRs showed no significant difference in the ALC between patients with and without COVID-19. Lymphopenia is common in our KTRs; in this cohort, both the average and median ALC were less than 1 in all patients who were tested for SARS-CoV-2 infection, both those with and without COVID-19. We therefore conclude that lymphopenia is not particularly suggestive of COVID-19 in KTRs and should not be used in deciding whom to test for the infection. Although the goal of our work was to describe a phenotype amongst our KTRs (and thus improve our testing process), it would seem that the baseline prevalence of lymphopenia among KTRs limits the overall utility of using this parameter in prognostication.

Based on knowledge of ABO blood groups and susceptibility risk during the previous SARS coronavirus outbreak in 2003 [17], there have been attempts to identify an association between ABO blood type frequencies and the novel coronavirus. Early preprint reports from China found a higher risk of SARS-CoV-2 infection among patients with ABO blood group A as compared to non-A blood groups [18]. Blood type O was found to have a protective effect toward the infection. Differential inhibition of adhesion of SARS-CoV-2 to angiotensin converting enzyme2 expressing cell lines by natural anti-blood group antibodies is hypothesized as a possible mechanism of these findings. Interestingly, the relative proportion of SARS-CoV-2 positive patients in our cohort with ABO blood type A was significantly higher (40%) than the rest of the non-A blood types. Although our observation did not reach statistical significance ($P$ value = .7) because of the limited sample size, further studies into this association with larger cohorts is certainly warranted.

Recognizing that our early cohort of KTRs tested for SARS-CoV-2 is relatively small, and additional factors that may have confounded our results or their interpretation are unknown at this time, this data seeking to identify a KTR COVID-19 phenotype to aid test utilization in the setting of limited testing availability are valuable to the transplant community. As transplant centers across the world race to identify which of their KTRs are at a higher risk for COVID-19, data from our QAPI should influence decisions about whom to prioritize for testing while test availability remains a limiting factor.

## CONCLUSIONS

We initiated a QAPI at our transplant center to track KTRs through the SARS-CoV-2 testing process. Recognizing that limited testing availability forces health care professionals to restrict testing to KTRs most likely to benefit from testing, we collected data to determine if patient demographics and reported symptoms could aid in predicting disease. Interestingly, we identified a strong association between the history of BKV and testing positive for SARS-CoV-2, indicating that this group of KTRs should be prioritized for SARS-CoV-2 testing. We also identified a signal suggesting that belatacept maintenance immunosuppression was protective against presenting with symptomatic COVID-19 infection. Additional data from larger and longer-term cohorts will be necessary to validate these associations.

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